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THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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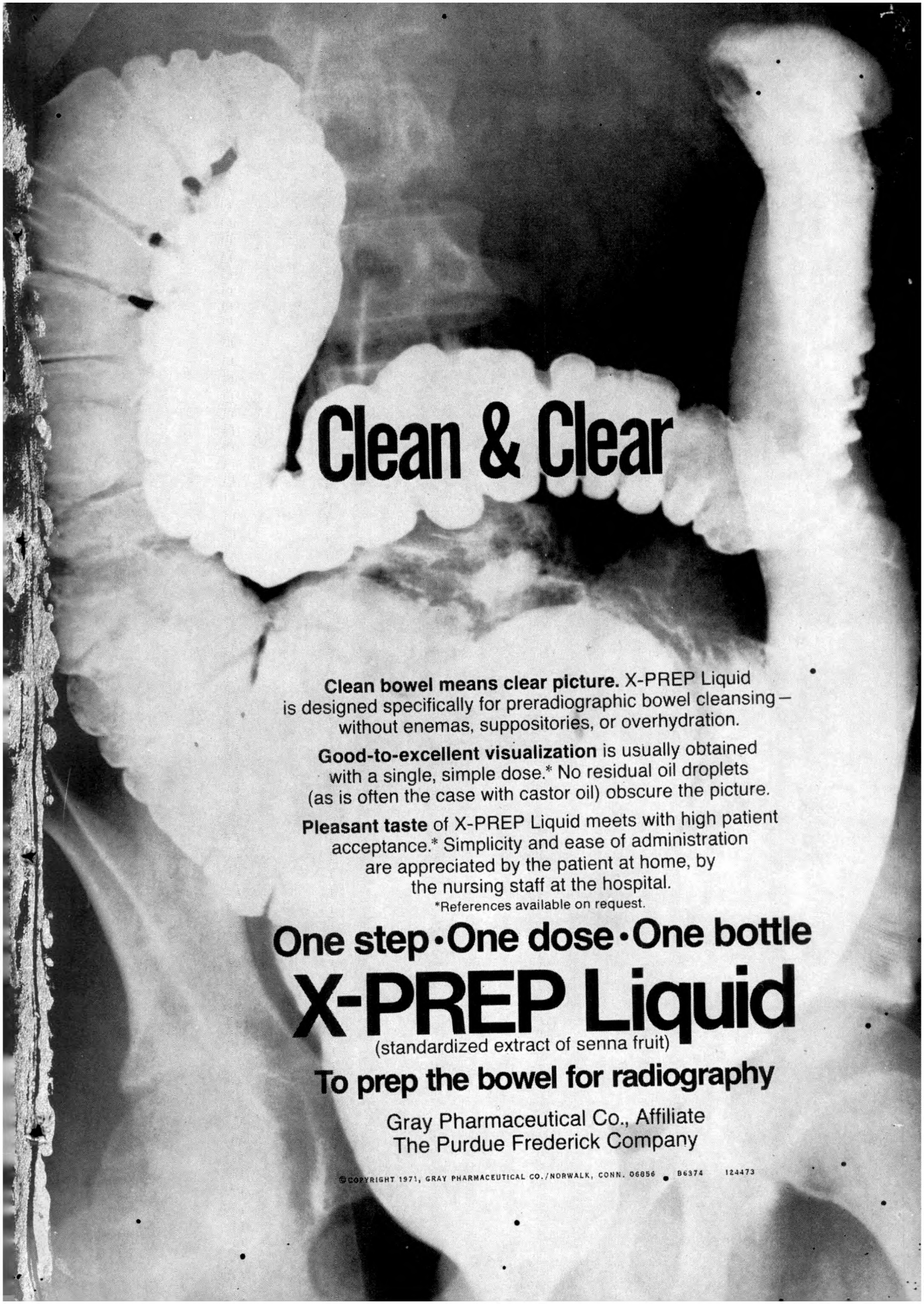
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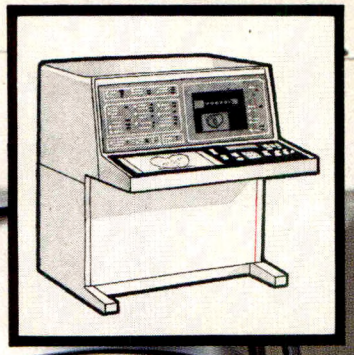

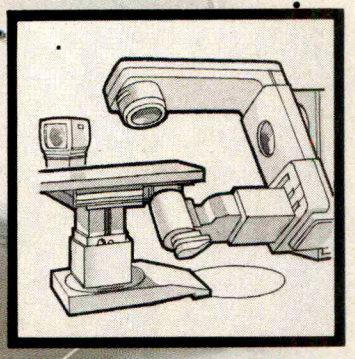

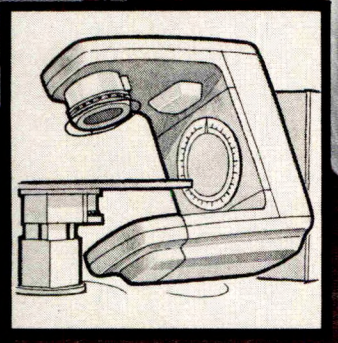

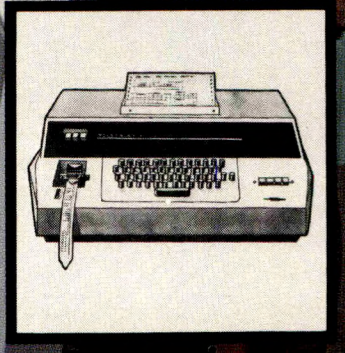

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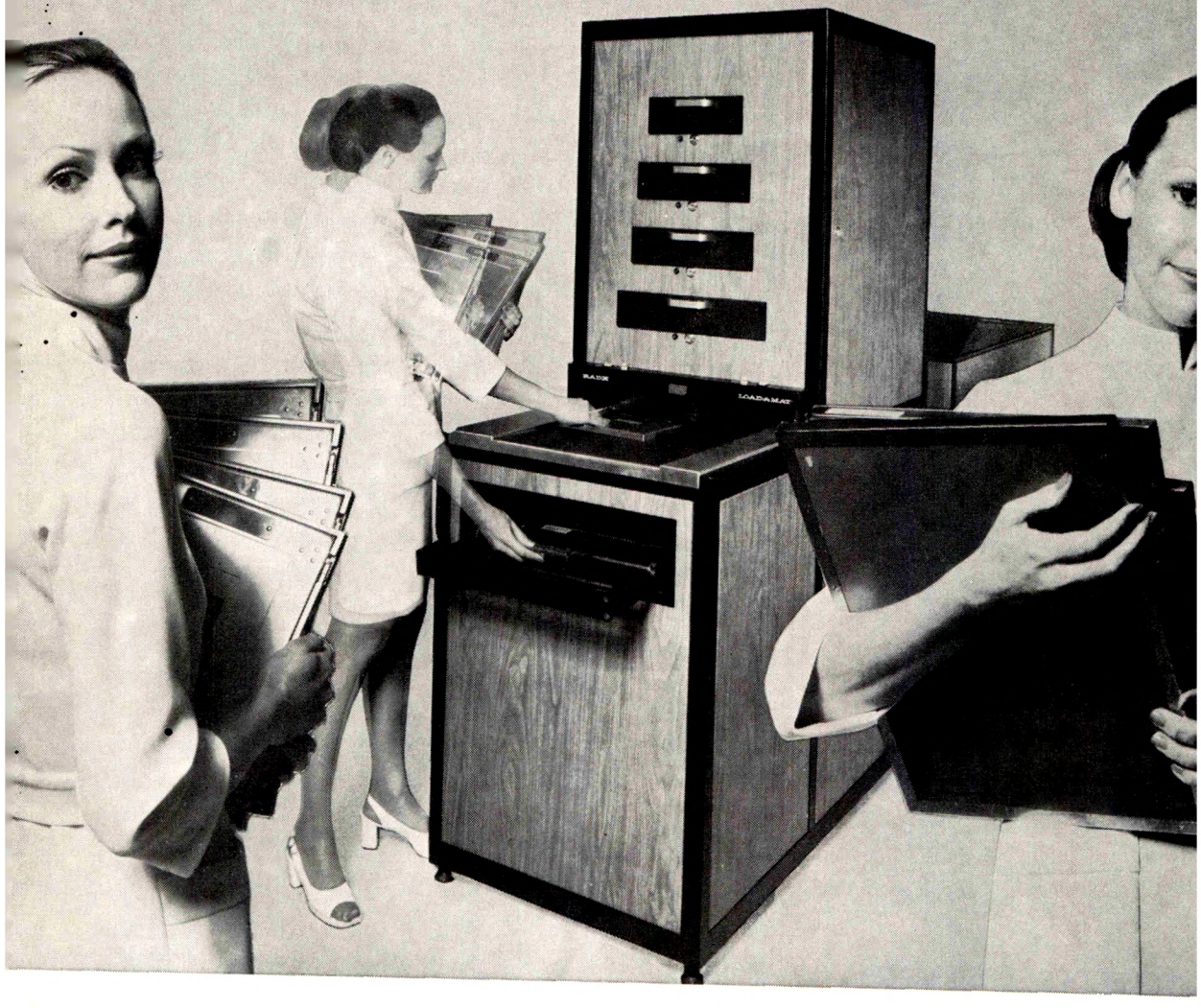

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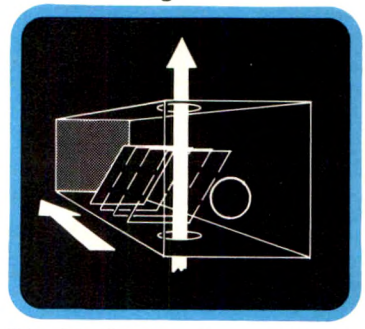

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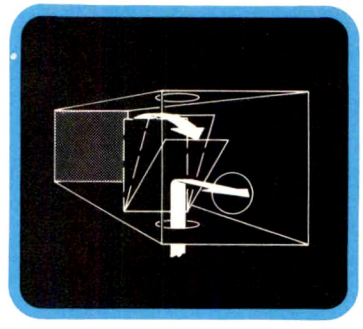

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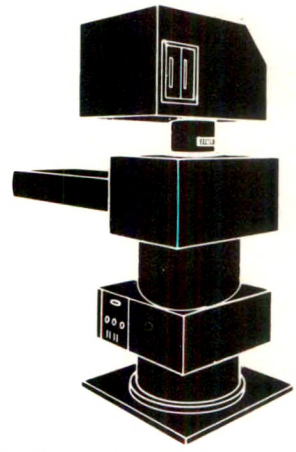

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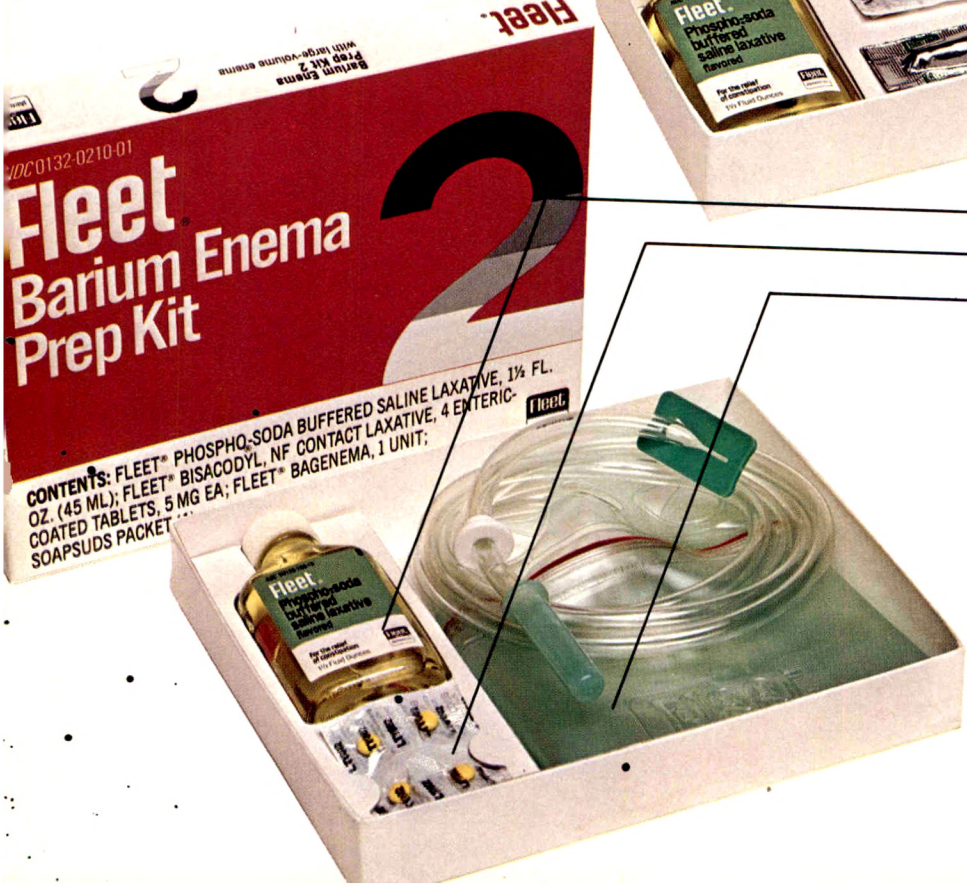

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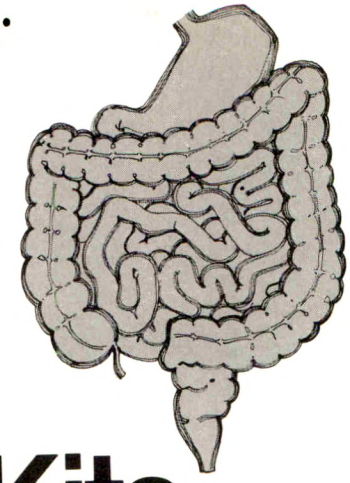

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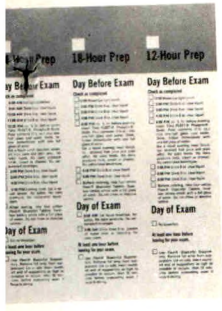

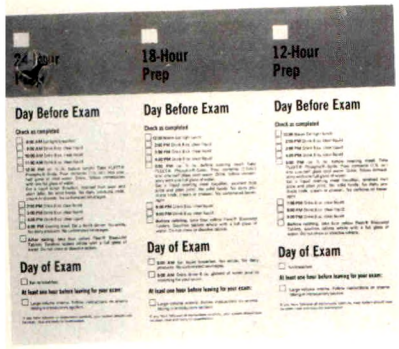

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- MINITEC has its own handle for easy lifting, easy carrying and reduced hand exposure
- Weighs only 24½ lbs., less than 5" in diameter, under 8½" high

signed for easy elution

Sets up in seconds

Elutes in only 3 minutes after eluent vial has emptied

signed for safety

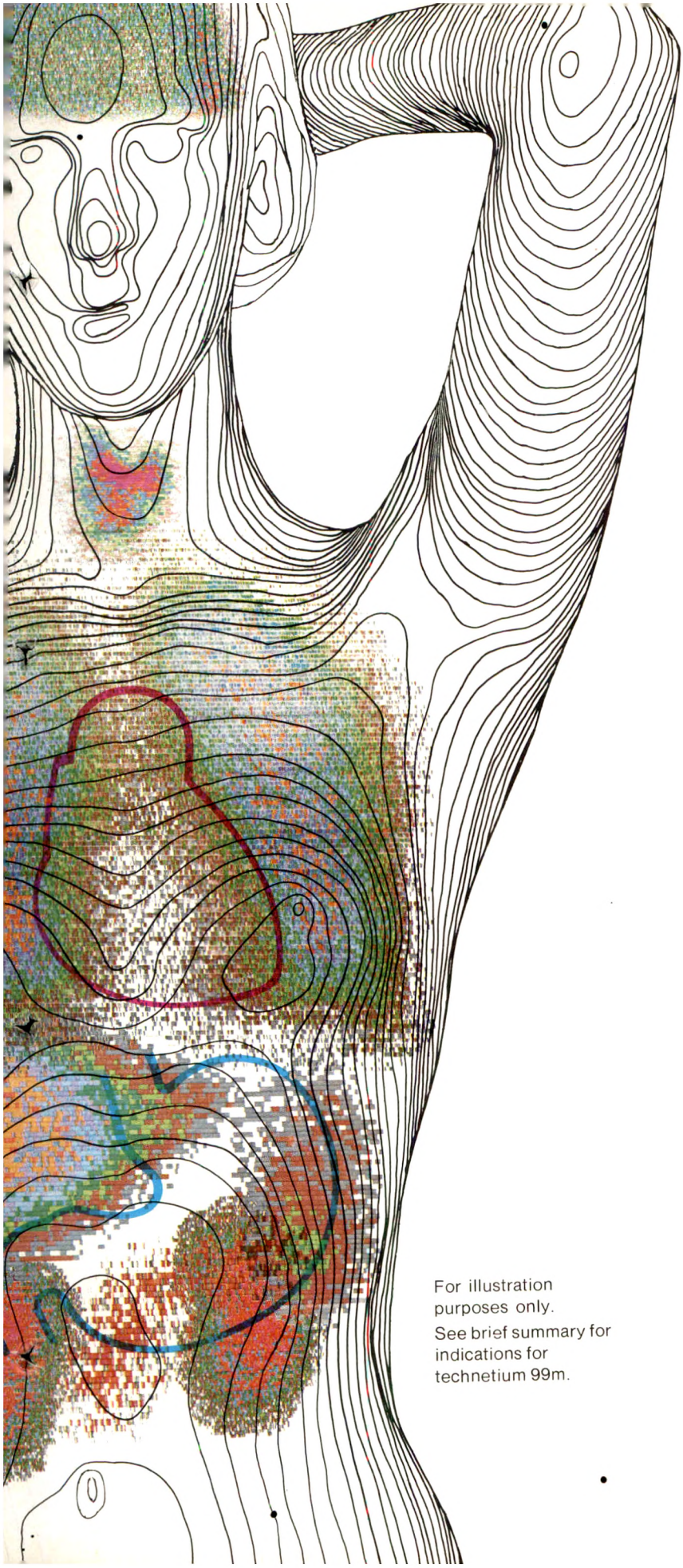
No exposed tubing when eluting

1⅝" lead surrounds the MINITEC column

1½" of extra lead protection from MAXI-SHIELD™. Base, cap and interlocking half rings easily assembled on site . . . only the cap is removed for elution. (You get MAXI-SHIELD free with your first MINITEC Generator purchase.)

signed for convenience

MINITEC Generator is available in 50,100, 200, 300, 400 and 500 mCi potencies. Delivery on Monday AM (precalibrated through Thursday) and Wednesday (precalibrated through Monday) provides maximum versatility to satisfy technetium requirements of your lab's work load.



For illustration purposes only.

See brief summary for indications for technetium 99m.

Minitec®
(Technetium 99m) Generator

Minitec® (Technetium 99m) Generator provides means of obtaining a sterile, non-pyrogenic supp of technetium 99m (^{99m}Tc) as sodium pertechneta ^{99m}Tc.

Indications: Sodium pertechnetate ^{99m}Tc is indicat for brain imaging, thyroid imaging, salivary gla imaging, blood pool imaging, and placenta loc ization.

Contraindications: At present, there are no kno contraindications to the use of sodium pertechr tate ^{99m}Tc.

Warnings: Radiopharmaceuticals should be us only by physicians who are qualified by speci training in the safe use and safe handling of rad nuclides, produced by nuclear reactor or cyclotr and whose experience and training have been a proved by the appropriate federal or state ager authorized to license the use of radionuclid

This radiopharmaceutical should not be adm istered to women who are pregnant or who m become pregnant or during lactation unless the formation to be obtained outweighs the possi potential risks from the radiation exposure involv Ideally, examinations using radiopharmaceutica especially those elective in nature, of a woman childbearing capability should be performed dur the first few (approximately 10) days following onset of menses.

Since radioactive pertechnetate is secreted milk during lactation, formula-feedings should substituted for breast-feedings.•

Important: Since material obtained from the g erator may be intended for intravenous admi tration, aseptic technique must be strictly observ in all handling. Only the eluent provided should used to elute the generator. Do not administer terial eluted from the generator if there is evidence of foreign matter.

Precautions: As in the use of any other radioac material, care should be taken to insure minim radiation exposure to the patient consistent w proper patient management and to insure minim radiation exposure to occupational workers.

At the time of administration, the solution sho be crystal clear.

Adverse Reactions: At present, adverse reacti have not been reported following the use of sod pertechnetate ^{99m}Tc.

For complete prescribing information, con package insert.

How Supplied: Minitec (Technetium 99m) G erator is available in potencies of 50, 100, 2 300, 400, and 500 mCi. Supplied with the gener are vials of eluent containing 5 ml. of a ste non-pyrogenic solution of 0.9% sodium chlorid water for injection. Also supplied is suitable eq ment for eluting, collecting, and assaying technetium 99m.

Medotopes®

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It's all given to you in a straightforward, easy-to-read fashion complete with curves and diagrams. It tells you exactly what each product is, how it's used and how it's available. Because each product is described in its own little booklet, you can keep the individual booklets that interest you.

The next time you see The Ilford Man coming down the hall, pull him aside and whisper in his ear, "I want to see your little black book."

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the image quality and exact diagnostic format you need

Searle Micro Dot Imager

Static, dynamic & whole body imaging . . . 15 formats, 3 film sizes

The Searle Micro Dot Imager offers Pho/Gamma users a versatile display system for single-organ or whole body imaging using economical X-ray film. Three film sizes and 15 image formats let you choose the exact format best suited for any study. State-of-the-art optics and electronics put as many as 80 images on one film with single-image fidelity. You can even mix static, dynamic and different size images on the same sheet of film. An exclusive, lightweight cassette design speeds and simplifies loading and unloading of film.

The Micro Dot provides distinct, well-focused scintidots in all image sizes; it gives you superior imaging clarity, constant focus and freedom from astigmatism regardless of dot intensity and location. Absolute exposure control—with pushbutton settings for routine studies—assures correct, repeatable exposures from day to day and month to month in all image sizes.

Designed for clinical utility and operational simplicity, the Micro Dot Imager is the most complete display system available for the Pho/Gamma Scintillation Camera. For more information—including complete specifications—just write or phone your Searle representative. He'll be glad to show you how it can add unmatched versatility, convenience and economy to your laboratory's gamma imaging capabilities.

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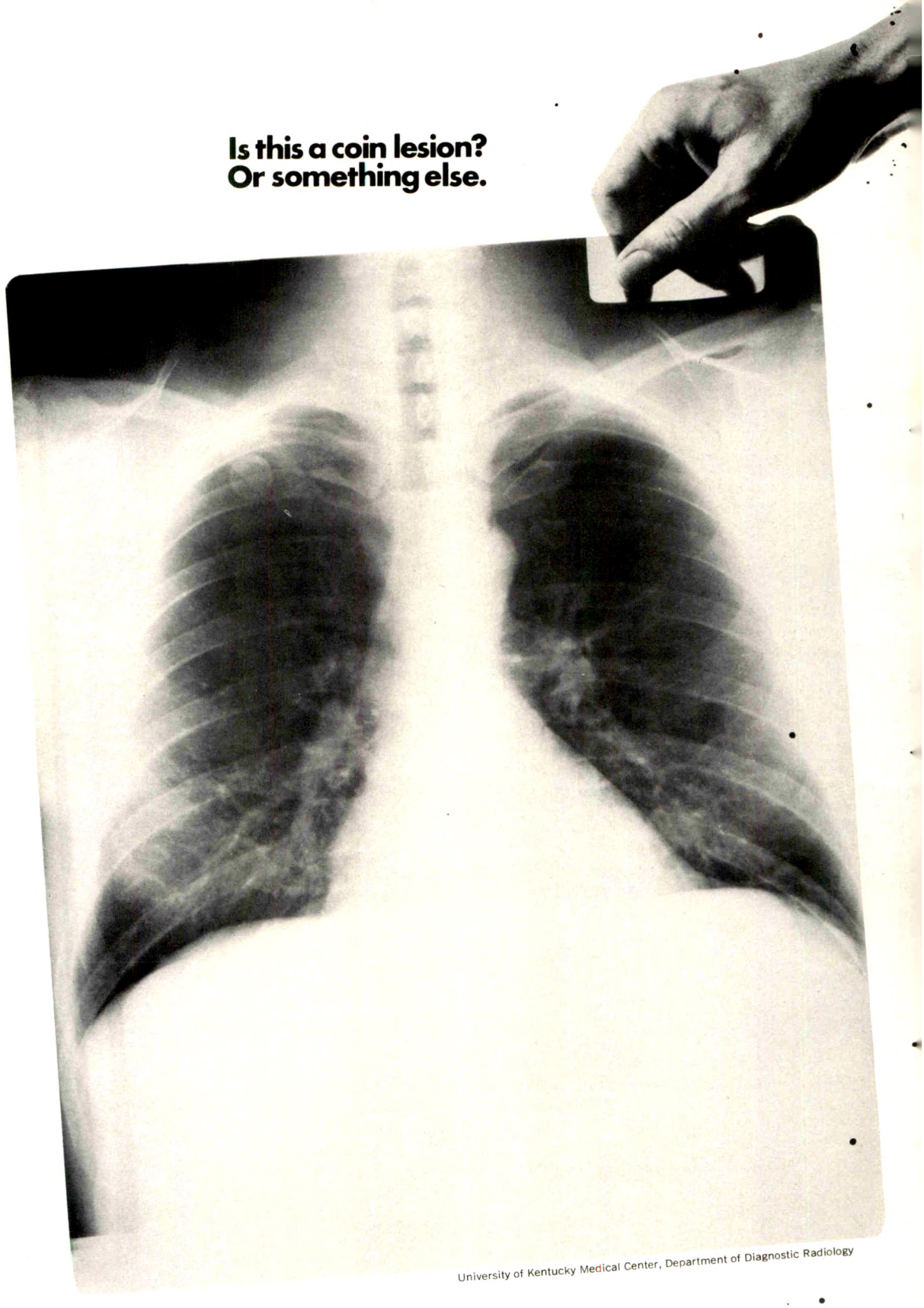
Subsidiary of G. D. Searle & Co.
2000 Nuclear Drive, Des Plaines, Illinois 60018
Phone 312-298-6600

JM-356

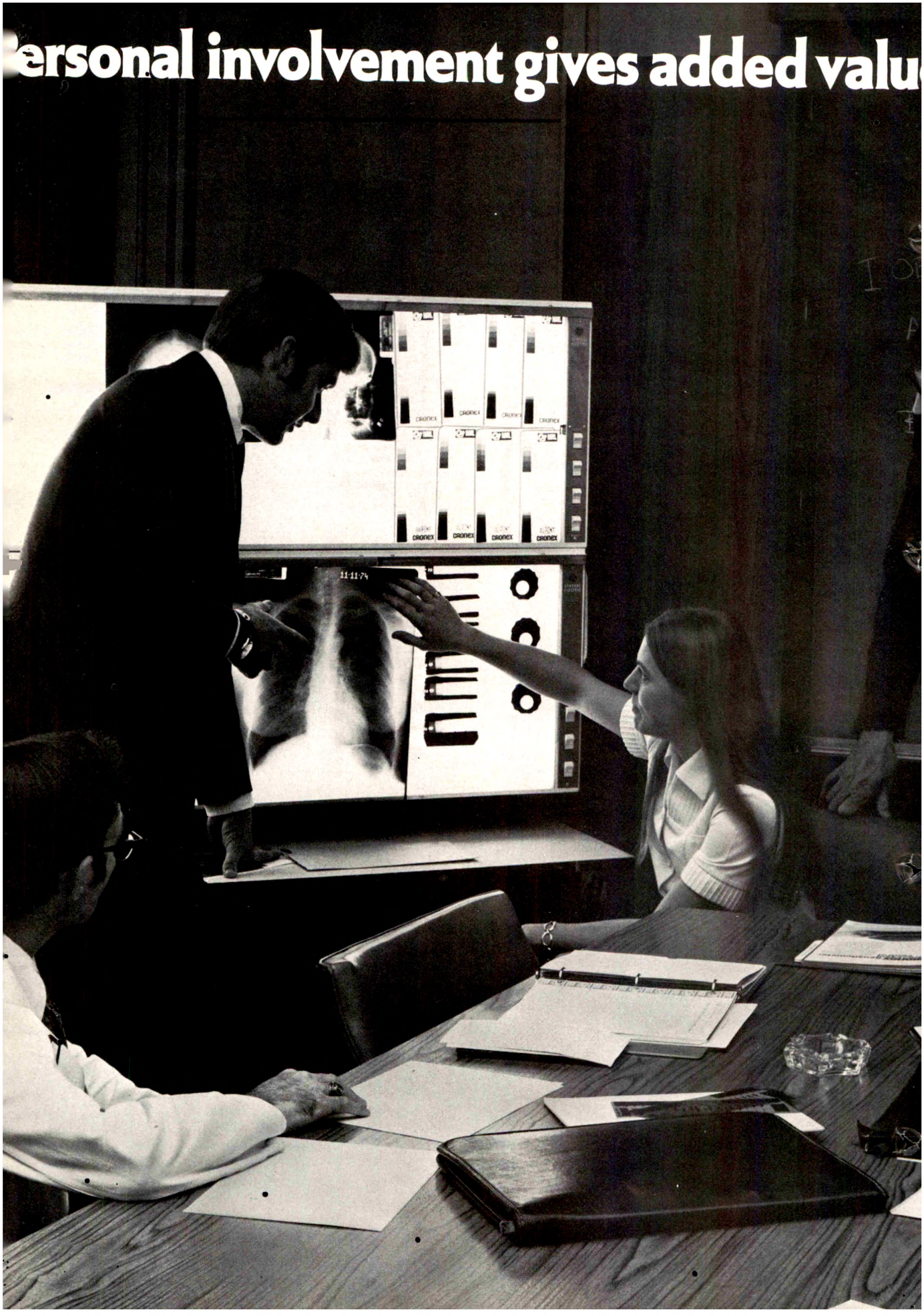
**Is this a coin lesion?
Or something else.**

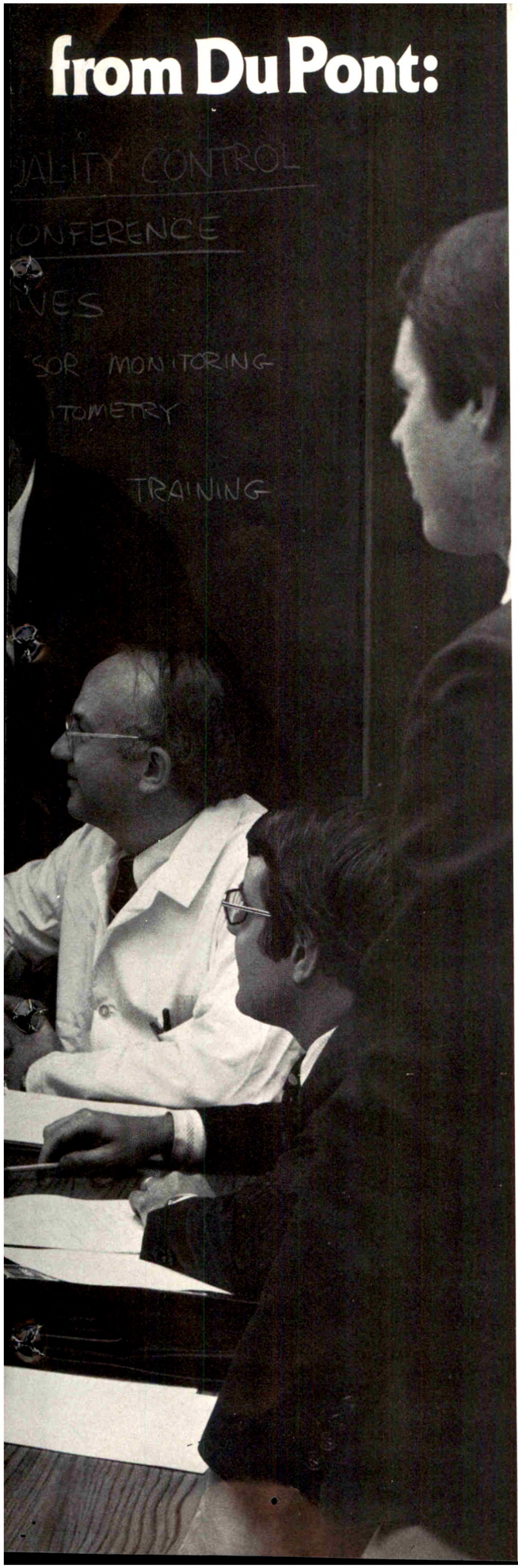


University of Kentucky Medical Center, Department of Diagnostic Radiology

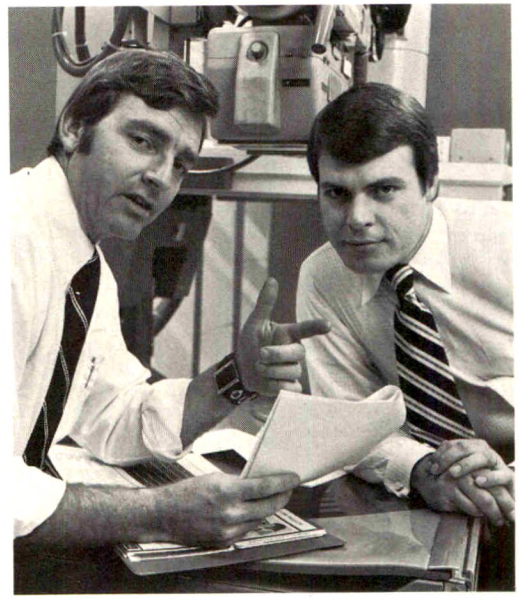
(See GAF® page xxxii)

ersonal involvement gives added valu





"We're saving years of staff time at a large University Hospital with a 5-point radiographic quality control program."



Jay Lynch, Du Pont Territ Manager.

Terry Reagan, Du Pont Medi X-ray Speciali

"Without Du Pont's involvement, it woul have taken years of our own staff work to solve X-ray quality problems caused by variables in processing and imaging techniques." That's a statement from Clive W. Morris, M.D., Associat Professor of Clinical Radiology at the University

But with Du Pont's involvement, Dr. Jam W. Lecky, Professor of Radiology, and his staff have a 5-point program designed to help them identify variables and set priorities for correctio improve imaging techniques; standardize film, screens, chemicals, illuminators and cassettes; conduct preventive maintenance on processors and analyze retakes to keep X-ray quality up to performance standards.

It's all part of Du Pont's commitment: personal involvement and quality in everything we do and sell.

DUPONT MEDICAL X-RAY

Straight talk about **uniform contact**

The* Halsey Comp-I-Sette I *by USR

Uniform contact between film and screen is essential to the diagnostic quality of a radiograph. Check your present cassette. Flex it. Remember that 1 mm of deflection equals a loss of 2 line pairs of resolution.

That is why the Halsey ***Comp-I-Sette I*** has a rigid, welded stainless steel frame with heavy duty hinges and latches. This insures that equal pressure is applied over the entire screen/film interface.

Here are some other outstanding features of the Halsey ***Comp-I-Sette I***:

- ***Highest abrasion resistance available***—exclusive epoxy finish applied electrostatically.
- ***Versatile***—compatible with new automatic film loading system as well as manual handling.
- ***Less induced static electricity***—film drops in flat, without sliding.
- ***Meets new international size standards.***

The straight talk is that the Halsey ***Comp-I-Sette I*** is a ***Premium Cassette at Standard Price.*** For more details, ask your dealer, or write to United States Radium Corporation, 1425-37th Street, Brooklyn, N.Y. 11218.

USR

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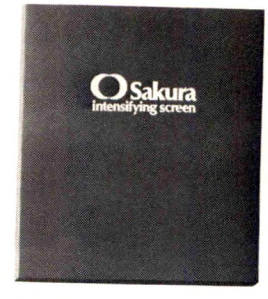
Definition is the difference.

Especially effective in the higher voltage ranges, Sakura X-ray intensifying screens offer higher contrast over a longer life. The advantages are worth considering. Reduced radiation. Minimal loss in resolution. And longer X-ray tube life.

Featuring improved physical and chemical properties, Sakura X-ray screens demonstrate higher surface purity and higher resistance to abrasion. And they are easy to maintain. A plastic coating guards against grime, guaranteeing superior performance after years of continual use.

Sakura X-ray intensifying screens come in popular sizes and are available in three speeds. QD type for excellent contrast without loss of definition. QH type for faster, finer definition. And QS type for fastest, finest definition.

Sakura X-ray intensifying screens. Higher contrast. Longer life. And less expensive than comparable types.



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The Trimax Rare Earth Imaging System is the fastest screen-film combination available. The sys comprises two new rare earth phosphor intensify screens and two specially matched medical X-ray films. The result: a two to four times increase in sp with no loss in information...ideal for special procedures.

Trimax permits maximum use of smaller focal sp Magnification angiography can be done with increased resolution and a higher level of informa Reduction in MaS holds tube loading to a minimu

Trimax minimizes problems with motion blur. Retakes due to movement are minimized. Radia exposure to both patient and staff is greatly redu

Trimax fits your rapid film changer. Both screens and film have been designed for optimal performa in rapid film changing equipment. Trimax films wi unique surface and antistatic properties are avail for all popular film changers.

Trimax calls for no major expenditures. In fact, it might save money by increasing the efficiency your existing equipment. We would be happy to schedule an evaluation of the Trimax System at y convenience. Just put the coupon in the mail; you'll hear from us shortly.

Mr. Robert Appleby
3M Center, St. Paul, Minnesota 55101

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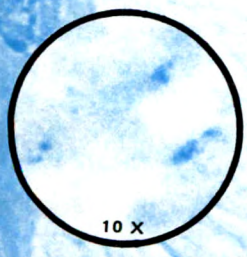
XERORADIOGRAPHY

for early detection of breast cancer



A 55-year-old, gravida-7 woman with no family history of breast cancer. Physical examination of both breasts was normal. Routine xeroradiographic mammography was performed also. Xeroradiograph of the right breast (reproduced approximately actual size) reveals a small cluster of tumor calcifications. Biopsy confirmed very early comedo carcinoma without evidence of metastatic disease. The lesion is so small that it occupies only one medium power field (insert). A radical mastectomy was performed and the patient will receive annual xeroradiographic examination of the remaining breast and right axilla.

Xeroradiograph and caption from
John N. Wolfe, M.D.
Hutzel Hospital, Detroit

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Introduction to Xeroradiographic Mammography
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The
Reason
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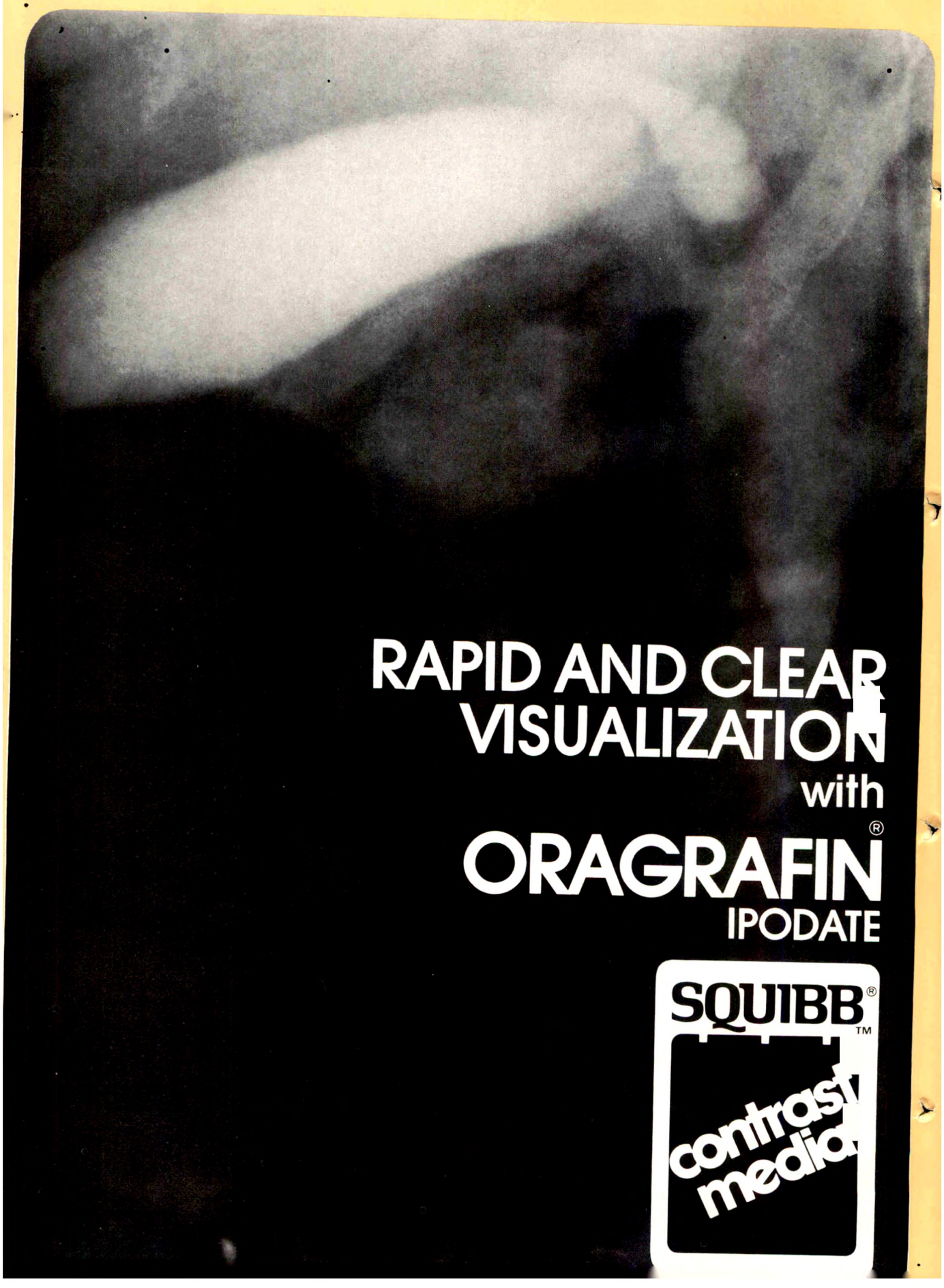
Clear..
Effectiveness


RAPID AND CLEAR
VISUALIZATION
with
ORAGRAFIN®
IPODATE
SQUIBB®
™
contrast
media

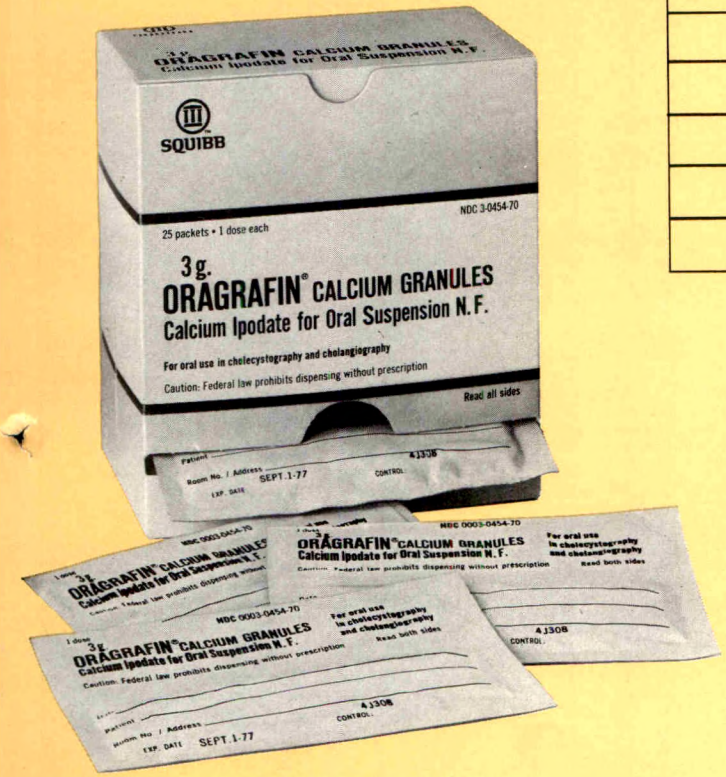
- Visualization of the ducts occurs in 1 to 3 hours and of the gallbladder within 5 hours or less with Oragrafin Calcium Granules (Calcium Ipodate for Oral Suspension N.F.).
- The gallbladder is opacified approximately 10 hours after ingestion of Oragrafin Sodium Capsules (Sodium Ipodate Capsules N.F.).
- Rapid absorption often permits visualization of even poorly functioning gallbladders. An unprepared patient can be studied the same day.
- When patients cannot take capsules, the granules are a convenient, palatable alternative.
- Both salts are usually well tolerated.

Dosage Schedules

FOR ROUTINE OVERNIGHT PROCEDURES Sodium Ipodate Capsules and/or Calcium Ipodate for Oral Suspension			
	Gallbladder Films		Films of the Gallbladder and Biliary Ducts
11 P.M.	6 Capsules (or 12 Capsules)	OR 1 packet of Granules (or 2 packets)	6 Capsules (or 1 packet of Granules, if preferred) and
next morning, 8 A.M.			1 packet of Granules
9 A.M.	Visualization of gallbladder	Visualization of gallbladder	Visualization of ducts and gallbladder
10 A.M.			
11 A.M.			

FOR RAPID, SAME-DAY PROCEDURES Calcium Ipodate for Oral Suspension	
Films of Gallbladder and Ductal System	
8 A.M.	2 packets of Granules
9 A.M.	Visualization of ducts
10 A.M.	Optimal visualization of ducts
1 P.M.	Visualization of gallbladder

See next page for complete prescribing information



ORAGRAFIN®
CALCIUM GRANULES
Calcium Ipodate for Oral Suspension N.F.

ORAGRAFIN®
SODIUM CAPSULES
Sodium Ipodate Capsules N.F.

the reason is clear

ORAGRAFIN®
CALCIUM GRANULES
Calcium Ipodate for Oral Suspension N.F.

ORAGRAFIN®
SODIUM CAPSULES
Sodium Ipodate Capsules N.F.

DESCRIPTION
Oragrafin Calcium Granules (Calcium Ipodate for Oral Suspension N.F.) and Oragrafin Sodium Capsules (Sodium Ipodate Capsules N.F.) are oral radiopaque media for cholangiography and cholecystography. *Calcium* ipodate is available in single-dose foil packets providing 3 g. calcium ipodate as granules dispersed in flavored sucrose. When mixed with water, the granules produce a pleasantly-flavored suspension. *Sodium* ipodate is available in capsules providing 500 mg. sodium ipodate per capsule.

The organically bound iodine content of calcium ipodate and sodium ipodate is 61.7% and 61.4%, respectively.

ACTIONS
Calcium ipodate and sodium ipodate are absorbed from the gastrointestinal tract, excreted by the liver into the bile, and stored and concentrated in the gallbladder. The calcium salt, which is absorbed somewhat faster than the sodium salt, appears in the ducts as early as 30 minutes after ingestion. Optimal concentration of either salt in the hepatic and biliary ducts occurs within 1 to 3 hours after ingestion in nearly all cases; rarely, maximal opacification may be delayed for as much as 5 hours. Adequate opacification of the ducts usually persists for about 45 minutes. The ducts may be visualized in patients who have undergone cholecystectomy.

The gallbladder is optimally opacified approximately 10 hours after ingestion of either salt. Diagnostically adequate filling, however, often takes place within 5 hours or less after ingestion, particularly with the calcium salt. Thus, studies of ducts and gallbladder may be carried out in as little as 5 hours when necessary or desirable. In this connection, it should be pointed out that both salts have been administered to unprepared patients as well as to previously prepared patients, with no significant difference in results.

INDICATIONS
Calcium ipodate and sodium ipodate are indicated for cholecystography. They may also be used for cholangiography, although they are not considered the drugs of choice.

CONTRAINDICATIONS
Both preparations are contraindicated in persons who are hypersensitive to iodate salts.

WARNINGS
A history of sensitivity to iodine *per se* or to other iodinated compounds is not an absolute contraindication to the use of these preparations, but calls for extreme caution in administration.

Usage in Pregnancy
The safety of these preparations for use during pregnancy has not been established; therefore, they should be used in pregnant patients only when, in the judgment of the physician, their use is deemed essential to the welfare of the patient.

PRECAUTIONS
Increasing the dosage above that recommended increases the possibility of hypotension.

Anuria may result when these preparations are administered to patients with combined renal and hepatic disease or severe renal impairment.

Renal toxicity has been reported in a few patients with liver dysfunction who were given oral cholecystographic agents followed by urographic agents. Administration of urographic agents should therefore be postponed in any patient with a known or suspected hepatic or biliary disorder who has recently taken a cholecystographic contrast agent.

Gastrointestinal disorders which interfere with absorption and liver disorders which interfere with excretion may result in non-visualization of the hepatic and biliary ducts and the gallbladder.

Contrast agents may interfere with some chemical determinations made on urine specimens; therefore, urine should be collected before administration of the contrast media or two or more days afterwards.

Thyroid function tests, if indicated, should be performed prior to the administration of these preparations since iodine containing contrast agents may alter the results of these tests.

ADVERSE REACTIONS
Sodium ipodate and calcium ipodate are usually well tolerated. Unwanted effects such as mild and transient nausea, vomiting, or diarrhea sometimes occur but the incidence can be reduced by restricting the dosage to 3 g. and administering only the Granules to patients who may be prone to gastrointestinal reactions. Headache, dysuria, or abdominal pains may occur infrequently as transient disturbances.

Hypersensitivity reactions may include urticaria, serum sickness-like reactions (fever, rash, arthralgia), other skin rashes, and, rarely, anaphylactoid shock. They are more likely to occur in individuals with a history of allergy, asthma, hay fever, or urticaria, and in those who have previously demonstrated hypersensitivity to iodine compounds.

DOSAGE AND ADMINISTRATION
The Capsules should be taken with as little water as possible. They may be swallowed in rapid succession or slowly over the course of ½ hour or more, depending on patient preference.

The Granules should be stirred vigorously into a small amount of water (¼ glass or less) and swallowed immediately. If lukewarm rather than cold water is used, the patient will find the suspension quite palatable and there will be less likelihood of nausea.

A total dose of 6 g. (12 Capsules or 2 packets of Granules) per 24 hour period should not be exceeded.

When an upper gastrointestinal barium study is scheduled, prior administration of either salt will not interfere. Either salt may also be used in conjunction with an intravenous cholangiographic agent.

The recommended dosage and patient preparation for cholangiography and/or cholecystography are as follows:

Routine Cholecystography
On the day before the examination, the patient may, if feasible, eat a high-fat lunch so that the gallbladder will evacuate and refill with opacified bile. The evening meal, which should be light and fat-free, should be eaten 2 hours before the contrast medium is taken. The contrast agent should be administered 10 to 12 hours before the roentgenologic examination. The usual dose is 6 Capsules (providing 3 g. sodium ipodate), although up to 12 Capsules may be given; 1 to 2 packets of Granules may be used instead. No other food or drink except small amounts of water should be taken before the examination. A mild laxative or other cathartic may be given, but is not generally necessary.

Roentgenologic examination of the gallbladder is performed the next morning in the usual manner, employing the standard positions. An immediate study of the wet films will determine any need for repositioning of the patient. Administration of a fat meal or other cholecystokinetic agent provides good dynamic studies of gallbladder function. If a barium meal examination is also to be performed, the fat meal may be given simultaneously with or following the barium meal. Gallbladder function studies are then made after the barium meal examination is completed.

Cholangiography
No prior preparation of the patient is necessary. For this procedure, the Granules should be used. They are administered one hour before cholangiography. Although only one packet (providing 3 g. calcium ipodate) may suffice, two packets (6 g.) are usually necessary for visualization of the ducts.

Beginning one hour after ingestion of the Granules, and at 15-minute intervals thereafter, films should be taken, preferably with the aid of tomography or laminography. With proper positioning of the patient, residual contrast medium in the gastrointestinal tract is not likely to be a problem, particularly with the more rapidly absorbed calcium salt. Optimal visualization of the ducts is generally achieved between 1½ and 2½ hours after ingestion of the medium.

Combined Cholangiography and Cholecystography
Preparation of the patient and administration of dosage should be as outlined under *Routine Cholecystography*, with a dosage of 6 Capsules or, if preferred, one packet of Granules taken on the evening before examination. On the following morning, one hour before the examination, one additional packet of Granules should be administered.

Beginning one hour after the morning dose, and at 15-minute intervals thereafter, films should be made, preferably with the use of tomography or laminography. While the gallbladder will have concentrated the evening dose of the medium, the additional morning dose will provide opacification of the ducts, and simultaneous visualization may be achieved. When satisfactory films have been obtained, a fat meal may be administered to provide gallbladder function studies.

Rapid Combined Cholangiography and Cholecystography
No prior preparation of the patient is necessary. On the day of examination, the patient should be given two packets of Granules. Beginning one hour later, examination of the ducts should be performed as described under *Cholangiography*. If the gallbladder has not opacified sufficiently by the time the cholangiographic examination is completed, further cholecystograms may be taken after 2 or 3 hours. Dynamic studies of gallbladder function may be carried out in the usual manner.

HOW SUPPLIED
Oragrafin Calcium Granules (Calcium Ipodate for Oral Suspension N.F.), single-dose foil packets; boxes of 25 packets (3 g. calcium ipodate per packet). Oragrafin Sodium Capsules (Sodium Ipodate Capsules N.F.), boxes of 25 vials (6 capsules each vial); bottles of 100; and Unimatic® single-dose packages of 100.

SQUIBB®

The Reason

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Since this ad first ran **RAREX** has sold thousands

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United States Radium Corporation, 1425-37th Street, Brooklyn, N.Y. 11218.

USR

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Something else.

What you see in the radiograph on page **xvii** *is just a coin placed on the patient's posterior right hemithorax. It does bear a resemblance to the coin lesion produced by a benign solitary lesion, perhaps a hamartoma or tuberculoma.*

You naturally suspected "something else" rather than a coin lesion: the increased opacity of the posterior rib easily places the "lesion" behind the rib.

It is such consistently superior resolution which has made GAF® x-ray film the "film of choice" for thousands of radiologists for over 75 years.

As America's oldest manufacturer of film, photographic papers and chemicals, GAF has helped advance the art of radiography, particularly with increasingly effective x-ray film:

Our HR2000 film for moderate scale contrast and minimum base fog is ideal for resolving minute lesions and fractures.

HR3000 film is a high-contrast fine grain film well-suited for short exposure angiography.

And our HR1000 film provides optimum image and exposure latitude, and long-scale contrast.

Find out also about GAF Liquamat® 90 all-liquid chemistry. Fast and clean-working, it's also color-coded for quality assurance.

We've even produced a series of six teaching courses (utilizing overhead transparencies) used in numerous hospitals today.

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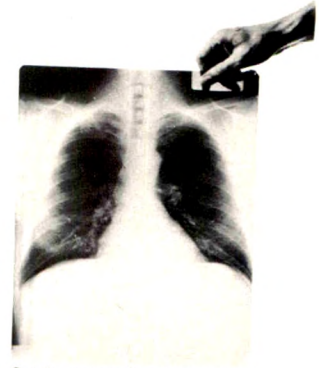


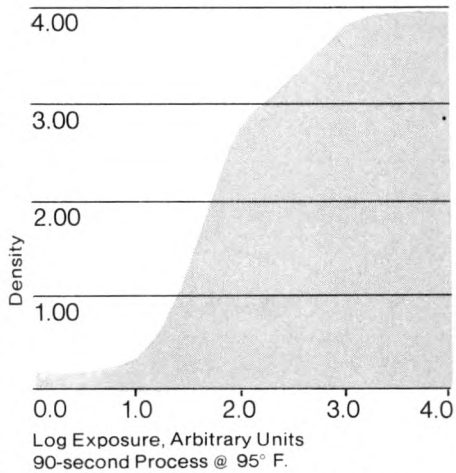
See for yourself: ask your local GAF x-ray representative to demonstrate the consistently high quality of GAF products. Or, if you wish, write GAF Corporation, X-Ray Marketing, 140 West 51st Street, New York, New York 10020.

More information from high-density areas

The shoulder area of the characteristic curve shows that extended contrast is available in the high-density region. This "soft" shoulder extends the latitude of the film while maintaining the contrast needed for visualization of soft-tissue areas.

More processing latitude

A high degree of tolerance to variations in processing machines and chemistry is built into 3M Type R film. A medium-speed film for use with Calcium Tungstate or UV-emitting screens, Type R also offers improved anti-static characteristics.



Characteristic curve of Type R film

Quality Plus Service

Two support organizations, Litton Medical Systems and the 3M Company, stand behind Type R film. It is your assurance of a fine quality product—along with the services of two teams of expertly trained specialists in film and processing.

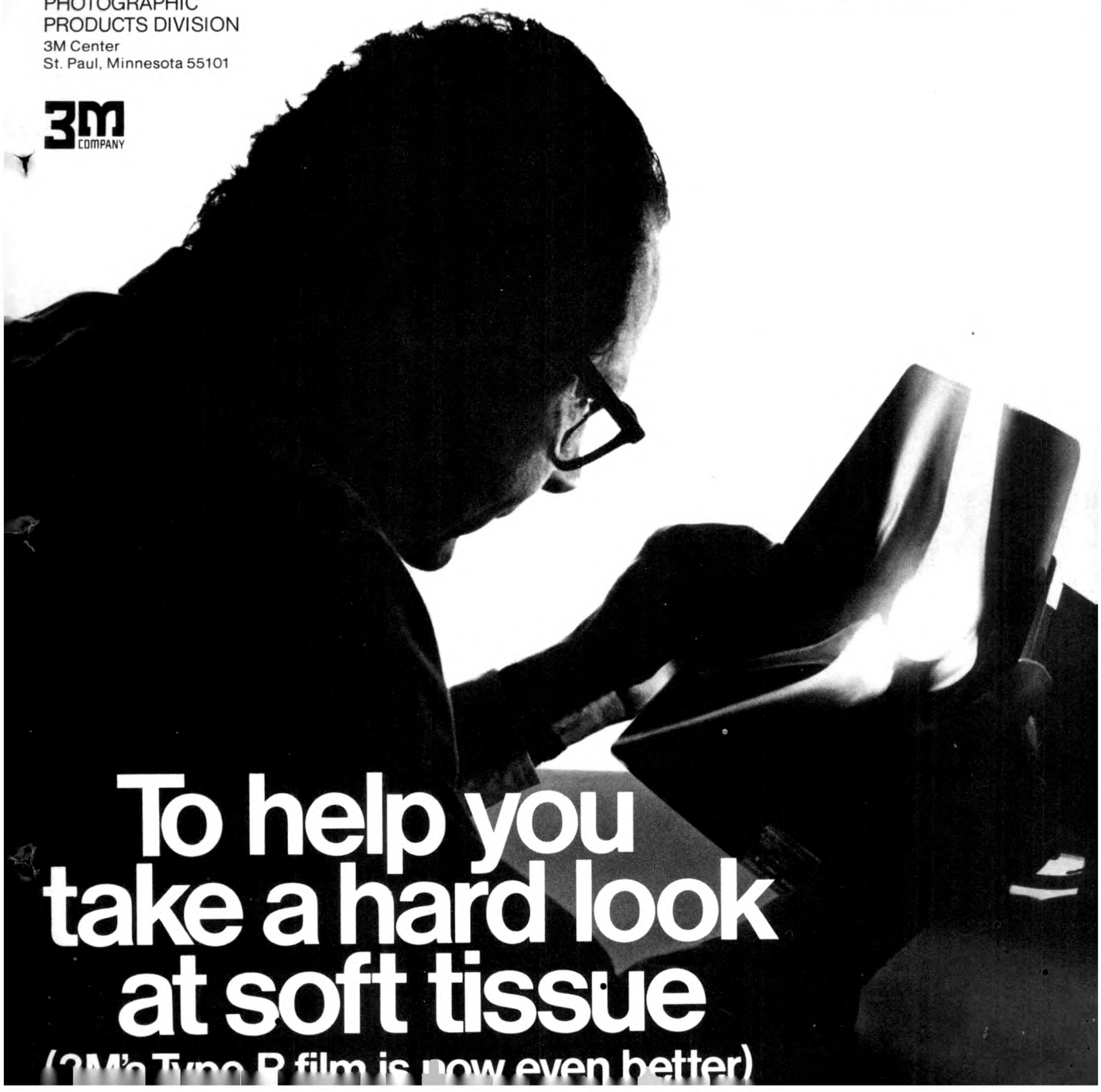
Equals Value

Your film business is important to us. It has been and will continue to be our policy to offer the best product and service available. Before you make another film purchase, check us out. It could be to your advantage.

Litton Medical Systems

PHOTOGRAPHIC
PRODUCTS DIVISION
3M Center
St. Paul, Minnesota 55101

3M COMPANY



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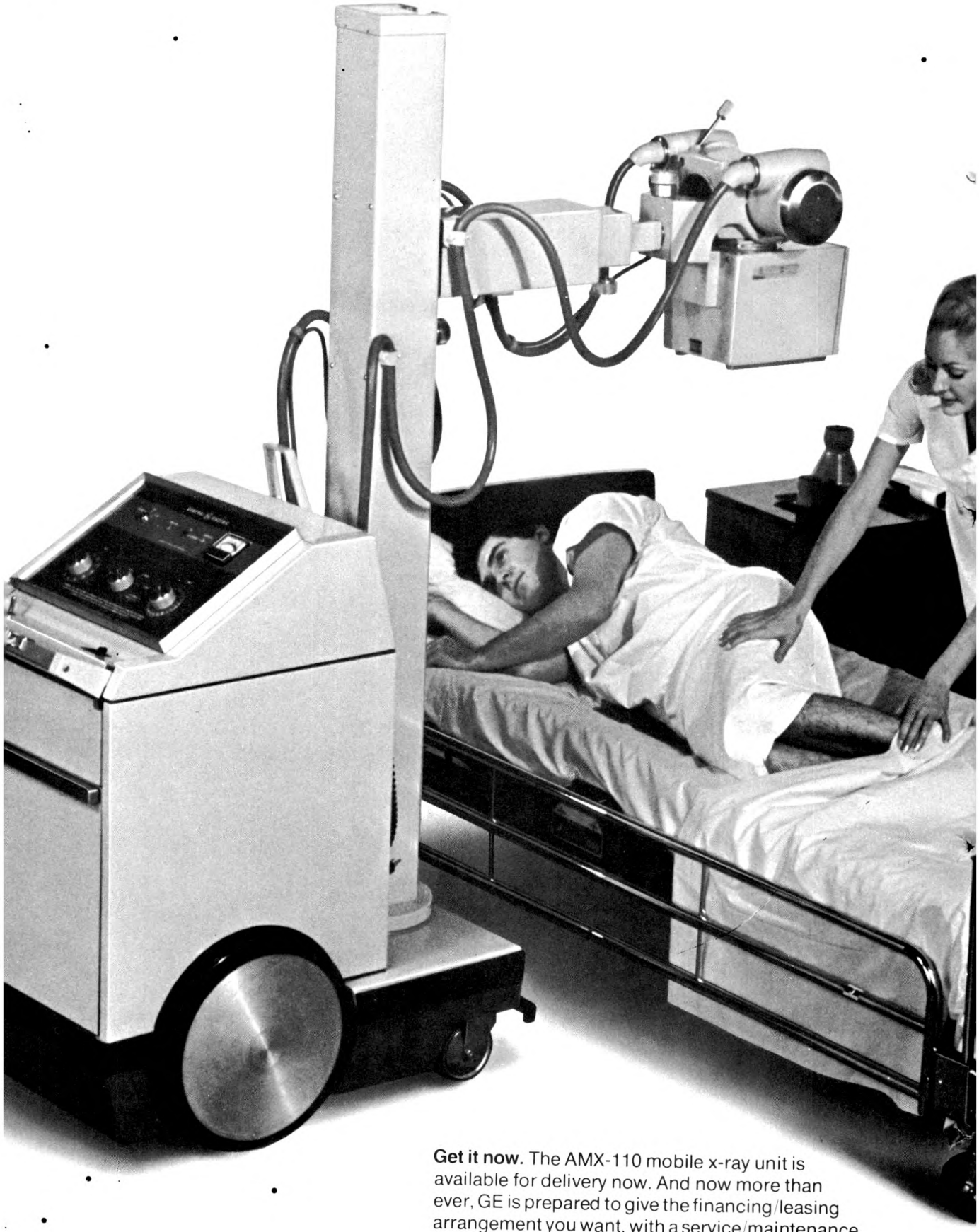

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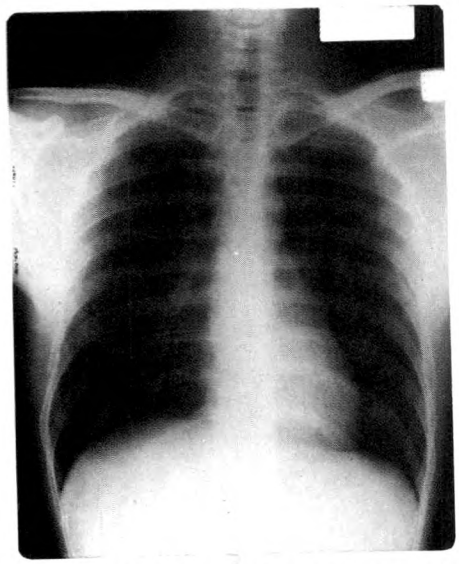

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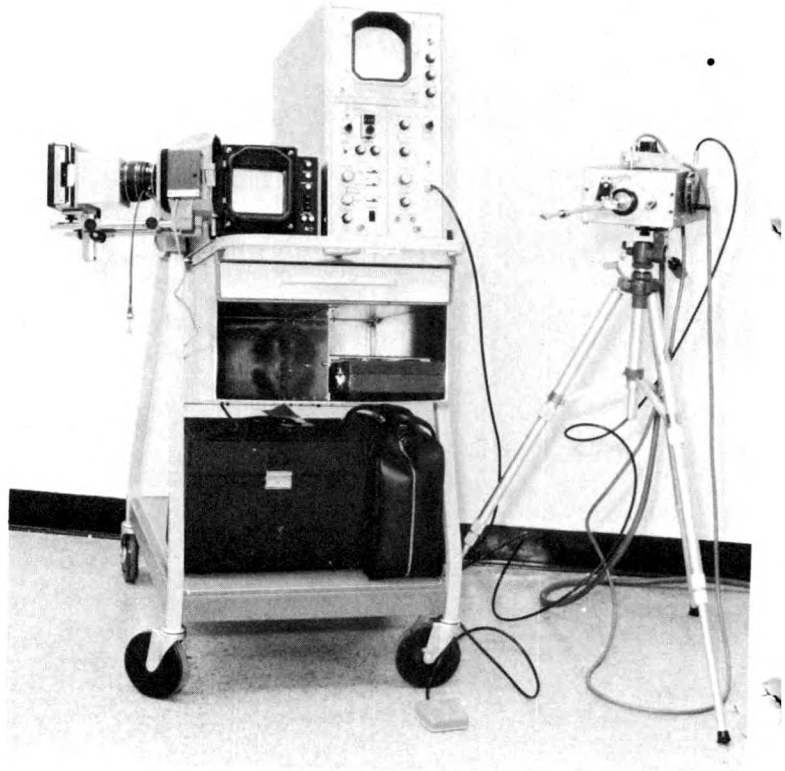

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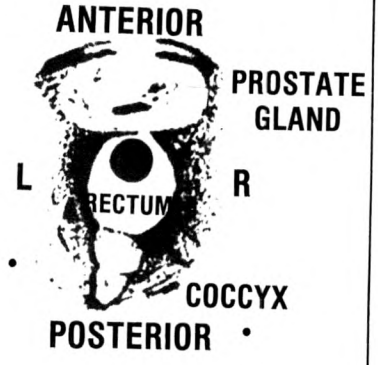

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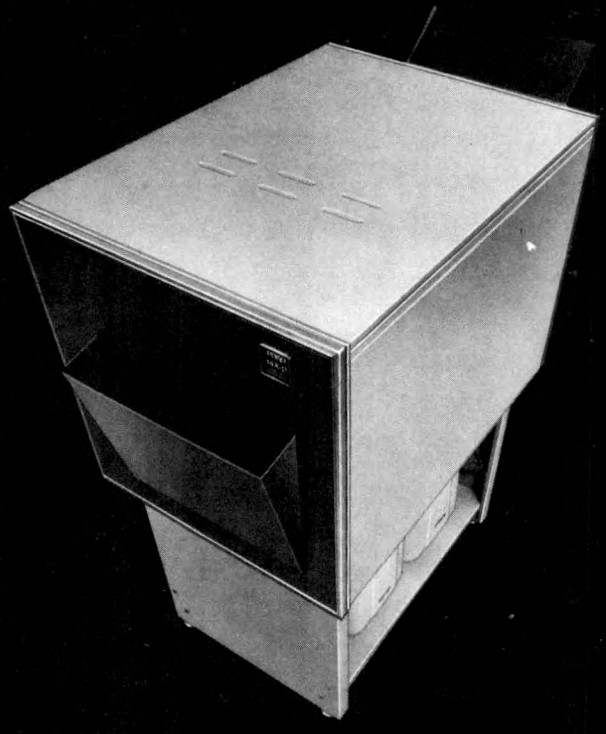

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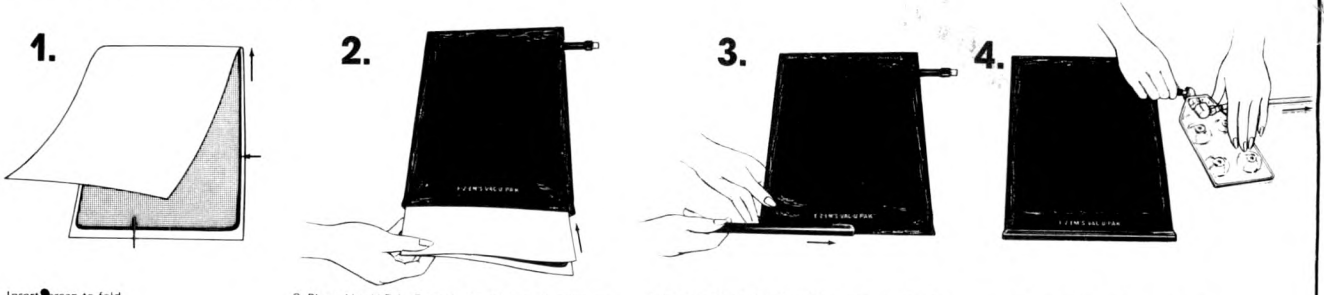

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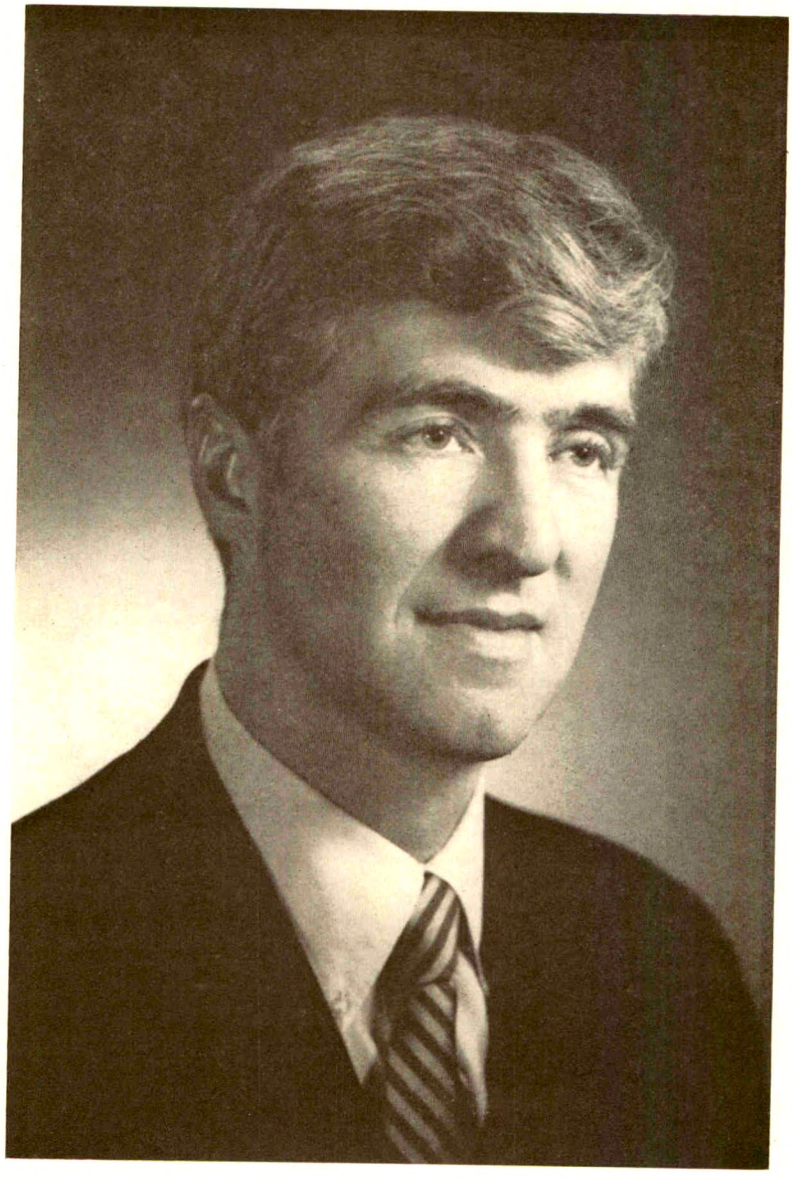


JOSEPH P. WHALEN, M.D.
HICKEY LECTURER, 1975

THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 125 SEPTEMBER, 1975 NO. 1

ANATOMY OF THE COLON*

GUIDE TO INTRA-ABDOMINAL PATHOLOGY

HICKEY LECTURE, 1975

By JOSEPH P. WHALEN, M.D.
NEW YORK, NEW YORK

It is indeed a great honor to be here this evening delivering a lecture named for such an outstanding contributor to radiology as Dr. Preston M. Hickey. These contributions are obviously too numerous to elaborate, but can be characterized as being "basic" to all aspects of radiology, both academic and organizational. Their effects are still felt today. The topic I have chosen to discuss is an anatomic topic, related to radiology of the abdomen. Hopefully, this also will prove to be basic information which will meet the test of time, albeit in a less significant manner.

The normal anatomic relationships of the colon as applied to radiographic anatomy have been previously described.[4] *This work was done by Dr. Paul A. Riemenschneider and me, initiated during my residency with him. This will only briefly be reviewed and emphasis this evening will be primarily on illustrating the pathologic alteration of this normal anatomy. This likewise was inspired and done in part with Dr. Riemenschneider and his contribution is gratefully acknowledged.*

THE colon is an organ ideally suited for the primary classification of abdominal masses since it traverses most of the abdominal cavity and has both intra- and extraperitoneal relationships. The ascending and descending segments are primarily posterior without a mesentery and are related to the extraperitoneal fat.[5] The transverse colon and sigmoid colon, however, are on a mesentery, are anterior, and are more central, related to central intraperitoneal and anterior extraperitoneal organs (Fig. 1). A fact not commonly appreciated is the difference in depth of portions of both the right colic flexure and the left colic flexure. The very lateral aspect of the right colic flexure is quite far posterior and related to the posterior portion of the liver and extraperitoneal organs such as the kidney. As the colon turns medially, it passes anterior over the right kidney and the descending duodenum and then assumes a mesentery. It comes into relationship with the more anterior structures such as the gallbladder and the anterior visceral

* Presented at the Meeting of the Wayne County Medical Society, the Wayne State University College of Medicine and the Michigan Radiological Society, Detroit, Michigan, March 6, 1975.
From the Department of Radiology, Cornell University Medical College, The New York Hospital, New York, New York.

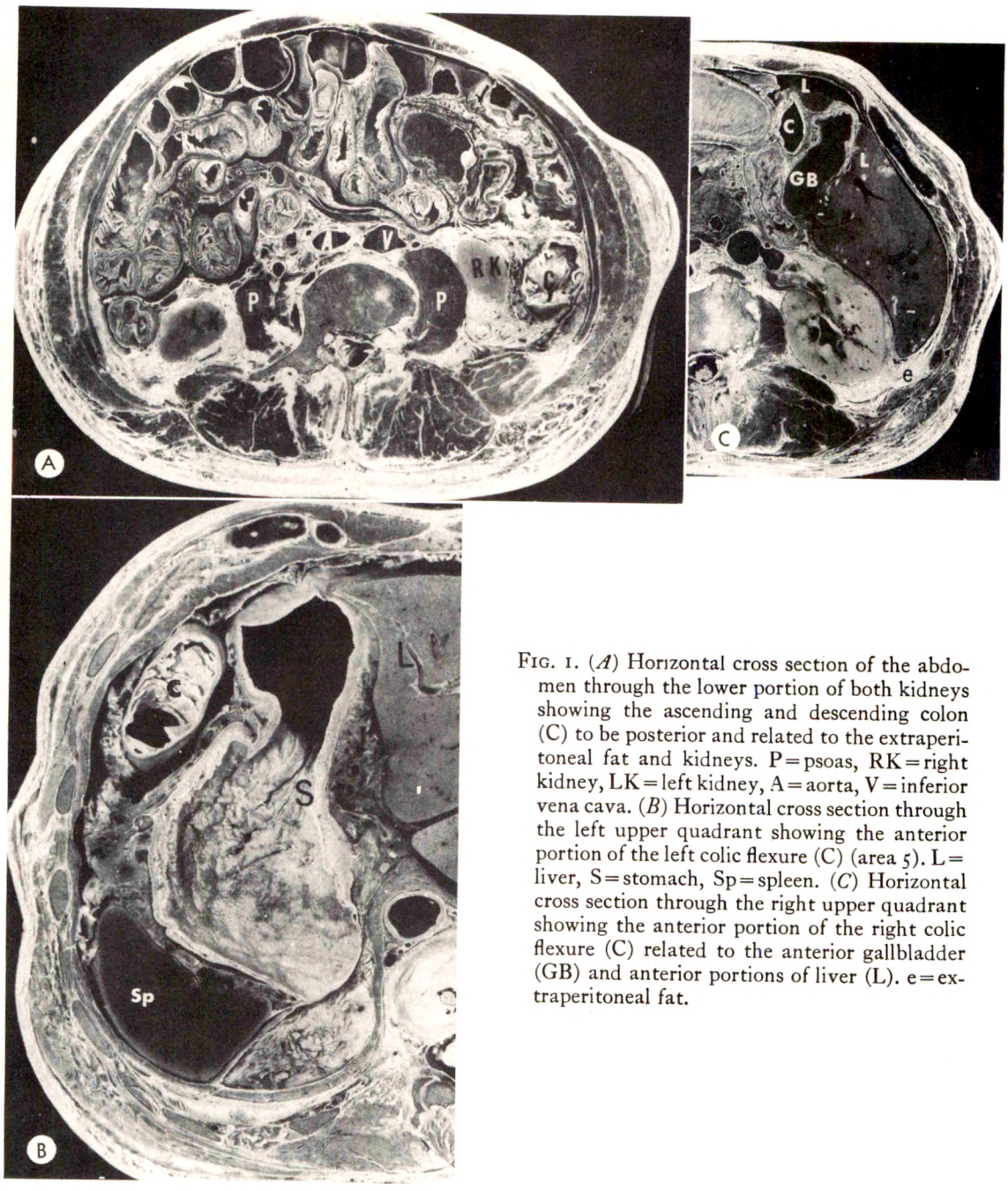


FIG. 1. (*A*) Horizontal cross section of the abdomen through the lower portion of both kidneys showing the ascending and descending colon (C) to be posterior and related to the extraperitoneal fat and kidneys. P = psoas, RK = right kidney, LK = left kidney, A = aorta, V = inferior vena cava. (*B*) Horizontal cross section through the left upper quadrant showing the anterior portion of the left colic flexure (C) (area 5). L = liver, S = stomach, Sp = spleen. (*C*) Horizontal cross section through the right upper quadrant showing the anterior portion of the right colic flexure (C) related to the anterior gallbladder (GB) and anterior portions of liver (L). e = extraperitoneal fat.

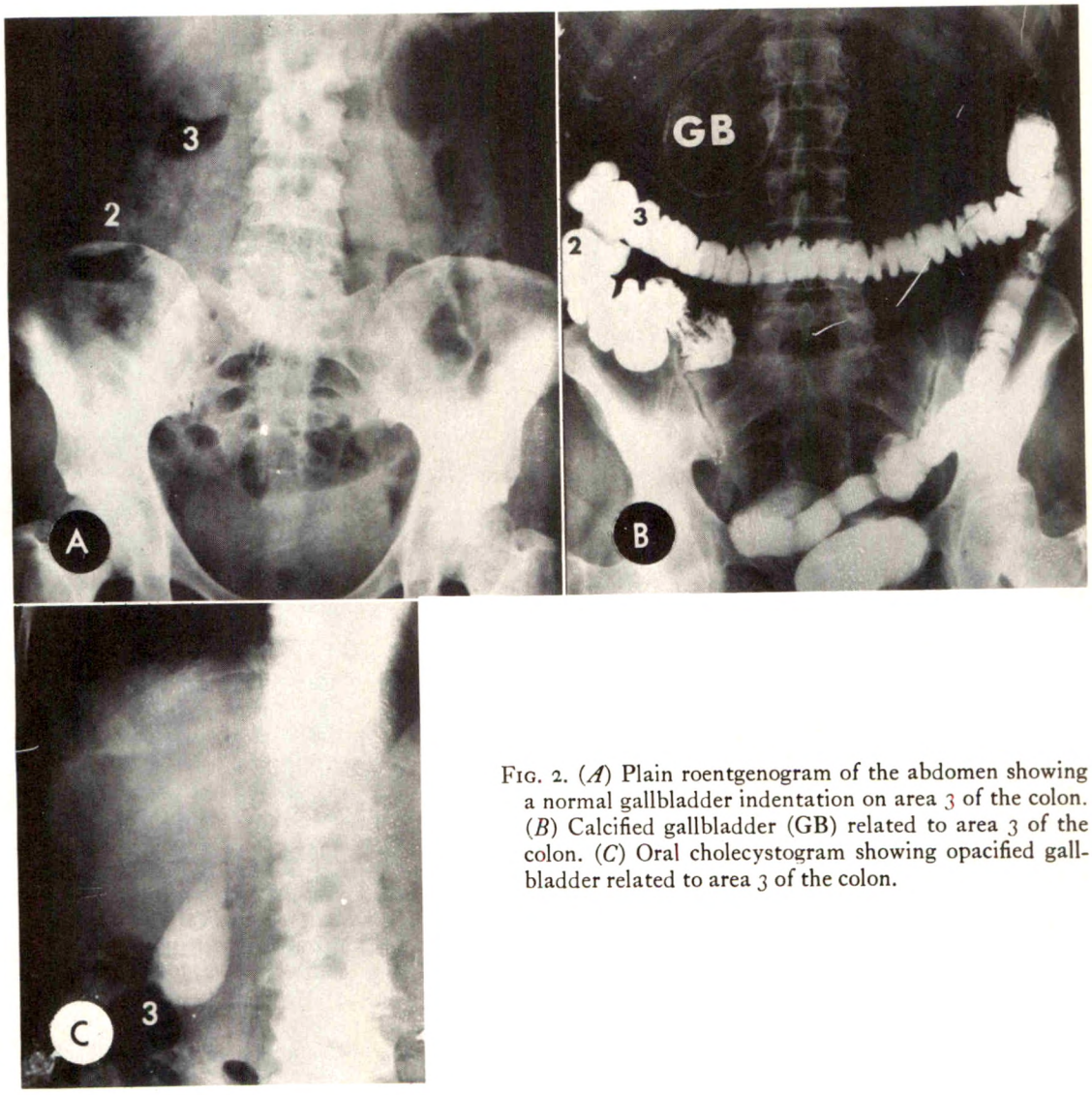


Fig. 2. (*A*) Plain roentgenogram of the abdomen showing a normal gallbladder indentation on area 3 of the colon. (*B*) Calcified gallbladder (GB) related to area 3 of the colon. (*C*) Oral cholecystogram showing opacified gallbladder related to area 3 of the colon.

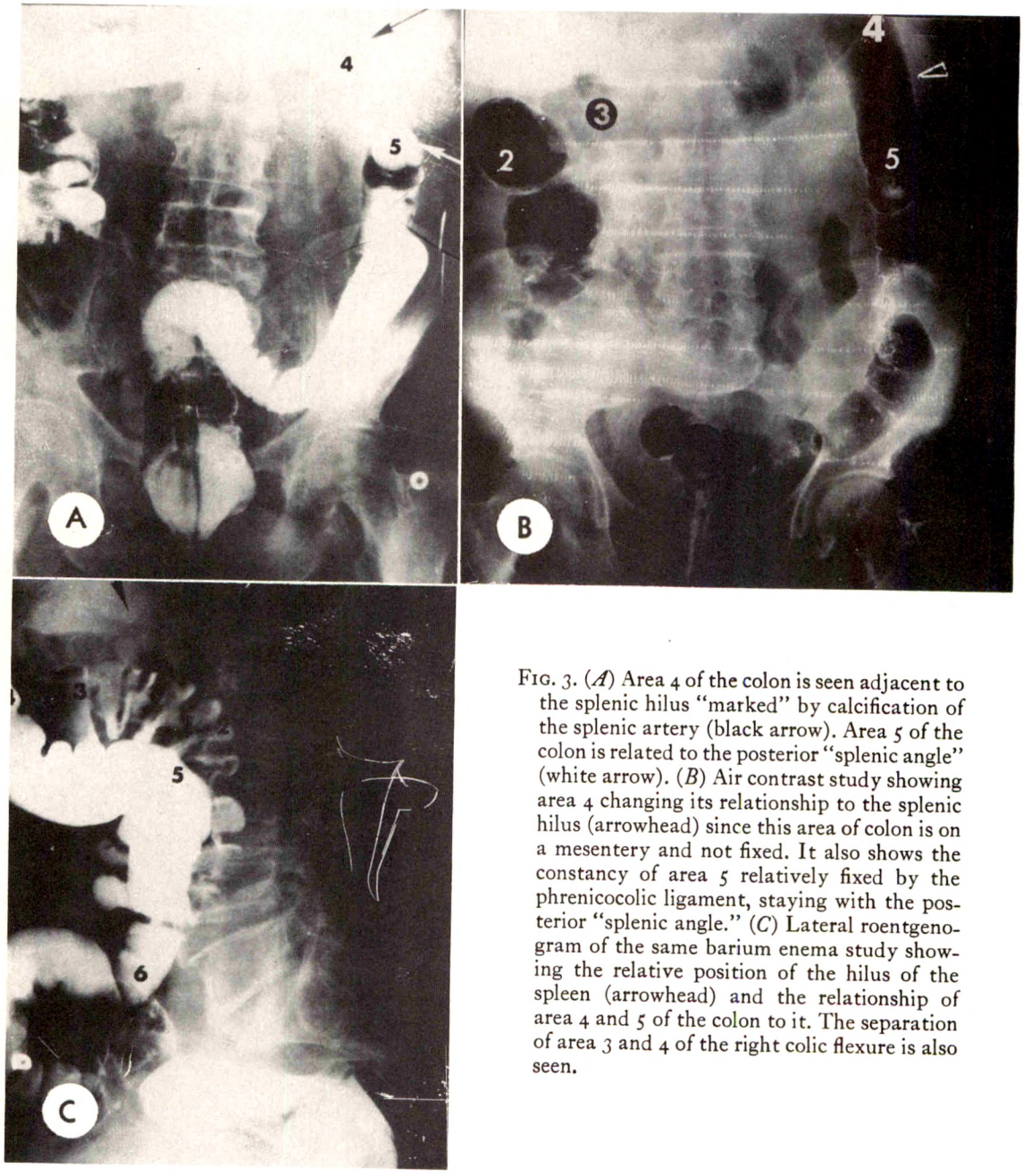


FIG. 3. (*A*) Area 4 of the colon is seen adjacent to the splenic hilus "marked" by calcification of the splenic artery (black arrow). Area 5 of the colon is related to the posterior "splenic angle" (white arrow). (*B*) Air contrast study showing area 4 changing its relationship to the splenic hilus (arrowhead) since this area of colon is on a mesentery and not fixed. It also shows the constancy of area 5 relatively fixed by the phrenicocolic ligament, staying with the posterior "splenic angle." (*C*) Lateral roentgenogram of the same barium enema study showing the relative position of the hilus of the spleen (arrowhead) and the relationship of area 4 and 5 of the colon to it. The separation of area 3 and 4 of the right colic flexure is also seen.

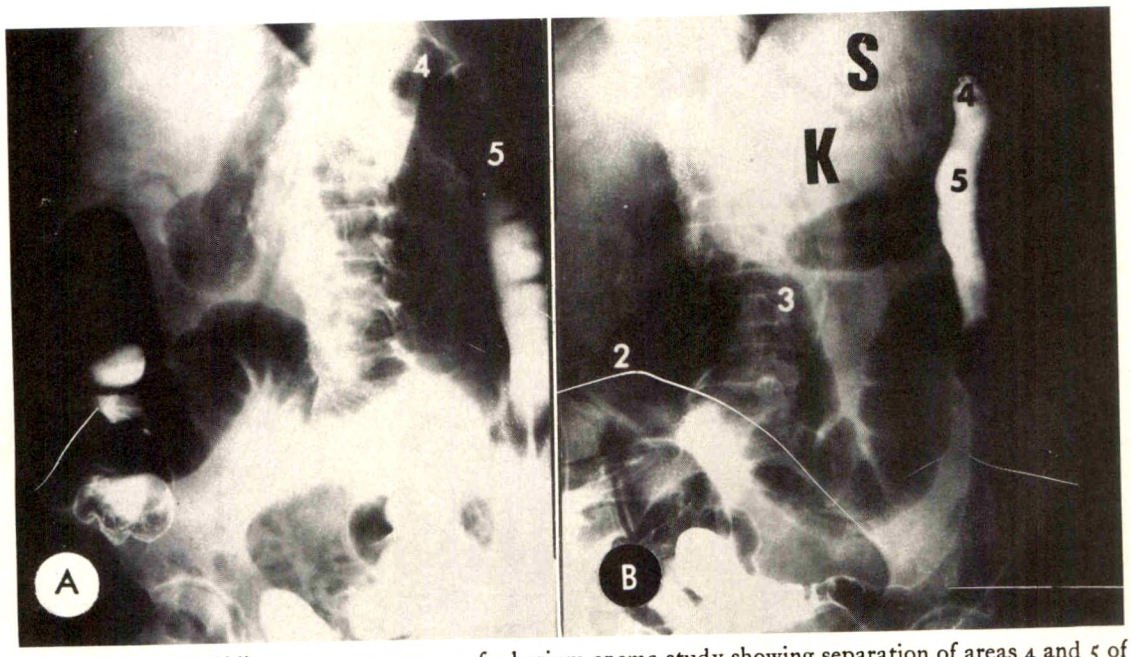


Fig. 4. (*A* and *B*) Oblique roentgenograms of a barium enema study showing separation of areas 4 and 5 of the colon. In *B*, area 5 can be seen related to kidney and area 4 to anterior spleen. K = kidney, S = spleen.

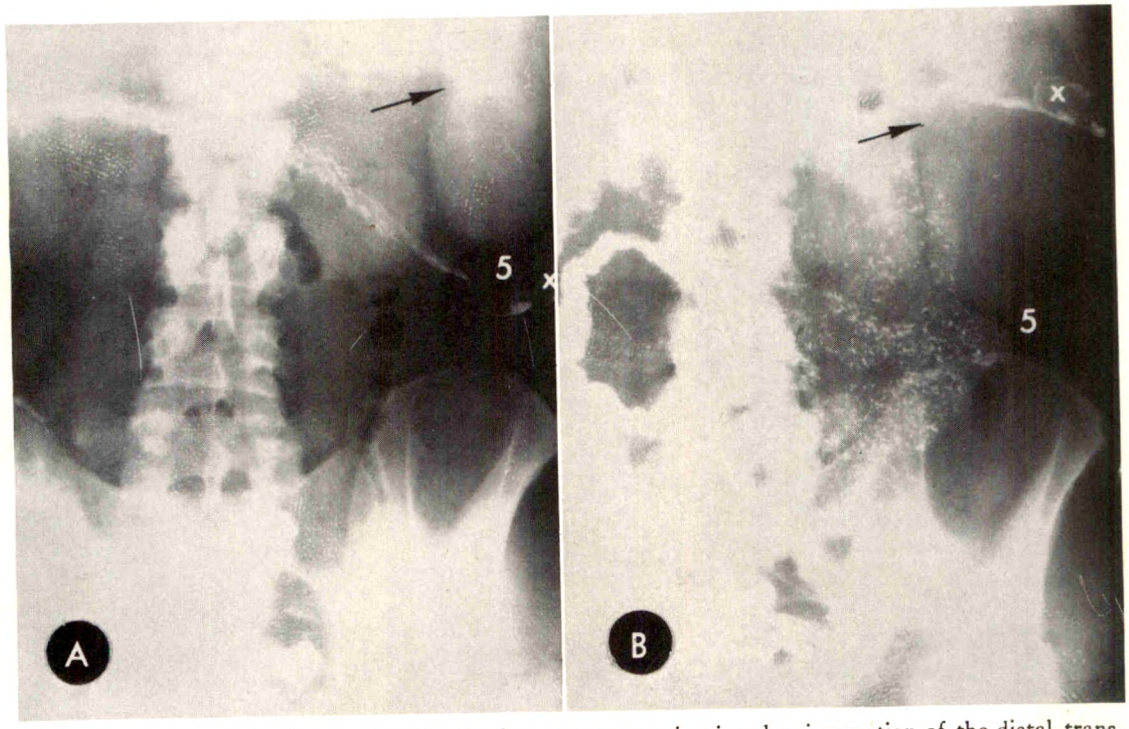


Fig. 5. Roentgenograms of postevacuation barium enema examination showing motion of the distal transverse colon which can be identified by a polyp in its lumen (X). The varying relationship of this area of colon to the splenic hilus (arrow) is apparent. Area 5 which is air-filled is seen constantly related to the enlarged posterior "splenic angle."

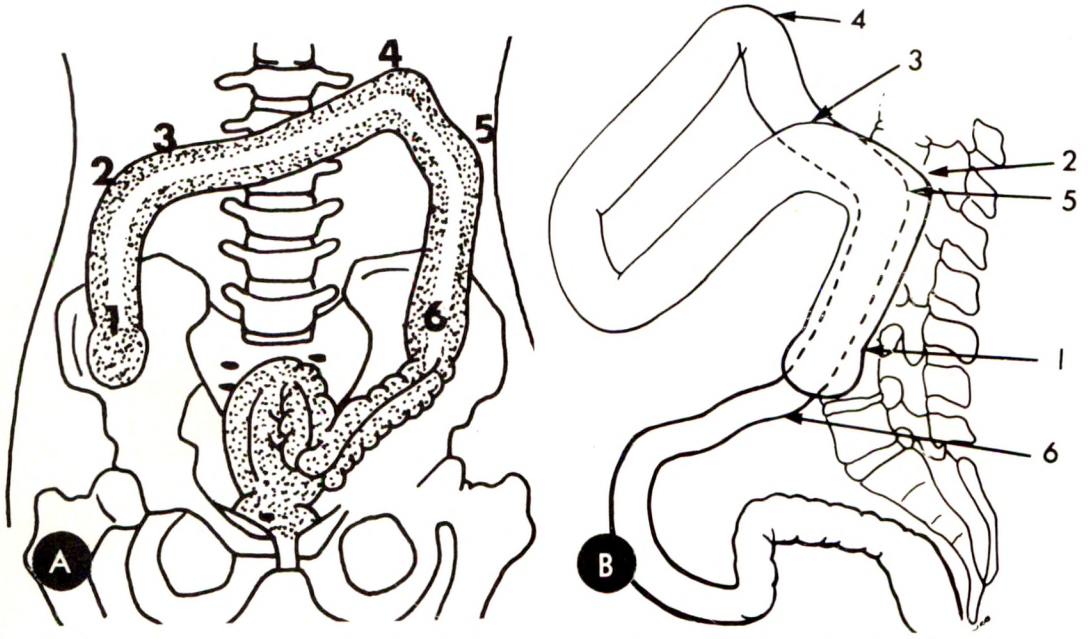


FIG. 6. Diagrammatic representation of colonic anatomy in the (*A*) frontal and (*B*) lateral projections.

surface of the liver. We have designated these areas as 2 and 3, respectively (Fig. 2; and 3). The transverse colon then continues, assuming a longer mesentery, and ends in the area of the splenic flexure, the area of the phrenicocolic ligament. At this point the colon is again posterior and related to the posterior structures such as the left kidney and the posterior aspects of spleen. What has generally been accepted roentgenologically as the splenic flexure, that area on a roentgenogram which shows marked angulation, in reality is merely a portion of the distal transverse colon and is quite far anterior. This portion of colon is related to the anterior portion of spleen and the greater curvature of stomach.[1,2,4] For convenience, we have labelled these areas 5 and 4, respectively (Fig. 4; and 5).

A lateral roentgenogram during a barium enema examination clearly shows the marked separation of these areas of the flexures of the colon. Figure 2 shows the gallbladder's intimacy with area 3. This is further illustrated on a barium enema examination in the filled and air contrast

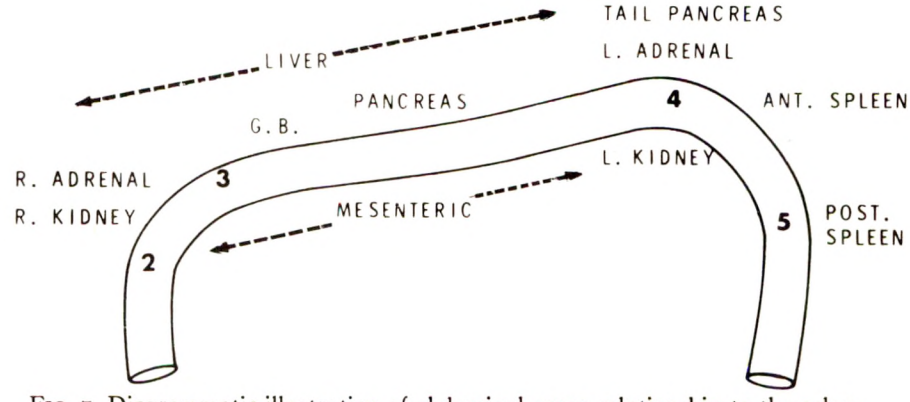


FIG. 7. Diagrammatic illustration of abdominal organ relationship to the colon.

states as these areas relate to a calcified splenic artery, which marks the hilus of the spleen (Fig. 3, *A* and *B*). The lateral barium enema roentgenogram shows the very posterior position of both area 2 and area 5 (Fig. 3*C*). This is diagrammatically represented in Figure 6. The insertion of the transverse mesocolon is also of prime importance since it separates those organs and masses of organs which are supra- and inframesocolic and therefore project above or below the colon (Fig. 7).

Right Renal Masses. Since the upper two-thirds of the right kidney are supramesocolic, the majority of right renal lesions will depress the colon, and specifically area 2 of the colon, and displace the colon forward (Fig. 8; and 9). If the renal lesion arises in the inferior third of the right kidney, it will

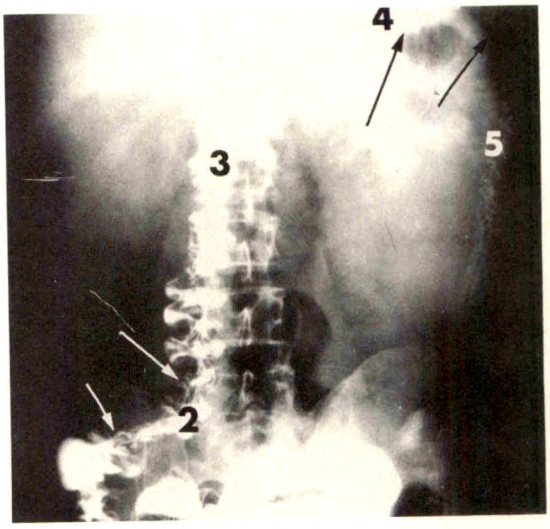


Fig. 8. Bilateral renal enlargement in polycystic disease showing the right kidney depressing the right colic flexure, specifically, area 2.

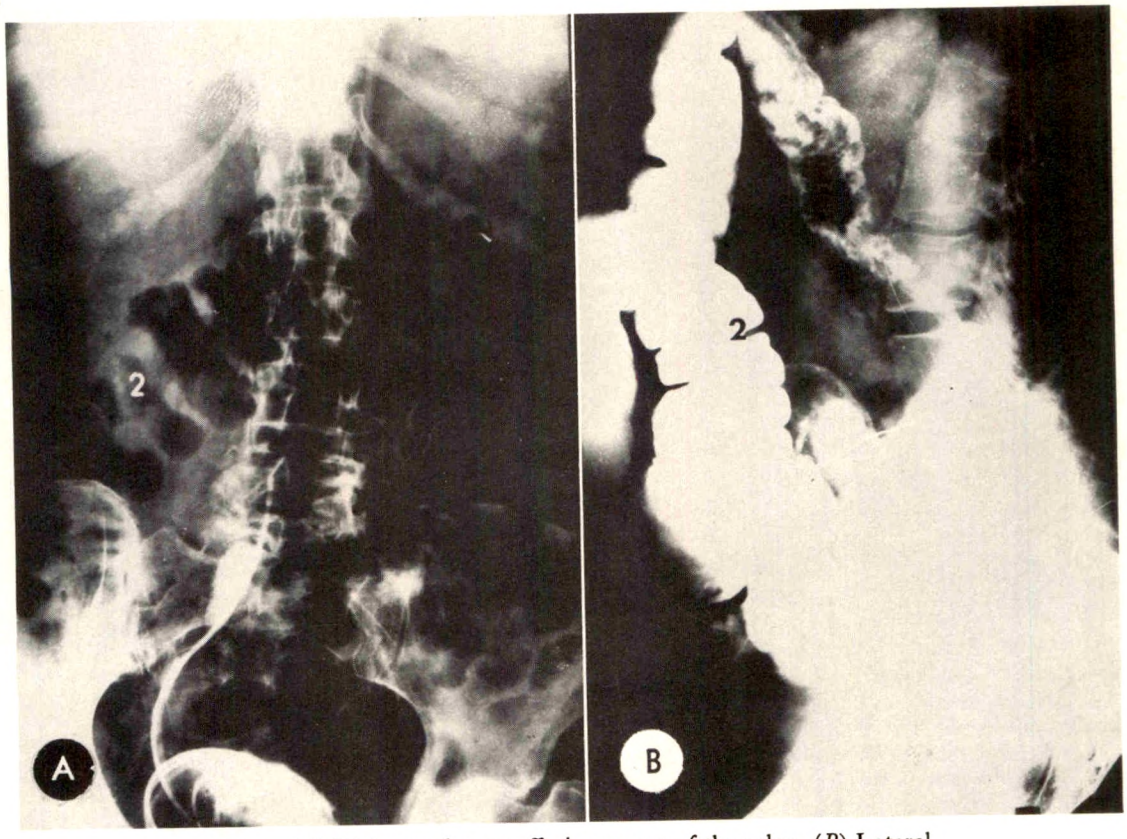


Fig. 9. (*A*) Right renal mass effacing area 2 of the colon. (*B*) Lateral roentgenogram shows the mass to be retrocolic.

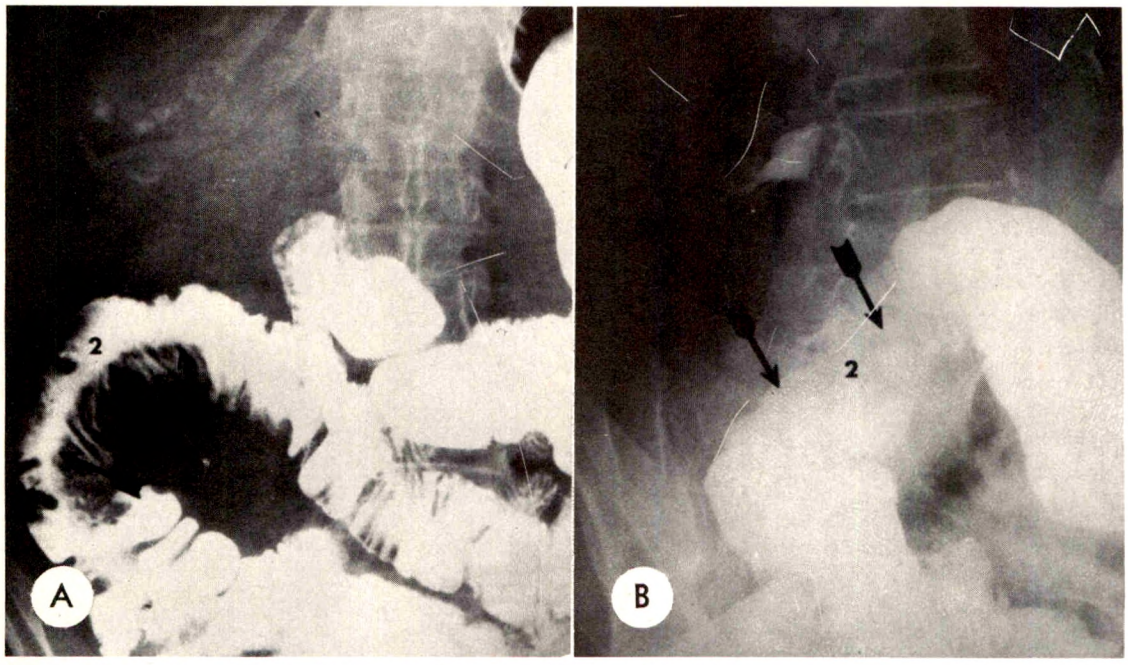


FIG. 10. Mass arising from the lower pole of the right kidney. Although the mass is inframesocolic, it involves area 2 of the colon (*A*) but its retrocolic nature excludes a peritoneal mesenteric cyst (*B*).

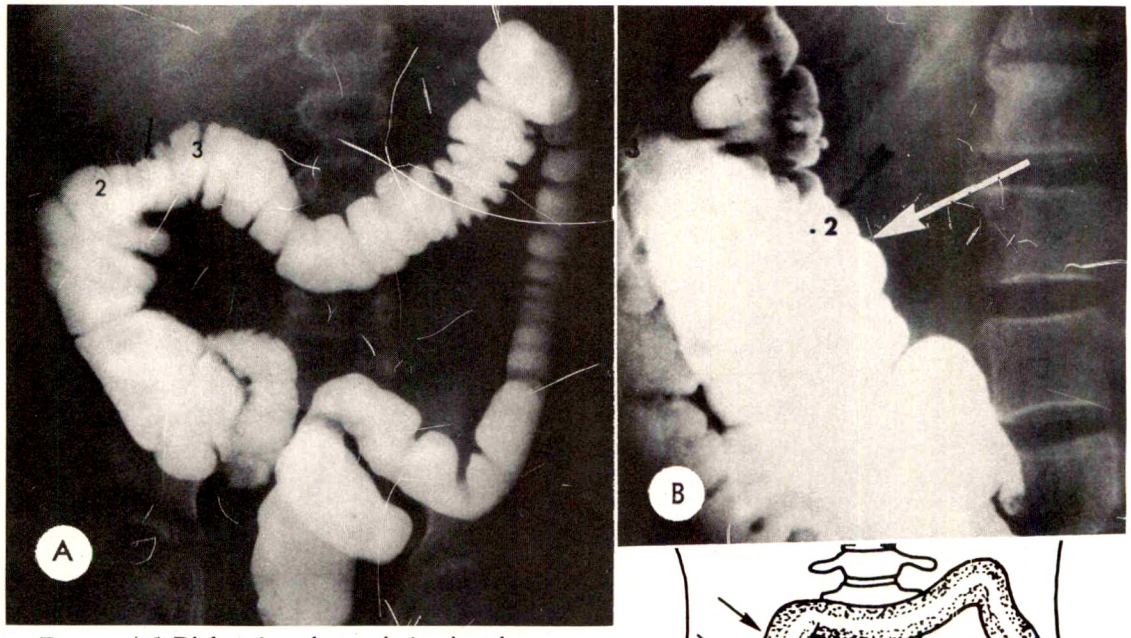
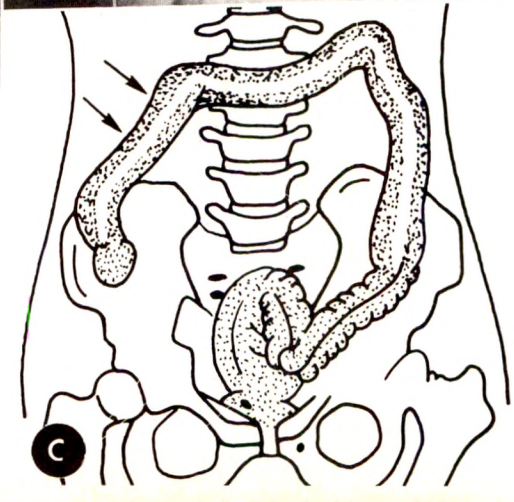


FIG. 11. (*A*) Right adrenal mass indenting the more posterior aspect of the right colic flexure and displacing the right kidney inferiorly (arrow). (*B*) The displaced kidney pushes the colon forward (area 2) (arrow). (*C*) Diagrammatic representation of displacement of area 2.



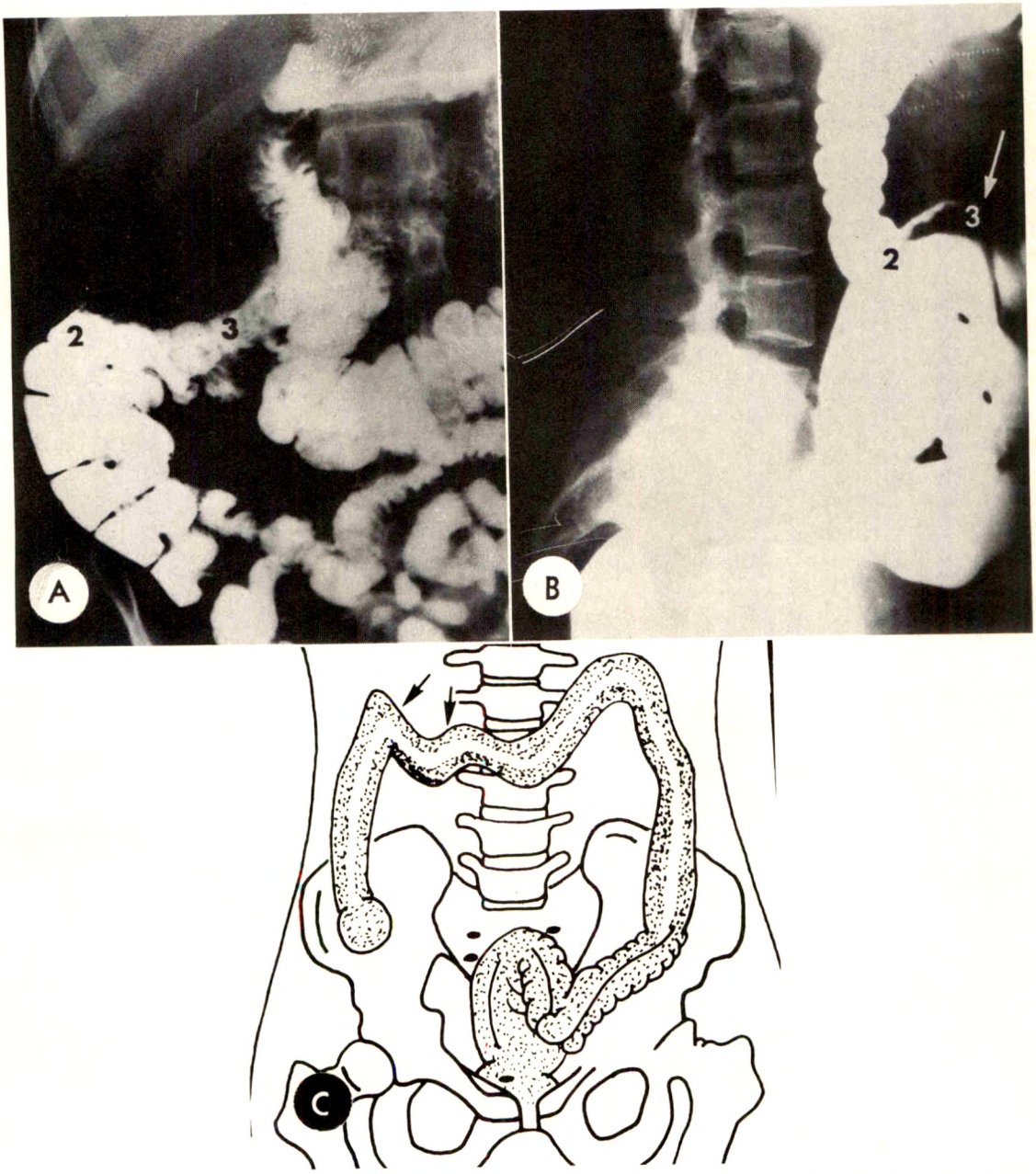


Fig. 12. Hydrops of the gallbladder. (*A*) Frontal roentgenogram showing downward displacement of area 3 of the colon. (*B*) Lateral roentgenogram showing normal position of area 2 with slight indentation of the top surface of area 3 verifying its anterior position (arrow). (*C*) Diagrammatic representation.

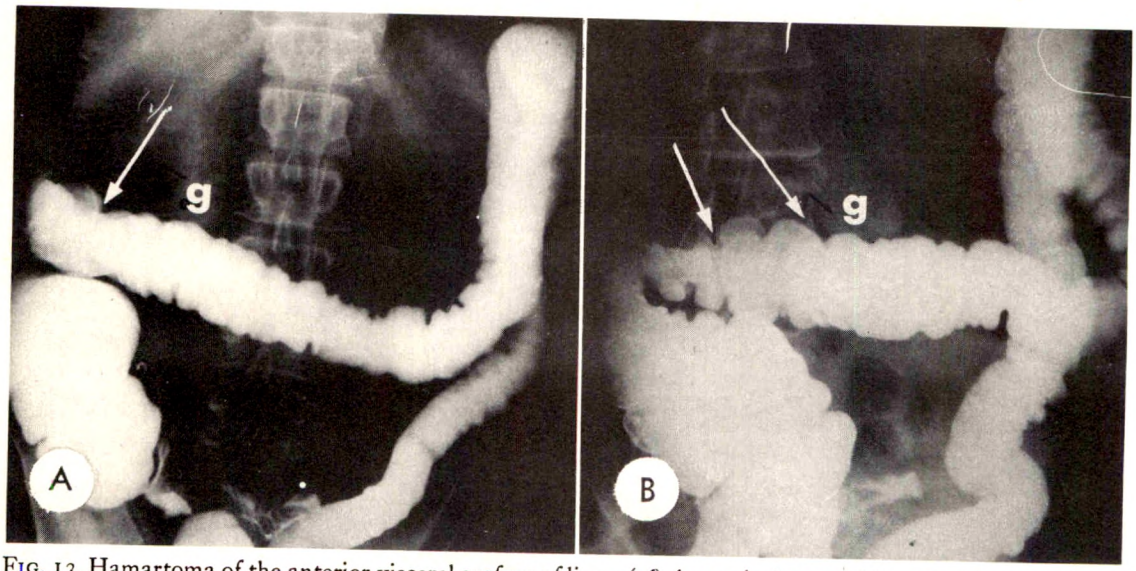


FIG. 13. Hamartoma of the anterior visceral surface of liver. (*A*) Area 3 is depressed but the faintly opacified gallbladder (g) is displaced medially. (*B*) Left posterior oblique view further shows mass effect on anterior portion of colon.

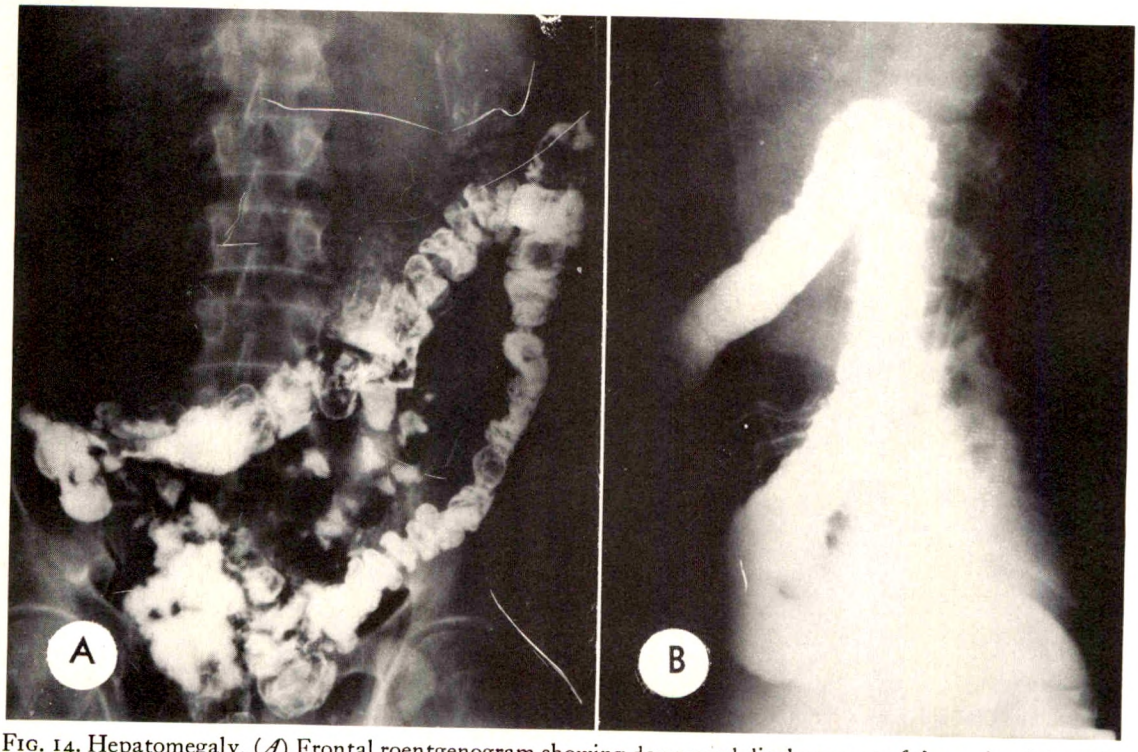


FIG. 14. Hepatomegaly. (*A*) Frontal roentgenogram showing downward displacement of the entire right colic flexure and portions of the transverse colon sparing the left colic flexure. (*B*) Lateral roentgenogram showing the posterior displacement of these areas of the colon.

elevate the colon, again in area 2, but this may be separated from more common inframesocolic masses such as mesenteric cysts by its retrocolic position (Fig. 10).

Right Adrenal Masses. These are supramesocolic and also retrocolic (Fig. 11). A prominent posterior portion of liver will also displace the posterior portion of the right colic flexure.

Gallbladder and Anterior Visceral Surface of Liver. The gallbladder and anterior visceral surface of liver are anterior and supramesocolic and therefore will depress the colon and specifically area 3. At times only a cholecystogram will differentiate a gallbladder mass and one arising from the visceral surface of liver in the area of the bed of the gallbladder (Fig. 12; and 13).

Hepatomegaly. Generalized hepatomegaly will displace the colon in all its parts related to the liver, which includes all of the right colic flexure and much of the proximal transverse colon. It will depress and posteriorly displace the colon (Fig. 14; and 15).

Masses in the Gastrocolic Ligament. These will displace the transverse colon inferiorly but generally spare the anterior portion of both the right and left colic flexures. Unless the stomach is visualized, a dilated stomach cannot be differentiated from a gastrocolic mass (Fig. 16).

Left Adrenal Masses. These are supramesocolic and therefore tend to depress the distal transverse colon and, if they assume a large enough size, all of the left colic flexure (Fig. 17). Visualization of the duodenojejunal junction in an abnormal position would further separate left adrenal masses from more anterior masses such as a gastrocolic mass.[3]

The Body and Tail of the Pancreas. These are also supramesocolic and will depress the transverse colon. Generally, the anterior portion of the left colic flexure and the distal transverse colon are the areas depressed, since the pancreas, although billed as a retroperitoneal organ, is actually quite far anterior[5] (Fig. 18).

Peritoneal Abnormalities. Masses which

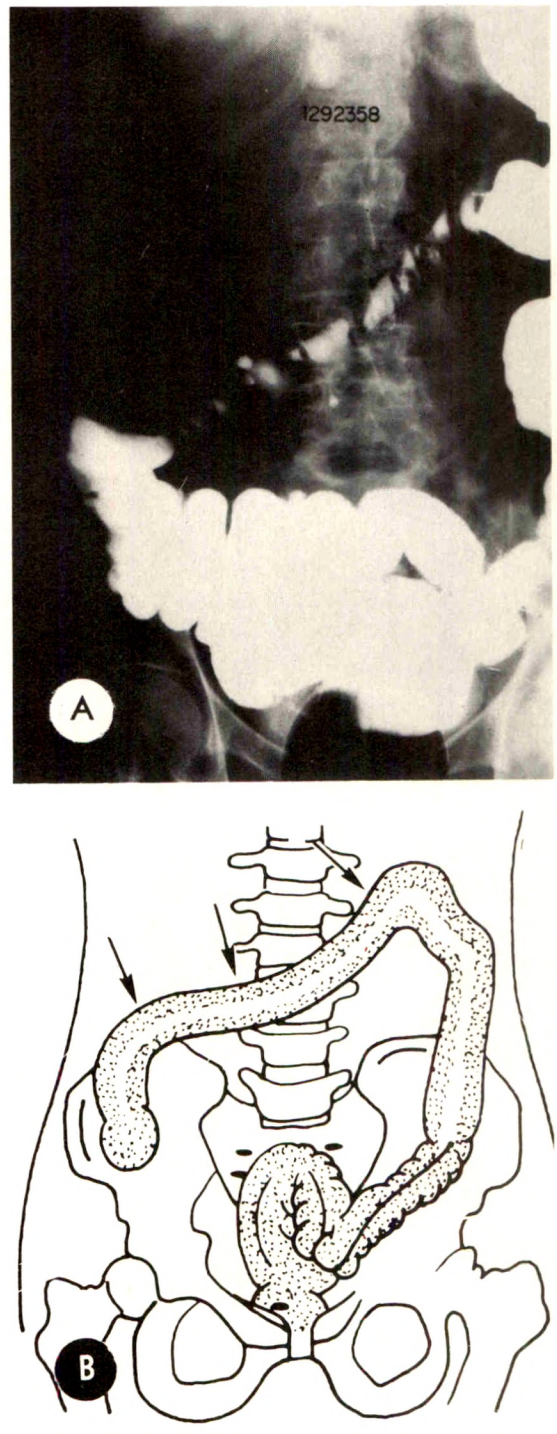


Fig. 15. (*A*) Generalized hepatomegaly showing displacement of right colic flexure and much of transverse colon sparing the left colic flexure. (*B*) Diagrammatic representation of the effect of hepatic enlargement on the colon.

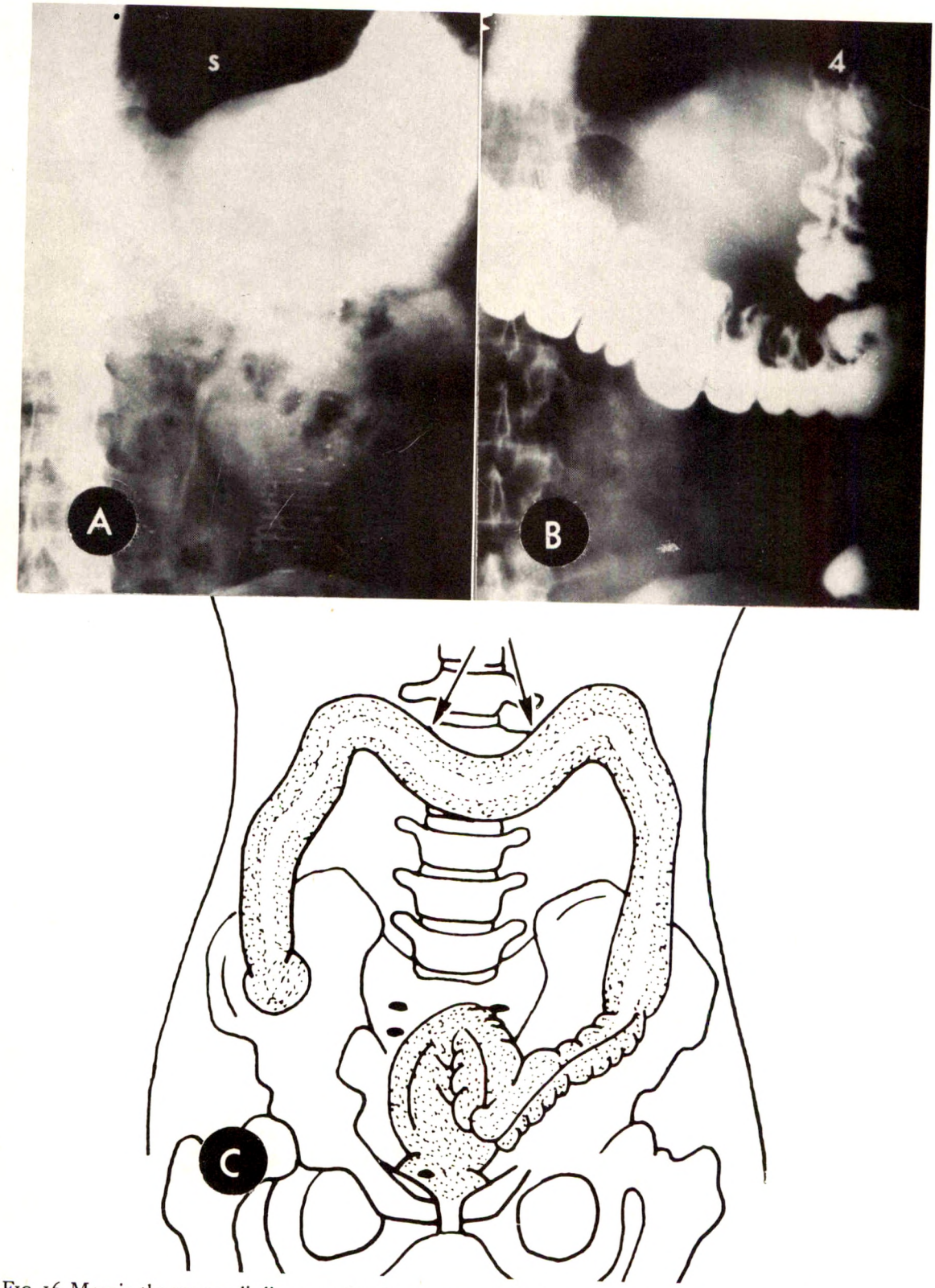


FIG. 16. Mass in the gastrocolic ligament showing downward displacement of the transverse colon but sparing the left and right colic flexure. (*A*) Frontal view showing elevation of stomach, excluding a dilated stomach as the cause. (*B*) Barium enema examination showing the displacement of the transverse colon but not extending to area 4. (*C*) Diagrammatic representation of displacement seen with either a dilated stomach or a mass in the gastrocolic ligament.

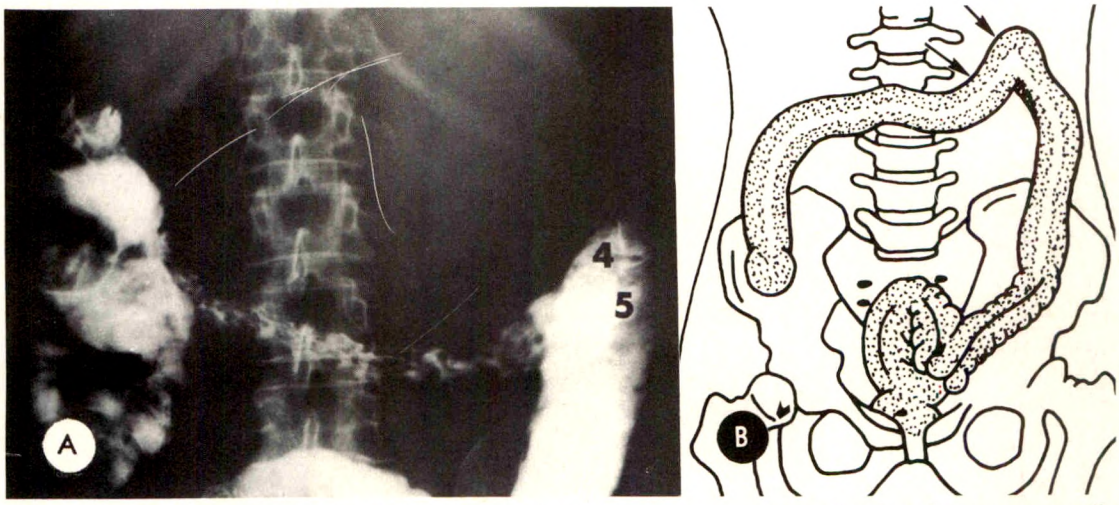


Fig. 17. Large adrenal carcinoma. (*A*) Frontal barium enema roentgenogram showing the supramesocolic location of the mass, large enough so that both area 4 and 5 are depressed. (*B*) Diagrammatic representation of colonic displacement by a left adrenal mass.

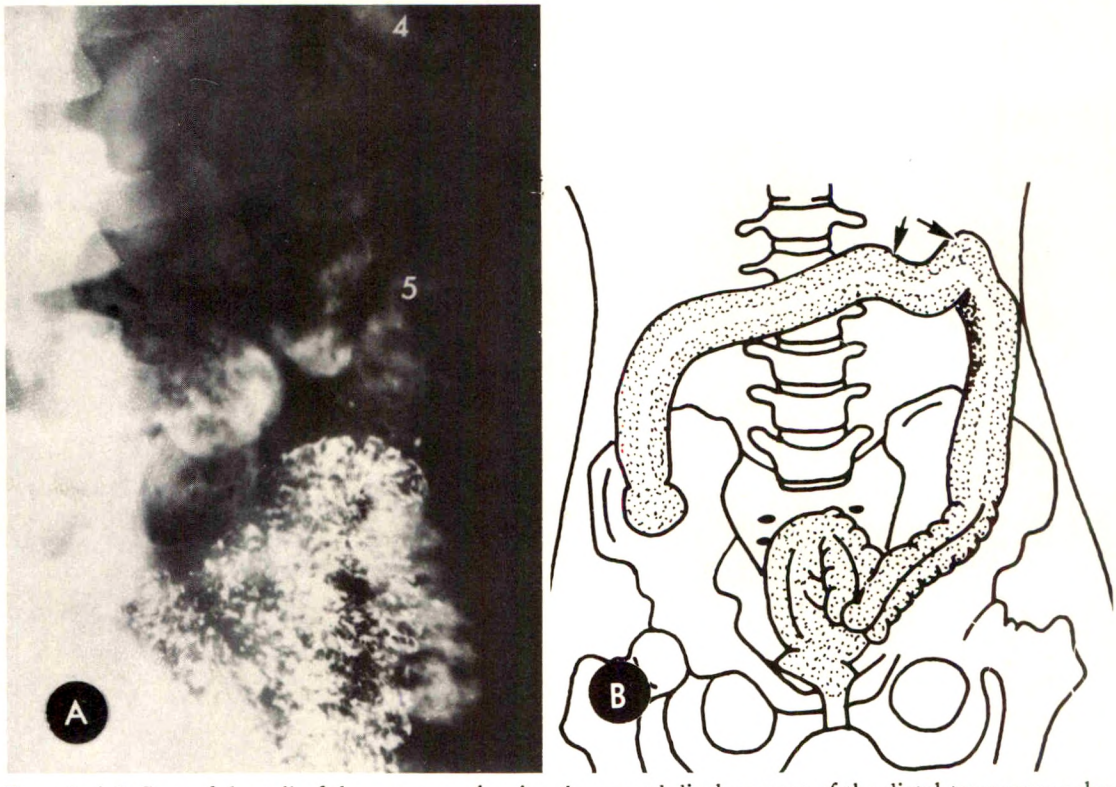
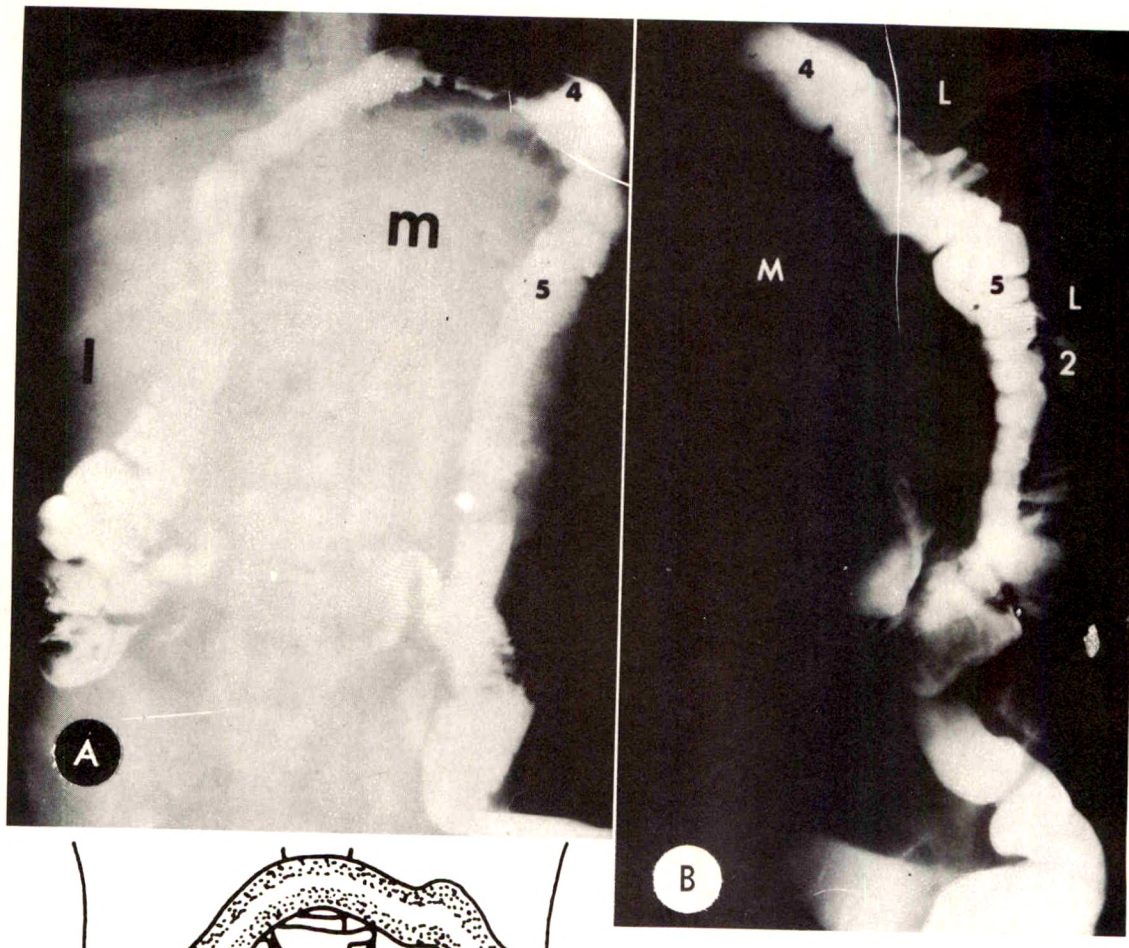


Fig. 18. (*A*) Cyst of the tail of the pancreas showing downward displacement of the distal transverse colon causing approximation of distal transverse colon to area 4 and 5. This will be difficult to separate from a left adrenal mass. (*B*) Diagrammatic representation of a mass in the tail of the pancreas.



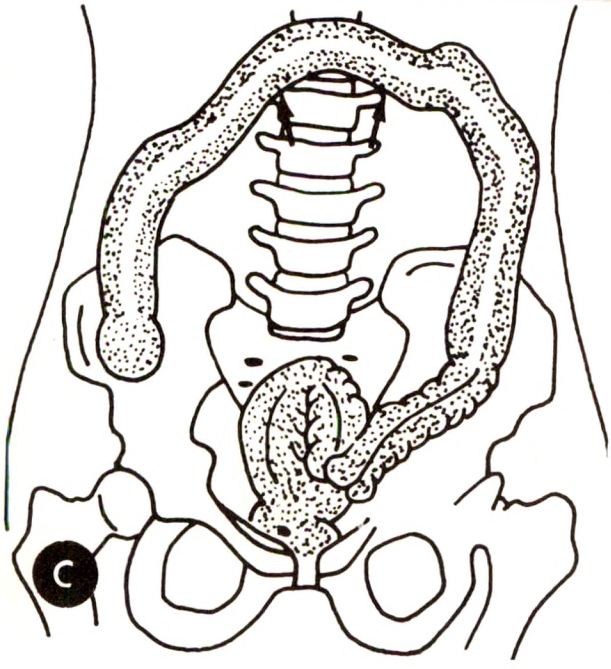


FIG. 19. (*A*) A large ovarian cyst extending superiorly in the peritoneal cavity displacing the sigmoid colon and the anterior transverse colon, sparing the posterior aspects. The posterior aspect of liver (1) can be seen separated from the mass (m) by the right colic flexure. (*B*) The mass (M) is seen to be anterior to the ascending and descending colon (5 and 2), and the posterior portion of right lobe of liver (the lower L). The more anterior portion of liver (upper L) has been elevated in continuity with the transverse colon by the anterior inframesocolic intraperitoneal ovarian mass. (*C*) Diagrammatic representation of a peritoneal mass.

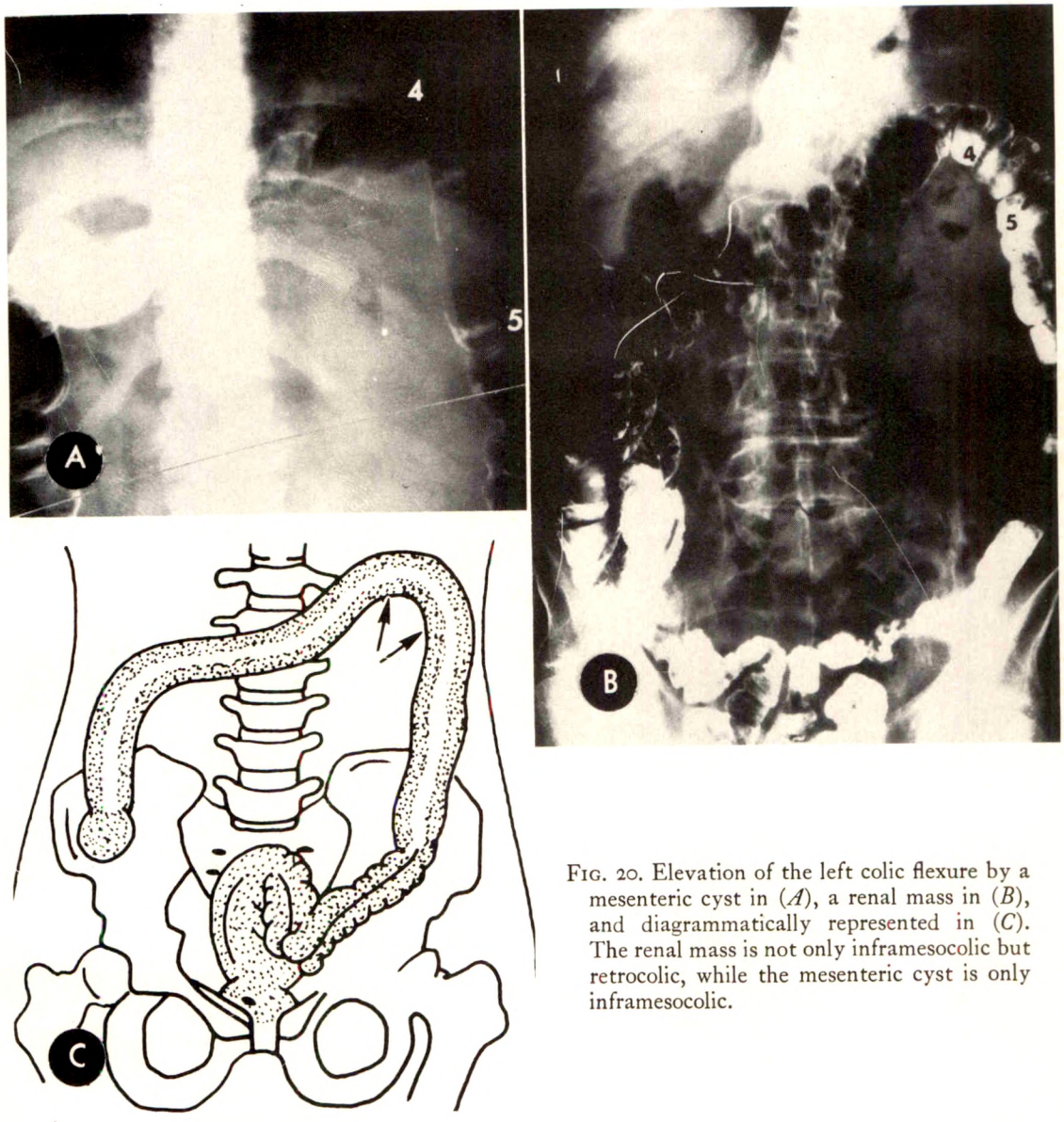


Fig. 20. Elevation of the left colic flexure by a mesenteric cyst in (*A*), a renal mass in (*B*), and diagrammatically represented in (*C*). The renal mass is not only inframesocolic but retrocolic, while the mesenteric cyst is only inframesocolic.

either arise in the peritoneal cavity or extend to it are all inframesocolic and will elevate that portion of the colon with which they come in contact. They will generally involve the anterior portions of colon (areas 3 through 4) and sigmoid, elevating those portions, and will merely flatten the more posterior portions since little posterior displacement can occur because the structures behind the ascending and descending colon are rigid, such as bone and muscle (Fig. 19).

Left Kidney. Since the major portion of the left kidney is inframesocolic the left flexure will be elevated. This may be differentiated from a mesenteric cyst in this area by the retrocolic position of the renal mass (Fig. 8; and 20).

Anterior Portion of Spleen. The entire spleen is supramesocolic with the anterior portion of the colon (area 4) being related to the anterior area of the spleen. As that portion of the spleen enlarges, there is some downward, but primarily medial, displace-

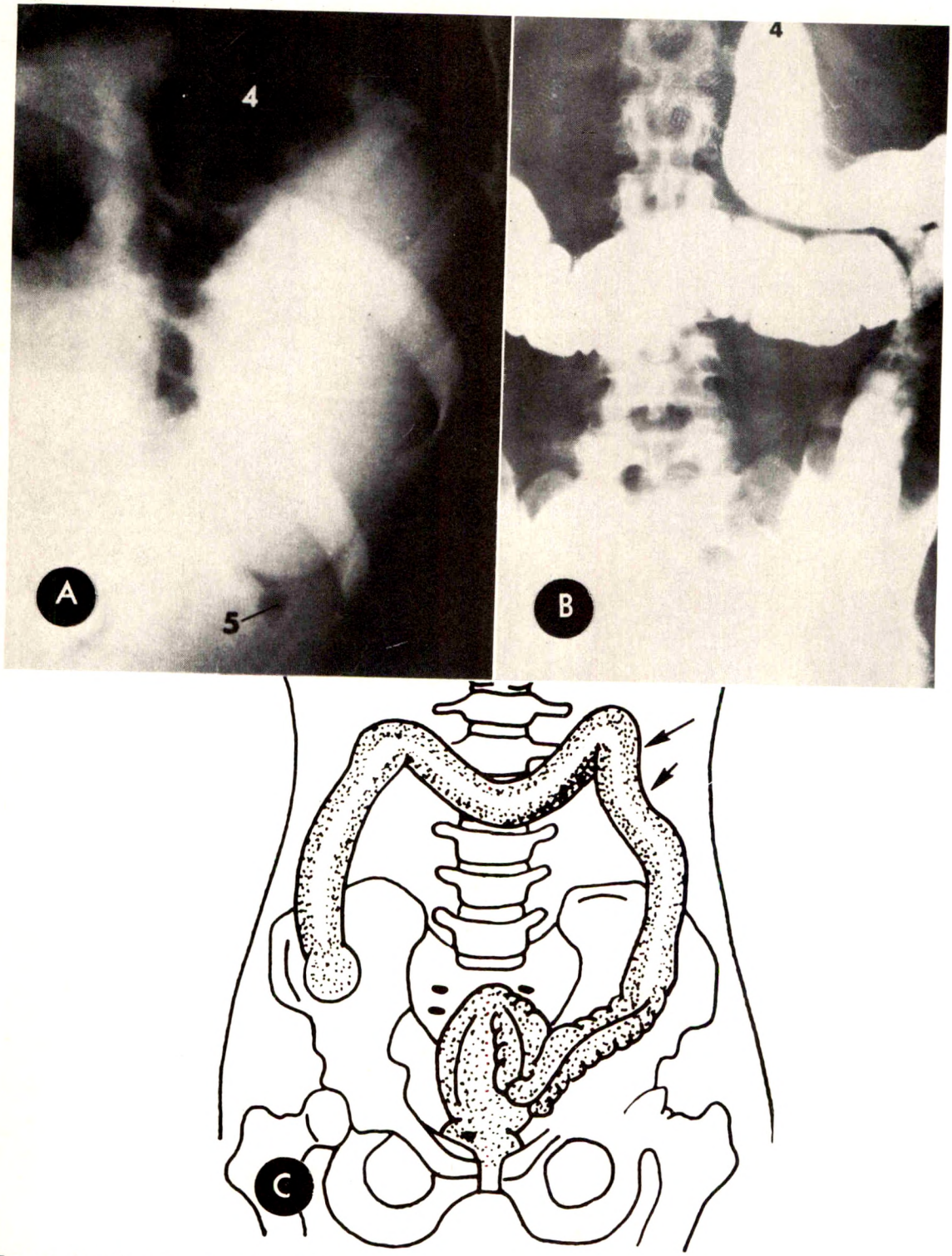


FIG. 21. Anterior splenomegaly. (*A*) Medial displacement of area 4 of the colon by a large anterior spleen. Area of phrenicocolic ligament is partially air-filled in the normal position (area 5). (*B*) Another patient with anterior splenomegaly showing characteristic medial displacement of area 4. (*C*) Diagrammatic representation of anterior splenomegaly.

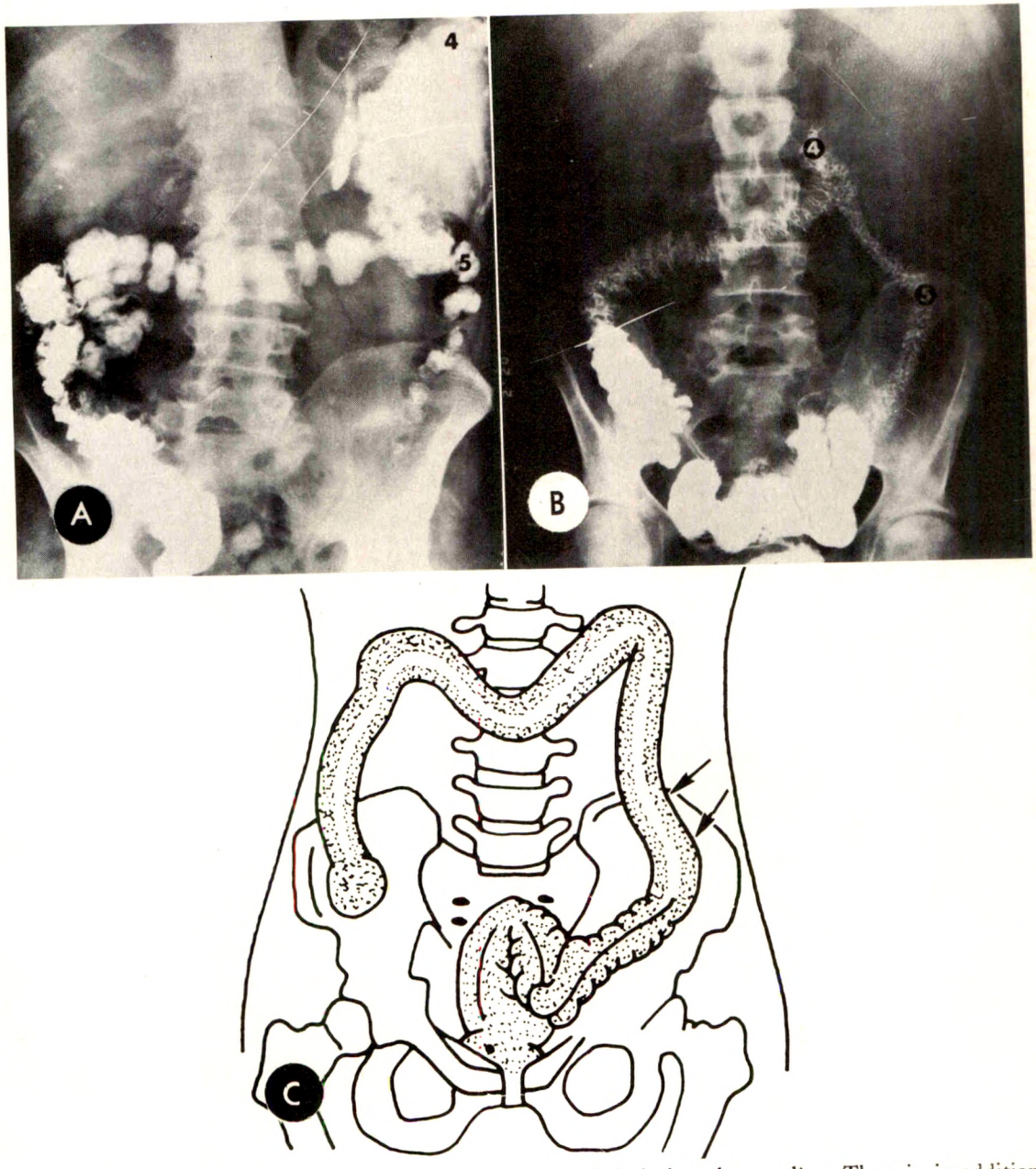


Fig. 22. Posterior splenomegaly. (*A*) Area 5 is displaced inferiorly by a large spleen. There is, in addition, medial displacement of the area of colon between 4 and 5 reflecting anterior enlargement. (*B*) Marked posterior enlargement of the spleen. Downward displacement of area 5. There is, in addition, anterior splenomegaly. (*C*) Diagrammatic representation of posterior splenomegaly.

ment of that area of colon. This is the type of splenomegaly which is often palpable by the clinician as the edge extends beneath the rib cage.

Posterior Portion of Spleen. Posterior splenomegaly manifests itself by depressing the posterior portion of the left colic flexure (the true splenic flexure or area of the phrenicocolic ligament). If splenomegaly is primarily posterior, the spleen will not be palpable[1,2] (Fig. 21; and 22).

SUMMARY

The normal contours and relationships of the colon have been described in a previous publication.[4] I have attempted to show how enlargement of these organs or tumefactions arising within them affect the colon in a characteristic manner. Hopefully this will become useful in: (1) determining the normality of the external contour and variation of the external contour of the colon; (2) detecting a clinically unsuspected mass by an abnormal indentation on the colon; and (3) providing, for the diagnostic work-up of abdominal masses, a primary classification of abdominal masses. Further, I have tried to emphasize that the lateral aspects of both colic flexures are posterior and tend to reflect abnormality of the posterior structures. The medial aspects of the colic flexures are anterior and likewise reflect abnormality of anterior structures. In addition, I re-emphasize that what in the past has been called the splenic flexure (the area of marked angulation of the colon in the left upper quadrant) in reality is merely a portion of distal transverse colon. The more subtle angulation of the colon related to the "splenic angle" and left kidney is the true splenic flexure, the area of the phrenicocolic ligament.

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REFERENCES

1. RIEMENSCHNEIDER, P. A., and WHALEN, J. P. Relative accuracy of estimation of enlargement of liver and spleen by radiographic and clinical methods. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *94*, 462–468.
2. WHALEN, J. P. Clinical and radiological diagnosis of splenomegaly. To be published.
3. WHALEN, J. P., EVANS, J. A., and SHANSER, J. Vector principle in differential diagnosis of abdominal masses. I. Left upper quadrant. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 104–118.
4. WHALEN, J. P., and RIEMENSCHNEIDER, P. A. Analysis of normal anatomic relationships of colon as applied to roentgenographic observations. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *99*, 55–61.
5. WHALEN, J. P., RIEMENSCHNEIDER, P. A., and BERNE, A. S. Extraperitoneal perivisceral fat pad. I. Its role in roentgenologic visualization of abdominal organs. *Radiology*, 1969, *92*, 466–472.

VOL. 125, No. 1

THE CHOLECYSTO-COLIC RELATIONSHIPS*

A ROENTGEN-ANATOMIC STUDY OF THE COLONIC MANIFESTATIONS OF GALLBLADDER DISORDERS

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ABSTRACT:

In approximately 80 per cent of cases the gallbladder is closely applied to the superior medial aspect of the right colic flexure. This intimate anatomic relationship provides pathways for direct extension of both inflammatory and neoplastic lesions of the gallbladder to involve the adjacent colon. The resultant secondary colonic abnormalities noted in 15 patients have been analyzed and correlated with surgical-pathologic findings.

In acute cholecystitis, barium enema examination shows evidence of indentation by an enlarged gallbladder, spasm and mucosal edema in the anterior hepatic flexure. Chronic cholecystitis results in involvement of the adjacent colon by fibrous adhesions and inflammatory reaction. These may further lead to the development of pseudotumors simulating primary carcinoma of the colon. Similar findings including cholecysto-colic fistulae may be the initial manifestations of carcinoma of the gallbladder.

The spectrum of pathologic-roentgenographic alterations in the cholecysto-colic interface is described and illustrated. Recognition of these features is of critical importance for the correct interpretation of barium enema findings and the subsequent management of patients with gallbladder disorders.

THE nonspecificity of signs and symptoms of diseases of the gallbladder often makes the clinical diagnosis and differentiation from processes in other organs difficult.[8,12,27] The problem is further complicated by the fact that intimate anatomic relationships of the gallbladder to adjacent segments of the colon and duodenum provide natural pathways for extension of inflammatory and neoplastic lesions originating in the gallbladder.[2,3,10,13,24,29] At times, it is the striking manifestation of the secondary effects upon these neighboring organs that first brings the patient to medical attention.[5,7,8,22,25] Since roentgenologic evaluation of gastrointestinal disorders is often initiated by a barium enema study, detection of an abnormality in the colon may be very misleading.[7,9,11,23,24] It may even adversely influence the further course of work-up and treatment of the patient with gallbladder disorder.

The purposes of this report are multiple: (1) to clarify the anatomic features of the cholecysto-colic relationships and their significance in the spread of pathologic processes; (2) to document the variety of roentgenographic presentations of colonic involvement by inflammatory and neoplastic lesions of the gallbladder; (3) to establish criteria for recognition of the secondary nature of resultant abnormalities shown on barium enema study which may otherwise simulate a primary lesion of the colon; and (4) to emphasize the complementary information obtained by colon examination of patients in whom the wide range of possible causes for nonvisualiza-

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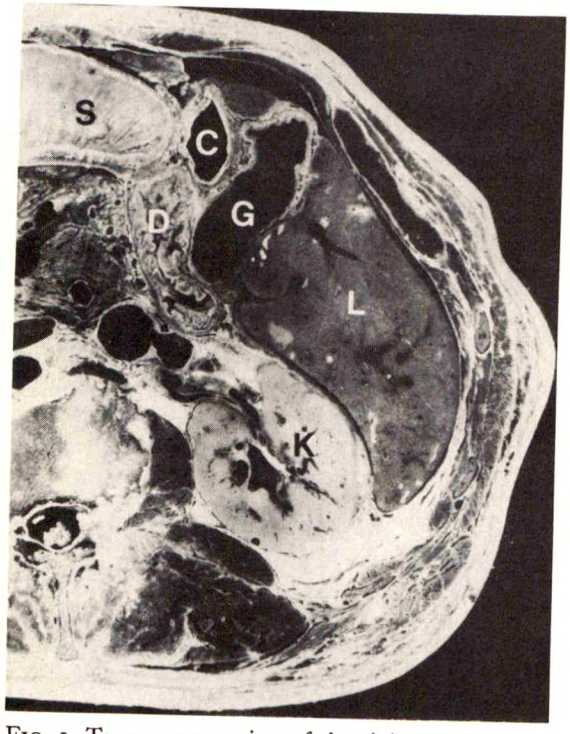

FIG. 1. Transverse section of the right upper abdomen at midplane of the right kidney (K) demonstrates intimate relationships of the gallbladder (G) to the hepatic flexure of the colon (C) and the liver (L). D = duodenal bulb and proximal descending duodenum; S = stomach.

tion of the gallbladder on cholecystography has previously resulted in a diagnostic dilemma.

MATERIAL AND METHOD

The cholecysto-colic relationships were evaluated by examining horizontal and sagittal sections of the abdomen of several frozen cadavers.

Fluoroscopic and roentgenographic observations were made on 100 patients undergoing oral cholecystography. In these individuals, the opacified gallbladder and the gas- or contrast material-containing right colic flexure permitted evaluation of their relationships in various positions and respiratory phases.

Fifteen patients in whom barium enema examination was the initial study revealing abnormalities in the colon subsequently proved to be the result of a gallbladder disorder were analyzed. The underlying pathologic conditions were acute cholecystitis in 3, chronic cholecystitis or its complications in 8, and carcinoma of the gallbladder in 4 patients. The roentgen findings were correlated with observations made at surgery and with changes noted in the resected specimens available in 12 cases.

The 12 female and 3 male patients were 30 to 88 years of age, with the majority in their sixth or seventh decade of life. The presenting clinical symptoms which led to barium enema examination included abdominal pain, weight loss, rectal bleeding, and change in bowel habits, usually with diarrhea. Nine patients had a palpable mass or tenderness or both in the right upper or midabdomen. There were no symptoms of biliary colic or evidence of jaundice on initial presentation. In 2 patients, however, gallstones had been diagnosed 5 and 9 years earlier. They were among the 10 of 15 patients from whom a history indicative of biliary tract disease could be elicited on subsequent careful inquiry.

ANATOMIC AND NORMAL ROENTGENOLOGIC CONSIDERATIONS

The hepatic flexure of the colon anatomically is actually composed of 2 segments.[21,30] The proximal or ascending section is a continuation of the retroperitoneally fixed ascending colon and is closely related to the posterolateral tip of the liver and the lateral margin of the right kidney. The distal or transverse section of the flexure lies in a more superior and anterior plane, and has limited mobility because of its attachment to the beginning of the transverse mesocolon. It is precisely the superior and medial surface of this segment of the hepatic flexure which is in close proximity to the gallbladder (Fig. 1; and 2), thus being potentially subject to involvement by processes originating in that organ. It has been recently shown by Meyers *et al.*[20] that the gallbladder is typically closely applied to the adjacent colonic haustra lo-

cated between the taenia mesocolica and the taenia omentalis. Although not necessarily conspicuous in all instances, these particular haustra are those primarily involved by gallbladder disorders.

The gallbladder is attached superiorly to the undersurface of the right and quadrate lobes of the liver. It is otherwise covered by peritoneum which reflects to continue onto the hepatic serosal surface. The separation of the gallbladder inferiorly from the adjacent hepatic flexure is provided by a "potential" intraperitoneal space, as shown by its opacification during peritoneography.[16] This intimate relationship, however, is influenced by anatomic variations in position, size, and the degree of mobility of the gallbladder, liver, and colon. For example, the gallbladder may be entirely invested by peritoneum, and connected with the liver by a long mesentery. The resultant "floating" gallbladder will not possess a constant relationship to the colon unless dictated by pericholecystic adhesions. The intrahepatic gallbladder and hepato-diaphragmatic interposition of the colon are also among congenital variations which effectively prohibit a close cholecysto-colic relationship.

Our fluoroscopic and roentgenographic evaluation of 100 patients undergoing oral cholecystography shows that the gallbladder is closely applied to the hepatic flexure in 78 per cent of the patients. In these individuals, the distance between the inner walls of the gallbladder and the adjacent colon is less than 1.5 cm. as measured from their luminal contours directly on the upright compression spot roentgenograms, disregarding the magnification. In only 7 patients (7 per cent) was the distance greater than 3.0 cm. Furthermore, the degree of separation of the 2 organs appears essentially independent of inspiration or expiration, whether the patient is supine or erect. These findings further define the results of a similar study reported by Jacobson *et al.*[6] These authors noted that the gallbladder and the hepatic flexure were virtually contiguous in 63.5 per cent, separated by 1–3 cm. in 31.3 per cent and "distantly removed" in 5.2 per cent.

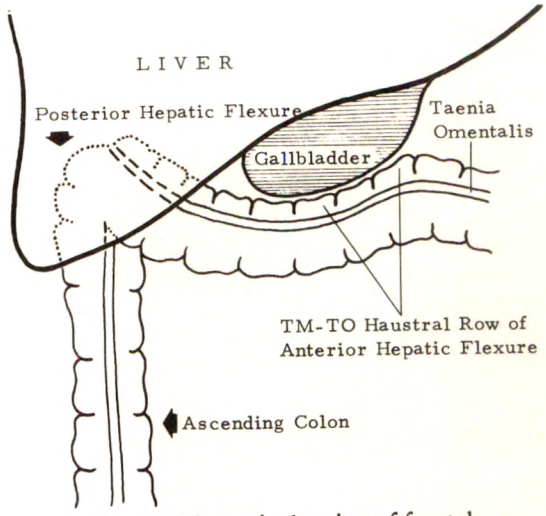

Fig. 2. Schematic drawing of frontal gallbladder—colon relationship.

REPORT OF CASES

Case 1. *Acute cholecystitis.* This 30 year old female with a history of chronic pyelonephritis and hypertension was being followed as an outpatient as a prospective candidate for renal transplantation. Her re-admission was prompted by 2 days of crampy periumbilical pain, nausea, diarrhea, and epigastric tenderness. The presumptive clinical diagnosis was uremic enteropathy.

Barium enema examination demonstrated evidence of localized extrinsic compression and mild mucosal irregularity on the superior border of the anterior hepatic flexure of the colon (Fig. 3*A*). Oral cholecystography 2 days later failed to opacify the gallbladder, and an upper gastrointestinal series showed spasm and extrinsic compression of the postbulbar segment of the duodenum. These roentgen findings, together with the development of a palpable tender gallbladder, were consistent with diagnosis of acute cholecystitis.

Under medical observation and antibiotic therapy, the symptoms subsided within a few days. An air-contrast enema study performed 2 weeks later showed the hepatic flexure to appear normal (Fig. 3*B*). However, 4 months later the patient's symptoms recurred, and at surgery an acutely inflamed gallbladder containing

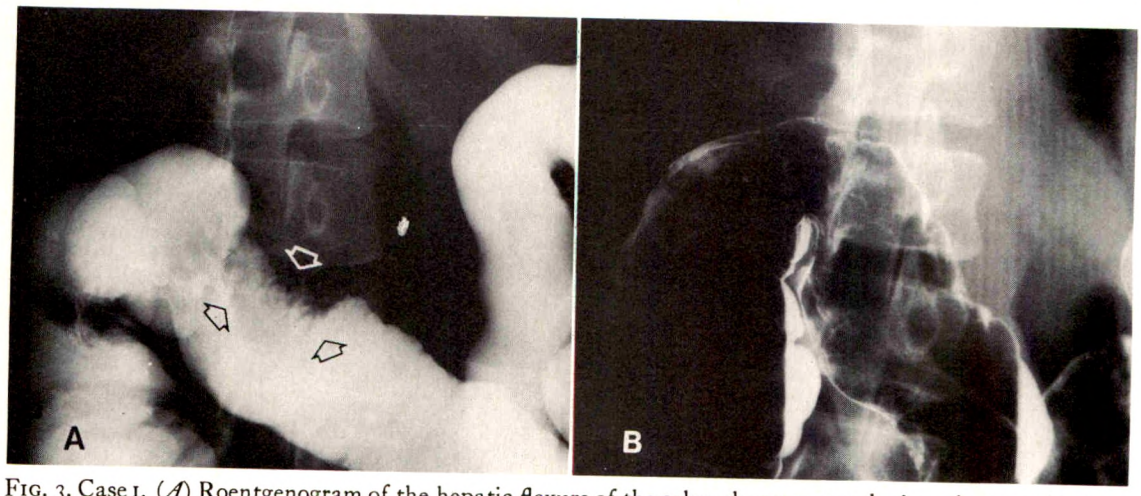

FIG. 3. Case 1. (*A*) Roentgenogram of the hepatic flexure of the colon shows mucosal edema in area of indentation by the enlarged gallbladder (arrows), and a focal contraction proximally. (*B*) Air-contrast colon examination reveals normal appearance of the right colic flexure 2 weeks after remission of symptoms.

several pigmented stones was removed without complication.

Comment. In acute cholecystitis the gallbladder is often enlarged due to distention from obstruction of the cystic duct by a stone or, less frequently, by edematous spiral folds (valve of Heister).[3] The wall of the gallbladder is diffusely thickened and edematous. The congested serosal surface usually shows patches of fibrinous exudate. At this stage, barium enema examination may reveal smooth extrinsic compression of the hepatic flexure by the distended gallbladder with evidence of localized mucosal edema (Fig. 3*A*). Furthermore, occurrence of segmental contractions in the hepatic flexure can be recognized during barium enema examination, and the antegrade passage of barium beyond the cholecystocolic interface may be delayed (Fig. 4, *A* and *B*).

CASE II. *Pseudotumor of the hepatic flexure secondary to chronic cholecystitis.* This 54 year old female had undergone vagotomy and pyloroplasty for chronic peptic ulcer disease 2 years previously. Subsequently, she developed crampy lower abdominal pain, usually occurring 1 hour postprandially and often relieved by defecation. Anorexia, a 27 pound weight loss, and frequent episodes of foul-smelling diarrhea were also present.

Physical examination showed slight epigastric tenderness. A mild hypokalemia was the only relevant laboratory finding. Gastroduodenal cytology was negative.

Barium enema examination showed an apparent 1.5 cm. filling defect with a probable central ulceration in the most superior aspect of the anterior hepatic flexure (Fig. 5*A*). On air contrast study 2 days later, however, only severe spasm of the hepatic flexure and minimal mucosal irregularity were evident, indicative of a localized inflammatory process (Fig. 5*B*). Oral cholecystography with single and double dose of contrast material did not visualize the gallbladder, although tomography showed faint opacification of a normal appearing common bile duct.

A shrunken and chronically inflamed gallbladder was resected at surgery. Following lysis of adhesions between the gallbladder, the right colic flexure, and the omentum, the adjacent colon appeared normal on palpation. Subsequent course has been uneventful.

Comment. In chronic cholecystitis the gallbladder may be either distended or shrunken, although its wall is often diffusely thickened. Marked fibrous adhesions are usually present between the thickened serosa and the adjacent structures, namely the colon, duodenum, and the greater omentum.[3,5,14,24,25] These adhesions must be removed to make the gallbladder visible and accessible when cholecystectomy is at-

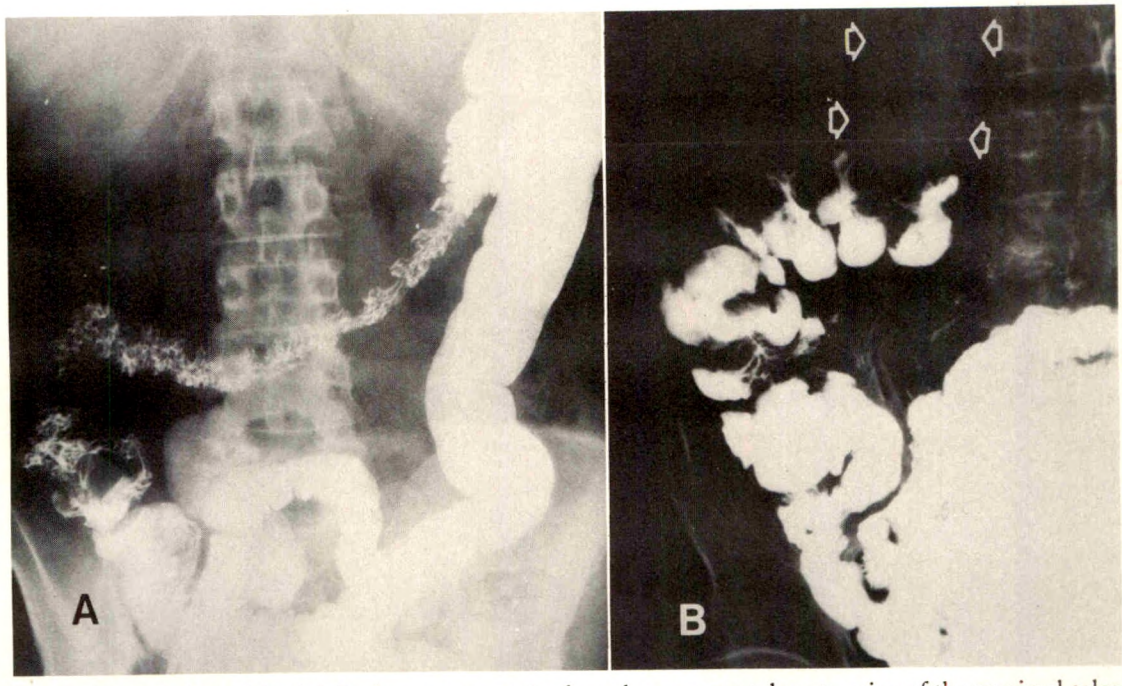

Fig. 4. Acute cholecystitis. (*A*) Barium enema study shows long segmental contraction of the proximal colon and slight inferior displacement of the anterior hepatic flexure. (*B*) The 4 hour roentgenogram of a small bowel series done 4 days later demonstrates intimate relationship of the fundus of the gallbladder (arrows) to the haustra in the superior aspect of the adjacent colon. The antegrade passage of barium beyond the cholecysto-colic interface was delayed. The gallbladder (arrows) containing radiolucent stones is faintly visualized on double dose cholecystography.

tempted. As a result of such pericholecystic adhesions and the secondary involvement of the hepatic flexure by chronic and recurrent inflammation of the gallbladder, a variety of findings may be noted on barium enema examination. In 2 of our patients a polypoid filling defect (Fig. 5*A*; and 6) was present in the superior aspect of the right colic flexure. On laparotomy pericholecystic adhesions involving this area and accounting for the roentgenographically demonstrated pseudotumors were lysed and the colon itself appeared normal on careful palpation. In other instances, however, segmental resection of the colon containing pseudotumors composed of granulation tissue has been necessary.[7,22]

Hydrops and empyema of the gallbladder are well-known complications of cystic duct obstruction by impacted gallstone, scarring, or inflammatory edema. Barium enema examination and sometimes a plain film roentgenogram of the abdomen may demonstrate the distended gallbladder compressing upon the superior aspect of the hepatic flexure.[26,29] This segment usually shows evidence of mucosal edema and spasm in cases with empyema, while lack of such inflammatory changes and absence of leukocytosis, jaundice, and localized tenderness are typical for hydrops. Smooth extrinsic indentation of the colon by an enlarged gallbladder may also occur in patients with a "porcelain" gallbladder (Fig. 7) and with Courvoisier's sign of pancreaticoduodenal malignancy (Fig. 8).

Case III. *Pericholecystic abscess involving the colon.* This 67 year old male was admitted with a 2 day history of mild fever, dizziness, and diarrhea containing frank blood. He also complained of anorexia resulting in a 30 pound weight loss during the past 3 months. The past history included hypertension, and recurrent urinary tract infection treated until 4 months prior to admission.

Physical examination revealed a slightly en-

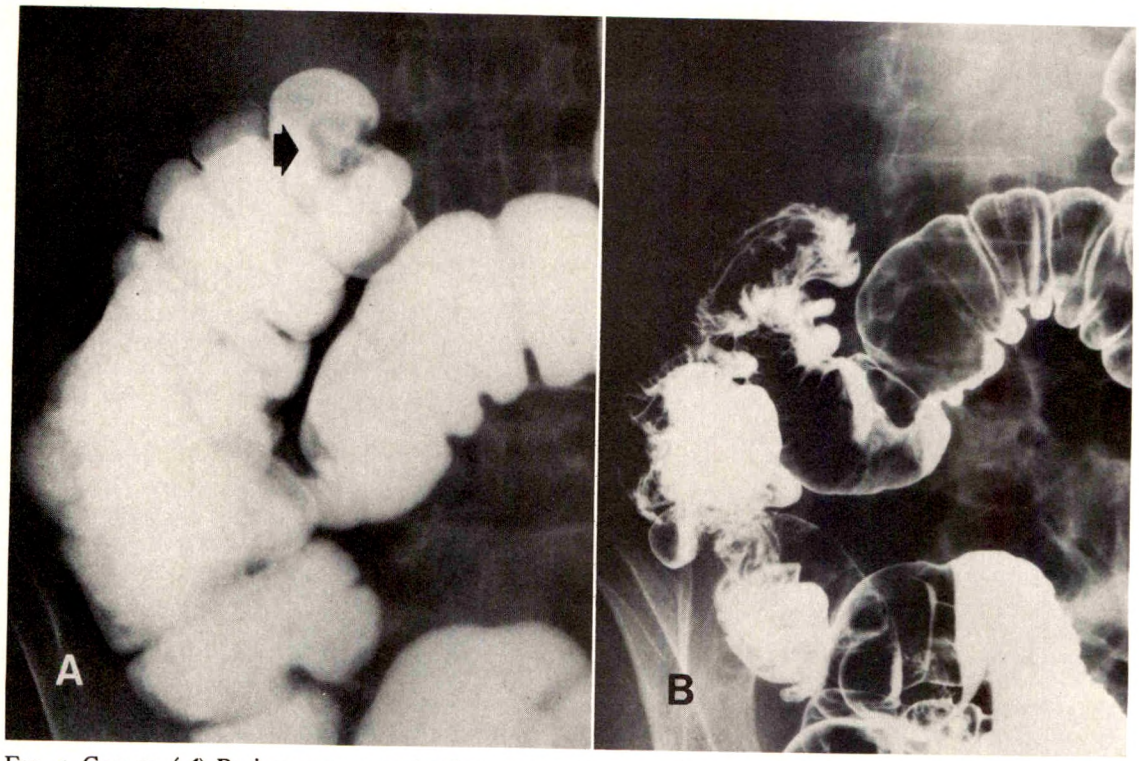

FIG. 5. Case 11. (*A*) Barium enema study shows a 1.5 cm. mass (arrow) with apparent central collection of barium in the anterior hepatic flexure. (*B*) Repeat examination by air-contrast enema shows evidence of segmental spasm of the right colic flexure.

larged firm liver with mild tenderness of the right upper abdomen. The bowel sounds were normal. The white blood cell count was 18,800 and the hemoglobin was 8.7 gm. per cent. The benzidine test of stools was positive but no enteropathogenic organisms were detectable in specimens. The provisional clinical diagnosis was a carcinoma of the colon with probable metastasis to the liver.

A barium enema examination showed a 6 cm. long constricting lesion of the anterior hepatic flexure, with a shelf-like distal border and irregular, prominent mucosa without ulceration (Fig. 9, *A–C*). The involved segment appeared rigid and fixed in position but it was not obstructive to the retrograde flow of barium. A liver scintigram with Tc^{99m}-sulfur colloid also showed a large defect in the anteroinferior margin of the liver consistent with extrinsic compression by a mass.

On laparotomy a large mass incorporating the proximal transverse colon and the gallbladder was found adherent to the undersurface of the liver. Frozen sections of 4 biopsy specimens demonstrated a dense focally hyalinized fibroadipose tissue with aggregates of chronic inflammatory cells and foci of hemorrhage and necrosis. Subsequently the mass was dissected from the liver, and upon incision a central cavity containing 2 small gallstones and yellowish-creamy material was entered. The analysis of the evacuated content revealed presence of *Clostridium perfringens* and 2 strains of *Escherichia coli*. The surgical and histopathologic findings were consistent with an old perforated empyema of the gallbladder resulting in infrahepatic and pericolic abscess formation. Therefore, a Pezzar catheter was inserted into the remnant lumen of the gallbladder, and Penrose drains were placed in the supra- and infrahepatic spaces. Postoperatively, the patient was treated with massive doses of antibiotics, blood transfusions, and other supportive medical measures. However, the course became complicated by development of pneumonia and septicemia, and the patient expired 5 weeks later.

Comment. Perforation of the gallbladder complicating phlegmonous inflammation

or decubitus ulceration by gallstones can lead to generalized peritonitis and subphrenic abscess. More commonly, however, such perforations remain localized because the omentum and serosa of the surrounding viscera adhere to and effectively seal the gallbladder. Nonetheless, the pericholecystic abscess can involve and encircle the hepatic flexure of the colon producing changes which closely simulate a primary tumor of the colon roentgenographically (Fig. 9, *A–C*)[1,9,24] and surgically.[1,7,22,28] A careful evaluation of the roentgenographic findings with respect to the specific location, length of the lesion, and preservation of the otherwise retracted and crowded mucosal folds should correctly indicate the secondary nature of the abnormality.

Internal biliary fistulae are most commonly a complication of calculous cholecystitis.[3,7,14,22] The cholecystocolic fistulae account for up to 20 per cent of all fistulae, and are second in frequency to those involving the duodenum.[25,28] Pericholecystic abscess and adhesions extending into the postbulbar segment of duodenum or the right colic flexure, or both, facilitate perforation of the gallbladder and expulsion of stones into the intestinal lumen (Fig. 10) through the fistula.[3,14,25] Cholecystocolic fistulae are detected incidentally when a barium enema study (Fig. 11) is performed because of diarrhea and weight loss.[25] These symptoms are due to the cathartic effect of bile salts entering the colon directly, and to the steatorrhea related to fat indigestion in the absence of bile in the small intestine. Lower gastrointestinal hemorrhage is occasionally the presenting symptom,[7,14] as it was in 1 of our 4 patients with fistulae from the gallbladder to the colon.

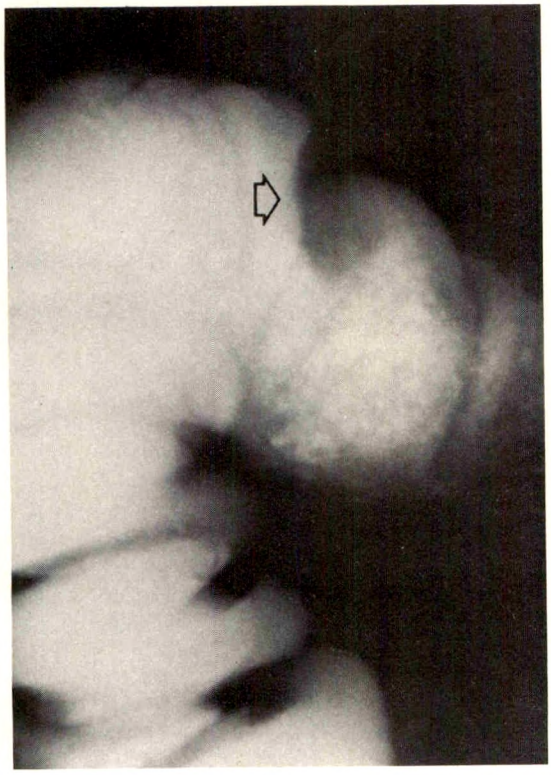

Fig. 6. Pseudotumor of the colon (arrow) due to pericholecystic adhesions in a 60 year old female. At operation, fibrous bands and adhesions between the gallbladder and the greater omentum were found adherent to and compressing the superior aspect of the anterior hepatic flexure.

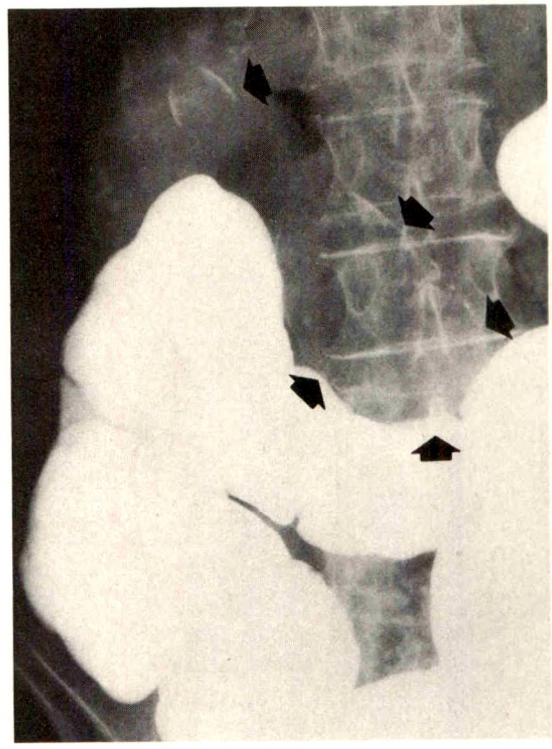

Fig. 7. Roentgenogram of barium enema study in an 81 year old man with rectal bleeding and a palpable abdominal mass. The large "porcelain" gallbladder (arrows) with an impacted stone in its neck was adherent to the depressed anterior hepatic flexure.

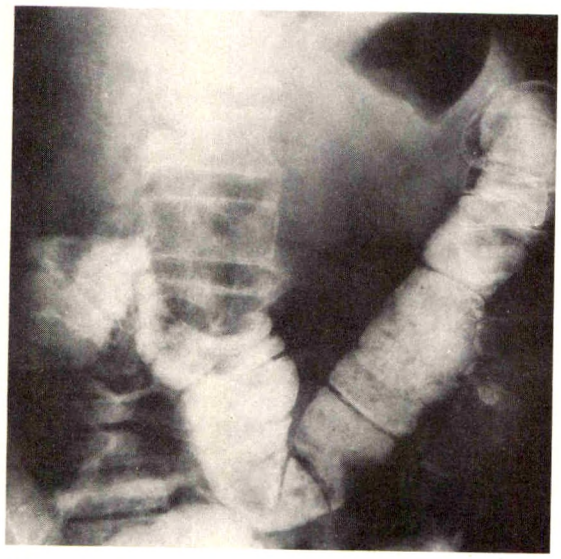

FIG. 8. This roentgenogram of a barium filled colon demonstrates smooth extrinsic compression of the hepatic flexure by an enlarged gallbladder secondary to carcinoma of the pancreas (Courvoisier gallbladder).

CASE IV. *Invasion of the colon by carcinoma of the gallbladder.* This 50 year old female was in good health until 6 weeks prior to admission when she developed epigastric discomfort, malaise, nausea, and loss of appetite. Progression of symptoms was associated with occasional passage of blood-stained stools. A barium enema examination done elsewhere demonstrated a 2.5 cm. sessile polypoid mass in the medial aspect of the proximal hepatic flexure compatible with a primary carcinoma of the colon (Fig. 12*A*). On admission she appeared mildly icteric, and complained of heartburn, fullness in the epigastrium, and nausea. There was a slight but diffuse tenderness in the upper abdomen without a palpable gallbladder. The findings were suggestive of metastatic involvement of the liver.

Repeat barium enema study revealed marked rigidity and infiltration of the superior aspect of the anterior hepatic flexure in addition to the previously noted sessile lesion (Fig. 12*B*). Attempts to opacify the gallbladder by oral and infusion cholecystography were unsuccessful.

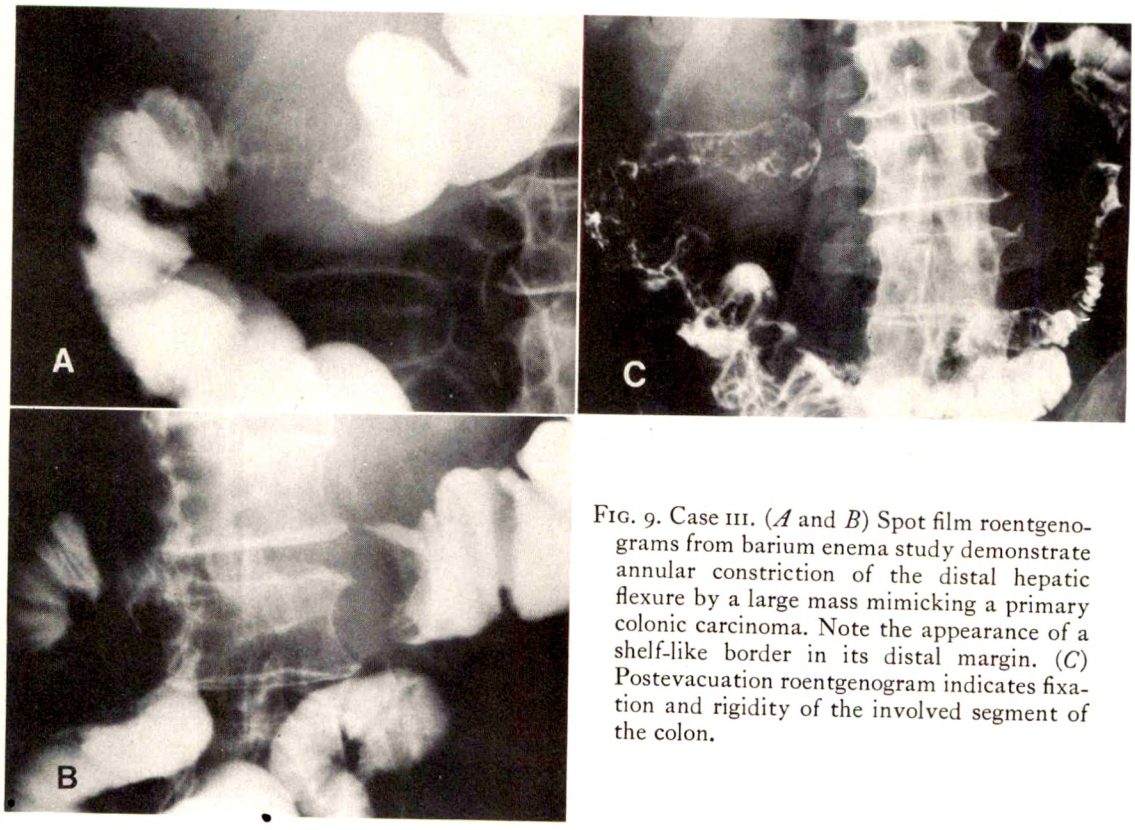

FIG. 9. Case III. (*A* and *B*) Spot film roentgenograms from barium enema study demonstrate annular constriction of the distal hepatic flexure by a large mass mimicking a primary colonic carcinoma. Note the appearance of a shelf-like border in its distal margin. (*C*) Postevacuation roentgenogram indicates fixation and rigidity of the involved segment of the colon.

An upper gastrointestinal series demonstrated extrinsic compression of the postbulbar duodenum from its lateral aspect (Fig. 12*C*). The mucosal folds in the medial wall of the descending duodenum also appeared irregular and distorted, and there was narrowing of the third part of the duodenum. The latter findings suggested possibility of a pancreatic carcinoma.

On exploratory laparotomy an infiltrative adenocarcinoma of the gallbladder directly extending into the adjacent liver, duodenum, and right colic flexure was found. There were metastases to the hepatic hilar lymph nodes and in the proximal hepatic flexure where the initial lesion was noted. Because of the extent of the malignancy, only biliary drainage was undertaken. The patient expired a few weeks later following rapid deterioration of her condition.

Comment. The malignancy commonly infiltrates all layers of the gallbladder wall and rapidly extends into the surrounding structures. Therefore, the anatomic relationships of the gallbladder to adjacent viscera become an important factor for the spread of cancer, production of clinical symptoms, and feasibility of surgical treatment.[2,8,10,13,15] In a series of 78 cases seen at Peter Bent Brigham Hospital, Litwin[13] found contiguous invasion of the liver by carcinoma in 63 per cent. The colon was involved in 22 per cent while spread to the duodenum and omentum each occurred in 26 per cent of patients. The experience of Fahim *et al.*[2] at the Mayo Clinic has been similar. Up to 15 per cent of patients with carcinoma of the gallbladder will show evidence of colonic invasion on barium enema examination.[8,15] While involvement of the colon (Fig. 12, *A–C*; and 13) is a late mani-

Fig. 11. Cholecysto-colic fistula due to chronic calculous cholecystitis. Barium enema study shows a wide communication between the fundus of the gallbladder and the narrowed adherent segment of the hepatic flexure. Note the presence of stones within the gallbladder and the reflux of barium into the common bile duct.

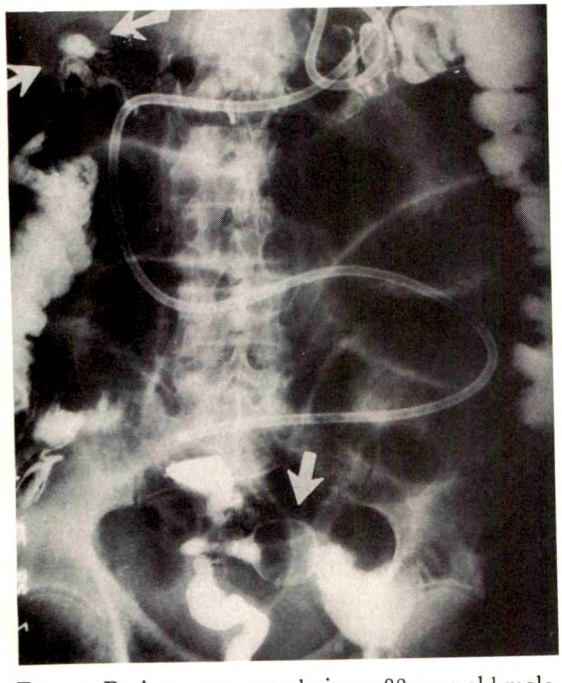

Fig. 10. Barium enema study in an 88 year old male shows an impacted gallstone (arrow) in the sigmoid colon, with distended intestinal loops proximally. The cholecysto-colic fistula is partially opacified (arrows).

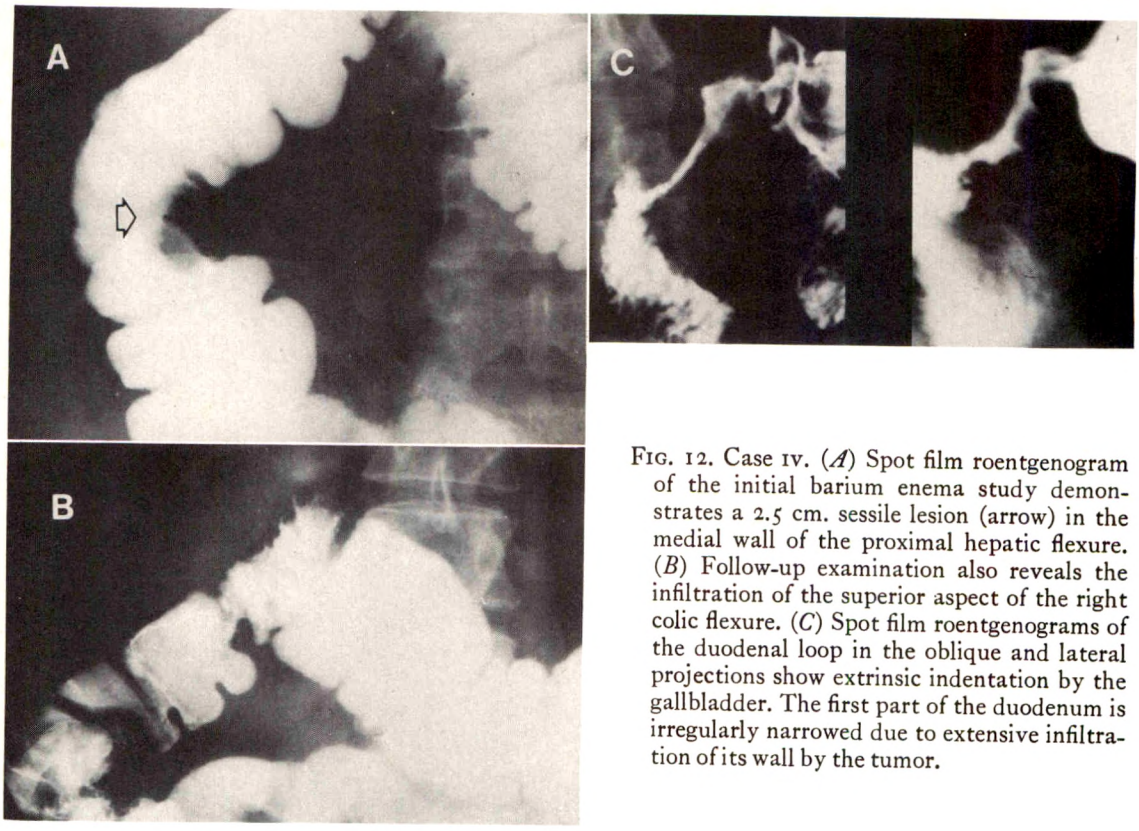

FIG. 12. Case IV. (*A*) Spot film roentgenogram of the initial barium enema study demonstrates a 2.5 cm. sessile lesion (arrow) in the medial wall of the proximal hepatic flexure. (*B*) Follow-up examination also reveals the infiltration of the superior aspect of the right colic flexure. (*C*) Spot film roentgenograms of the duodenal loop in the oblique and lateral projections show extrinsic indentation by the gallbladder. The first part of the duodenum is irregularly narrowed due to extensive infiltration of its wall by the tumor.

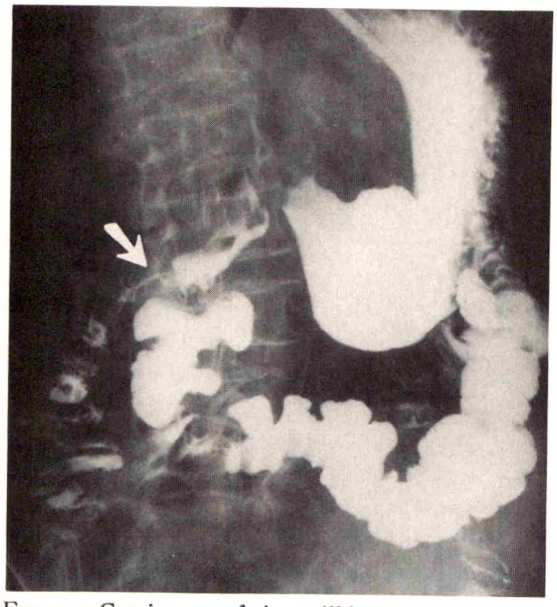

FIG. 13. Carcinoma of the gallbladder resulting in fistulous tract (arrow) between the right colic flexure and the postbulbar segment of the duodenum which are infiltrated by the tumor.

festation of the disease, it may also be the reason for initial clinical presentation as exemplified in Case IV.

The spectrum of findings noted on barium enema examination depends on factors such as the location of the carcinoma in the gallbladder and the extent of cholecystocolic relationship.[8,15] Infiltration of the superior aspect of the hepatic flexure primarily occurs when the carcinoma originates in or involves the inferior surface of the body and fundus of the gallbladder (Fig. 12, *A–C*; and 13). A carcinoma developing in the neck of the gallbladder has a greater tendency to extend into and obstruct the cystic and common bile ducts. Therefore, barium enema examination may show only an impression of an enlarged gallbladder (Fig. 14, *A* and *B*) while duodenal involvement is readily apparent on the upper gastrointestinal series.[8,10,15] Both examinations are invaluable and comple-

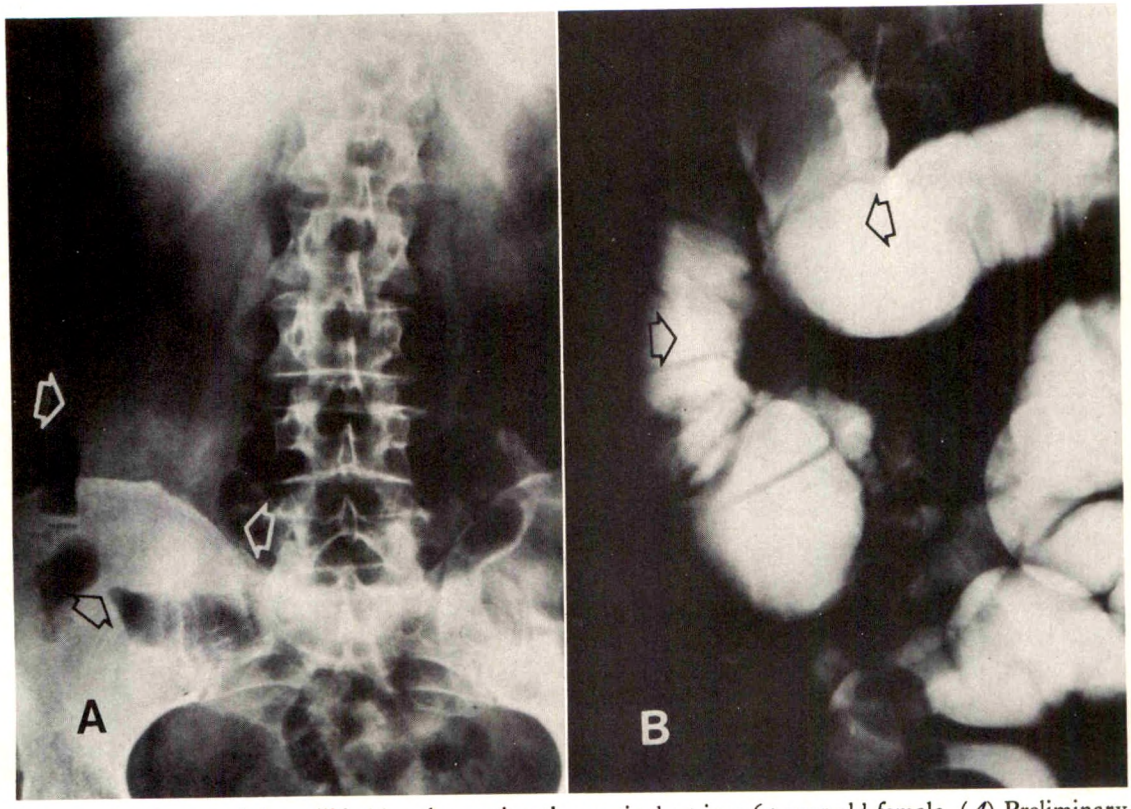

Fig. 14. Carcinoma of the gallbladder obstructing the cystic duct in a 65 year old female. (*A*) Preliminary roentgenogram of the abdomen in the supine position shows a markedly enlarged gallbladder (arrows) as outlined by inferior displacement of the hepatic flexure of the colon. (*B*) Barium enema study reveals impression of the distended gallbladder (arrows) upon the anterior surface of the hepatic flexure.

ment each other in providing a correct diagnosis.

Internal biliary fistulae occasionally occur with carcinoma of the gallbladder when the necrotic tumor perforates into the adjacent duodenum or colon or both.[3,15] As in our patient (Fig. 13), the clinical presentation can be diarrhea, melena and loss of weight. Although evidence of pneumobilia and irregular gas collections within the tumor may permit a plain film roentgenographic diagnosis, barium studies of the gastrointestinal tract and the colon are usually required to outline the fistula.[10,15] Differentiation of the basic etiology, namely cholecystitis from carcinoma, is often difficult on a roentgenographic basis. Since carcinoma of the gallbladder is usually superimposed upon chronic cholecystitis with or without stones, the presence of pericholecystic adhesions is often relevant for producing an even closer pathway for extension of the tumor into the colon. When there are extensive mucosal destruction and tumefaction of the involved colon, the findings clearly indicate the presence of gallbladder malignancy.

DISCUSSION

The unique anatomic relationships between the colon and other organs have been the subject of several recent pathologic-roentgenographic studies.[16–21,29,30] Both the inflammatory and neoplastic processes of adjacent viscera often secondarily involve the large intestine using anatomic crossroads.[2,3,13,17–21] In a significant number of patients, barium enema examination is the initial roentgen study with findings which lead to correct recognition of the primary

disease in other digestive organs, or in the genitourinary tract.[1,4,17,18]

The colonic manifestations of gallbladder disorders deserve particular attention because of the prevalence of biliary tract diseases and their often nonspecific or misleading clinical presentation.[8,12,13] Indeed, classic symptoms of large intestinal disease, *e.g.*, rectal bleeding, diarrhea, cramping abdominal pain and weight loss are not rare in gallbladder disorders, as was also true in our patients.[7,9,25,27]

Basic to the appreciation of alterations illustrated on barium enema studies in this report is an understanding of the intimate cholecysto-colic relationships which influence the spread of pathologic conditions. The gallbladder and the right colic flexure are closely related in most individuals. The degree of proximity of the 2 organs is even further enhanced when pericholecystic adhesions prevail. The extent of abnormalities noted on a barium enema examination of the colon often clearly reflects the severity of the pathologic process in the gallbladder. Thus acute cholecystitis results only in extrinsic compression and functional changes of the adjacent colon, which may be reversible (Fig. 3, *A* and *B*; and 4, *A* and *B*). Actual infiltration of the wall of the adjacent colon accompanies both chronic cholecystitis[1,7,9,13,24] and carcinoma of the gallbladder.[2,8] In most instances, involvement by an inflammatory or neoplastic process appears localized to the superior aspect of the anterior hepatic flexure (Fig. 3; 5; 11–13), but circumferential involvement of the lumen (Fig. 9, *A–C*) mimicking primary carcinoma of the colon may also occur.[1,9,24] The barium enema findings of chronic cholecystitis and carcinoma of the gallbladder are at times quite similar. In fact, a carcinoma may develop upon the preexisting chronic cholecystitis, and either disease may be complicated by cholecystocolic fistulae. Diarrhea and loss of weight were primary complaints in 3 of our 4 patients with biliary fistula detected during barium enema study. Preliminary roentgenograms of the abdomen also showed air in the region of the gallbladder in 3 cases, all of whom had cholecystocolic fistula due to chronic cholecystitis. Carcinoma of the gallbladder resulted in a duodenocolic fistula in 1 patient (Fig. 13). The communication with the gallbladder itself was incompletely shown on preoperative roentgenograms because of its obliteration by the tumor. It should be noted that duodenocolic fistulae are usually a complication of carcinoma of the right colic flexure rather than peptic ulcers or biliary tract diseases.[4,22,28]

Pseudotumors of the colon in patients with chronic cholecystitis (Fig. 5; 6; and 9) have also been observed by other authors.[1,7,9,24] The larger annular lesions as illustrated in Case III are manifestations of a pericholecystic abscess directly extending into the colon wall and resulting in epiplopericolitis.[1,24] Smaller pseudotumors (Fig. 5*A*; and 6) may be caused by pericholecystic adhesions and the related inflammatory or ischemic process in appendices epiploicae.[7,11,23]

Certain roentgenologic features have proved helpful in recognition of the secondary nature of colonic abnormalities shown in our patients and in cases previously described in the literature.[1,9,19,24] A notable sign is preservation of the colonic mucosal folds, although they may appear crowded and increased in number due to a localized contraction of the colon and desmoplastic response.[1,9,19] On careful inspection many such folds appear perpendicular to the longitudinal axis of the colon (Fig. 9*A*), with gradual transition toward the adjacent normal segments. Angulated axes of the folds may reflect adhesions and the fibrous reaction. Normal progression of peristalsis or occurrence of spasm and tonic contractions is usually seen in the involved segment during fluoroscopy. Complete obstruction of the intestinal lumen is highly unusual with either cholecystitis or carcinoma of the gallbladder.[1,7,15] These findings are in sharp contrast to those associated with a primary carcinoma of the colon which characteristically involves a shorter

segment of irregular stenosis with shelving defects and destroyed mucosa. Kent[9] has also pointed out that on postevacuation roentgenograms the retention of barium proximal to a constricting lesion of the colon is a frequent and useful clue in the diagnosis of a primary carcinoma.

As in cases illustrated herein, lesions originating in the gallbladder invariably present above the transverse mesocolon and affect the superior or medial aspects of the right colic flexure.[3,15,22,29,30] On the basis of the vector principle described by Whalen and co-workers,[29] one can easily differentiate colonic involvement by gallbladder lesions from those caused by other processes. For example, masses arising in the right kidney may elevate the proximal transverse colon, while gastric carcinoma and pancreatic lesions involve the more distal segment of the transverse colon.[9,18–21,29] The roentgenologic differential diagnosis can be even further refined because specific haustral rows of the colon are primarily affected in each condition, as recently shown by Meyers *et al.*[20] These authors have pointed out the close relationship of the body of the gallbladder to those haustra which are located between the taeniae mesocolica and omentalis of the adjacent colon.[20]

A barium study of the colon can provide complementary diagnostic evidence of gallbladder disease in certain instances when a multiplicity of causes for nonopacification of the gallbladder on cholecystography has created problems in reaching a definite conclusion. Awareness of the spectrum of secondary colonic manifestations and their correlation with other clinical and roentgen studies can be of critical importance for correct assessment of the underlying gallbladder disease and its therapeutic management.

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REFERENCES

1. Di Rienzo, S., and Mosca, L. G. Epipolericolitis: significance of its roentgenological diagnosis. Am. J. Roentgenol. & Rad. Therapy, 1951, *66*, 215–221.
2. Fahim, R. B., McDonald, J. R., Richards, J. C., and Ferris, D. O. Carcinoma of gallbladder: study of its modes of spread. *Ann. Surg.*, 1962, *156*, 114–124.
3. Glenn, F., and Mannix, H., Jr. Biliary enteric fistula. *Surg., Gynec. & Obst.*, 1957, *105*, 693–705.
4. Hall Drake, E. P., and Goodwin, J. F. Duodenocolic fistula complicating carcinoma coli. *Brit. J. Surg.*, 1948, *36*, 204–207.
5. Hoffman, G. L., Jr., von Kessel, K. F. L., and Hoerr, S. O. Obstruction of small intestine caused by acute cholecystitis: report of case. *A.M.A. Arch. Surg.*, 1958, *76*, 412–413.
6. Jacobson, H. G., Shapiro, J. H., Stern, W., and Poppel, M. H. Positional relation of gallbladder to hepatic flexure. *Am. J. Digest. Dis.*, 1956, *1*, 294–300.
7. Kaplan, B. J. Massive lower gastrointestinal hemorrhage from cholecystocolic fistula. *Dis. Colon & Rectum*, 1967, *10*, 191–196.
8. Keill, R. H., and De Wesse, M. S. Primary carcinoma of gallbladder. *Am. J. Surg.*, 1973, *125*, 726–729.
9. Kent, K. H. Extrinsic lesions affecting transverse colon. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *89*, 779–786.
10. Khilnani, M. T., Wolf, B. S., and Finkel, M. Roentgen features of carcinoma of gallbladder on barium meal examination. *Radiology*, 1962, *79*, 264–273.
11. Kyaw, M. M., and Koehler, P. R. Pseudotumors of colon due to adhesions. *Radiology*, 1972, *103*, 597–599.
12. Lame, E. L. Chronic cholecystitis: indications for revision of clinical diagnosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1962, *87*, 736–739.
13. Litwin, M. S. Primary carcinoma of gallbladder: review of 78 patients. *A.M.A. Arch. Surg.*, 1967, *95*, 236–240.
14. Marshak, R. H., and Henkin, W. Cholecystocolic fistula. *Radiology*, 1949, *53*, 555–558.
15. McConnel, F. Malignant neoplasm of gallbladder: roentgenological diagnosis. *Radiology*, 1957, *69*, 720–725.
16. Meyers, M. A. Peritoneography: normal and pathologic anatomy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 353–365.
17. Meyers, M. A. Colonic changes secondary to left perinephritis: new observations. *Radiology*, 1974, *111*, 525–528.
18. Meyers, M. A., and Evans, J. A. Effects of pancreatitis on small bowel and colon: spread along mesenteric planes. Am. J. Roentgenol.,

RAD. THERAPY & NUCLEAR MED., 1973, *119*, 151–165.

19. MEYERS, M. A., and MCSWEENEY, J. Secondary neoplasms of bowel. *Radiology*, 1972, *105*, 1–11.
20. MEYERS, M. A., VOLBERG, F., KATZEN, B., and ABBOTT, G. Haustral anatomy and pathology: new look. II. Roentgen interpretation of pathological alterations. *Radiology*, 1973, *108*, 505–512.
21. MEYERS, M. A., and WHALEN, J. P. Roentgen significance of duodenocolic relationships: anatomic approach. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *117*, 263–274.
22. NEVILLE, W. E. Duodenocolic fistula due to acute cholecystitis. *Am. J. Surg.*, 1954, *87*, 300–302.
23. OVERTON, R. C., BOLTON, B. F., and USHER, F. C. Extrinsic deformities of colon mimicking carcinoma: report of three cases. *Surgery*, 1954, *36*, 906–915.
24. ROCKOFF, S. D., and TUDDENHAM, W. J. Adherent gallbladder simulating intrinsic lesion of right side of colon: report of case. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1962, *87*, 749–751.
25. SAFAIE-SHIRAZI, S., ZIKE, W. L., and PRINTEN, K. J. Spontaneous enterobiliary fistulas. *Surg., Gynec. & Obst.*, 1973, *137*, 769–772.
26. SCANLAN, R. L., and YOUNG, B. R. Roentgen diagnosis of gallbladder and biliary tract disease without cholecystography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1954, *72*, 639–643.
27. STAHL, W. M., JR. Gastrointestinal tract hemorrhage due to gallbladder disease. *New England J. Med.*, 1959, *260*, 471–474.
28. WAGGONER, C. M., and LEMONE, D. V. Clinical and roentgen aspects of internal biliary fistulas: report of twelve cases. *Radiology*, 1949, *53*, 31–41.
29. WHALEN, J. P., EVANS, J. A., and MEYERS, M. A. Vector principle in differential diagnosis of abdominal masses. II. Right upper quadrant. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *115*, 318–333.
30. WHALEN, J. P., and RIEMENSCHNEIDER, P. A. Analysis of normal anatomic relationships of colon as applied to roentgenographic observations. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *99*, 55–61.

VOL. 125, No. 1

PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY*

PROBLEMS IN INTERPRETATION

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ABSTRACT:

The patient who presents with jaundice, regardless of the etiology, cannot be adequately examined by a gallbladder series and intravenous cholangiography. Clinical evaluation aided by laboratory analysis will not always differentiate between primary liver parenchymal disease and biliary tract obstruction. Percutaneous transhepatic cholangiography, when successfully performed, answers the question of whether the jaundice is due to primary liver parenchymal disease or due to biliary tract obstruction. The point to emphasize is that under no circumstances is it appropriate to presume any information if the biliary system is not entered and visualized successfully.

The proper interpretation of the level of block can fall prey to the mistake of incomplete aspiration of the thick, inspissated bile in the obstructed biliary tree before injection of contrast material. The problem will be worsened by a peripheral entrance into the biliary system rather than a more central one. Gastrointestinal series should always be available to aid in identifying pathology at the entrance of the common bile duct into the duodenum. Lastly, identifying the etiology at the site of the block will require additional procedures. Selective visceral angiography has contributed greatly in this area. During the 20 years of clinical usage of percutaneous transhepatic cholangiography, we feel it has become the single examination capable of preventing unnecessary exploration of the jaundiced patient with primary liver parenchymal disease and the most useful potential source of practical information if laparotomy is necessary to correct biliary tract obstruction.

PERCUTANEOUS transhepatic cholangiography is one of the older methods of visualizing the biliary tree. In the half century since its inception, its use has waxed and waned primarily because oral cholecystography proved to be safer and better. However, in the last 20 years with the widespread use of image intensifiers a visually controlled method has regained popularity for the study of the biliary tree in jaundiced patients to determine if there is mechanical extrahepatic block of the biliary tree, the location of the extrahepatic block, and finally, what is causing the block.

Numerous extensive reports on percutaneous transhepatic cholangiography have been published.[11,12,14] Methods and techniques have been studied in detail.[5] The risk was low in the early series and has continued to decrease.[3,4,7] The information obtained has been strikingly reliable. Patients have been repeatedly saved from laparotomy when the extrahepatic biliary system was shown to be widely patent. Identification of the site of extrahepatic block has been extremely helpful in the planning of the surgical approach and definitive treatment. The etiological agents have been divided into 3 categories according to the type of defect created on the transhepatic cholangiogram: stones, malignant tumor, either primary or metastatic, and finally, stricture. However, it

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FIG. 1. Case 1. Gastrointestinal examination demonstrated effacement of the mucosa (arrow) along the inner margin of the duodenal sweep in the region of the pancreatic head.

is accepted that no matter how soundly established a diagnostic procedure is in modern radiology, if performed often enough, sporadic, serious diagnostic problems will occur. We have selected the following group of cases to emphasize the difficulties which may arise in determining the site of obstruction and the cause of obstruction.

REPORT OF CASES

CASE 1. This was the first hospital admission of a 50 year old white man who entered with a history of 6 months of weakness and 3 weeks of jaundice. Physical examination revealed a well developed man with jaundice. Pertinent laboratory material revealed elevated bilirubin and alkaline phosphatase levels. A gastrointestinal series demonstrated effacement of the mucosa (arrow) along the inner margin of the duodenal sweep in the region of the pancreatic head (Fig. 1). A diagnosis of extrahepatic common duct obstruction secondary to carcinoma was made. A percutaneous transhepatic cholangiogram on the morning of operation suggested a complete block at, or close to, the porta hepatis (Fig. 2). The aspirated bile was dark green and extremely viscous. The most likely diagnosis was carcinoma of the pancreas, metastatic to periportal lymph nodes. At operation, a 4×5 cm. mass in the head of the pancreas was identified causing complete block of the common duct just above the ampulla of Vater. There was no evidence of metastatic disease in the porta hepatis. Histologic examination revealed a carcinoma of the head of the pancreas.

In retrospect, the technical diagnostic problem here centered around the inability of the contrast material to filter through the dense inspissated bile down to the true site of block. There is no question that from many points of view, regardless of the thickness of the contents of the biliary tree, an extensive aspiration should be attempted: first, to relieve pressure from the biliary system; second, to make the injection of contrast material more comfortable for the patient by preventing any further distention of the common duct and biliary system; and lastly, so that the contrast material can reach the actual site of block, rather than pool behind a dense column of heavy, inspissated biliary material.

CASE II. This 57 year old white man was admitted to the hospital with a 10 day history of lower abdominal pain associated with intermittent constipation, grayish stools and dark

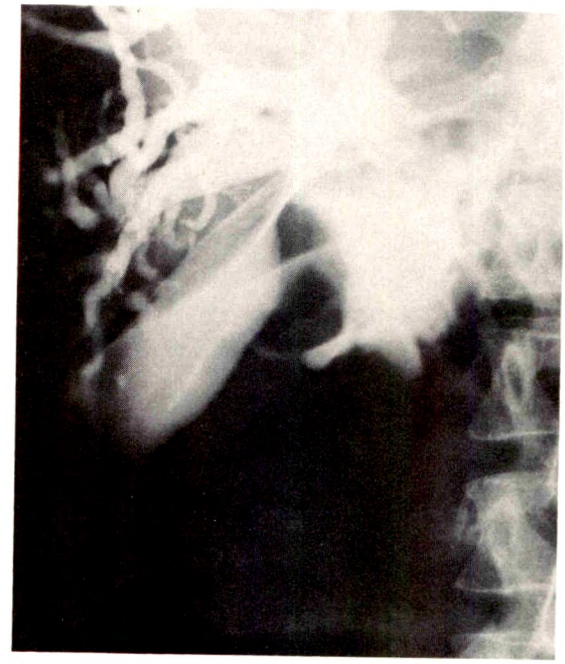

FIG. 2. Case 1. Percutaneous transhepatic cholangiography suggested a complete block at, or close to, the porta hepatis.

urine. Laboratory values indicated elevated levels of amylase, bilirubin and alkaline phosphatase. Shortly after admission a distended gallbladder was noted.

Gastrointestinal series showed marked pressure (arrow) on the postbulbar area of the duodenal sweep (Fig. 3). A reverse 3 sign in the descending duodenum was noted. Hypotonic duodenography minimized the findings (Fig. 4); however, some pressure along the inner margin of the sweep remained. Percutaneous transhepatic cholangiography suggested a high porta hepatis block. The needle puncture was in the peripheral left hepatic duct radicles. Note the extrinsic pressure defect (arrow) in the more central left hepatic duct (Fig. 5). Decompression of the biliary system via the needle in this location was limited.

At operation a 2.5 cm. carcinoma of the pancreatic head was identified with obstruction of the distal intrapancreatic common duct. No metastatic disease was present. Again, failure to visualize the entire common duct down to the distal block seemed best explained by poor aspiration of the biliary system before attempts to place contrast material into the ducts. A more central puncture would have eliminated the problem by allowing better drainage of the obstructed system.

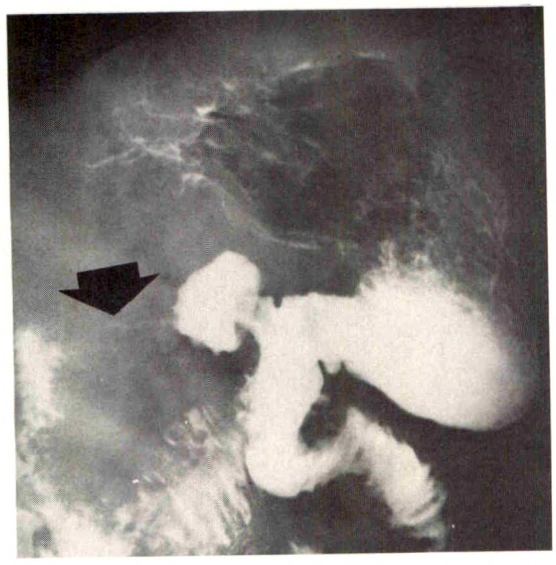

Fig. 3. Case II. Gastrointestinal series showed marked pressure (arrow) on the postbulbar area of the duodenal sweep. A reverse 3 sign of the descending duodenum was noted.

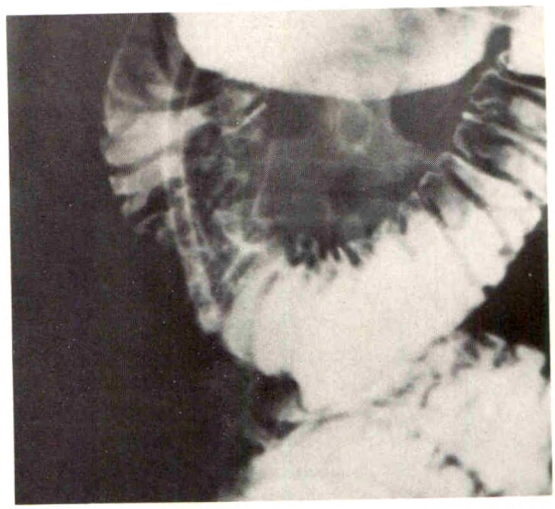

Fig. 4. Case II. Hypotonic duodenography minimized the findings, however, some pressure along the inner margin of the sweep remained.

Case III. This 70 year old woman entered the hospital deeply jaundiced, anorexic with markedly elevated bilirubin, alkaline phosphatase, LDH, and SGOT values. Five years prior to admission she had had a cholecystectomy for cholelithiasis. Incidentally noted then was a microscopic carcinoma of the gallbladder.

Gastrointestinal series showed pressure in the postbulbar area (arrow) consistent with a dilated common duct (Fig. 6). Percutaneous transhepatic cholangiography showed mild dilatation of the right hepatic radicles and more severe dilatation of the left hepatic ducts with a narrowed area in the proximal left hepatic duct (arrow). Multiple calculi in the distal dilated common duct were identified. Only partial obstruction to the biliary drainage was present. Narrowing of the left main hepatic duct suggested the possibility of recurrent carcinoma of the gallbladder or a benign stricture (Fig. 7 and 8). At operation benign stricture was found. Three stones were removed from the common duct. In this case 2 abnormal areas suggested the possibility of separate etiologies. Only at operation could recurrent carcinoma of the gallbladder be properly ruled out as the cause of the narrowing of the proximal left hepatic duct. Two diagnostic points can be made here. First, choledocholithiasis can present as either defects due to the actual presence of stones or as areas of narrowing secondary to trauma to the ductal system. Second, single or multiple localized narrowings of the

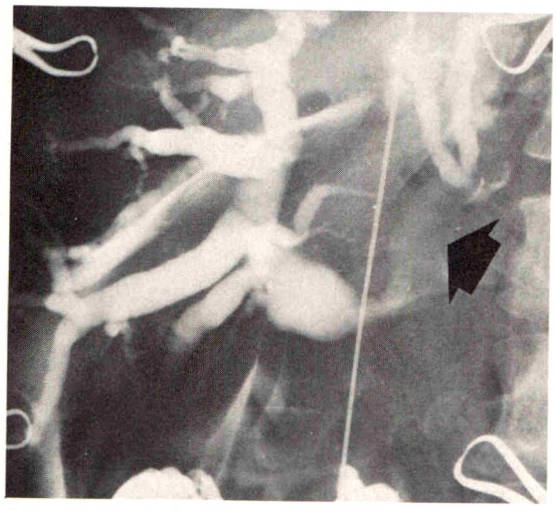

FIG. 5. Case II. Percutaneous transhepatic cholangiography suggested a high porta hepatis block. The needle puncture was in the periphery of the left hepatic duct radicles. Note the extrinsic pressure defect (arrow) in the more central left hepatic duct. Decompression of the biliary system via the needle in this location was limited.

biliary system can be caused by numerous diseases, as will be discussed in Cases VI, VII, and VIII.

CASE IV. This 53 year old Cuban woman presented with 2 month history of right upper quadrant pain, 2 weeks of increasing jaundice with light colored stools and 1 week of increasingly dark urine. Laboratory data revealed elevated levels of alkaline phosphatase and bilirubin, and a markedly increased prothrombin time. Liver biopsy showed acute pericholangitis and bile stasis consistent with biliary obstruction.

Percutaneous transhepatic cholangiography revealed an eccentrically tapered distal common duct which had the appearance of a meniscus sign on some films and an irregular defect suggesting tumor on others. The presence of a large stone in the cystic duct remnant (arrow) favored the presence of a second stone obstructing the common duct (Fig. 9 and 10). At operation, only stones were found proximally and distally.

CASE V. This 62 year old female entered the hospital with a 4 week history of deepening jaundice. The bilirubin and alkaline phosphatase values were elevated; the liver was markedly enlarged.

A gastrointestinal series showed a large lesser curvature ulcer (arrow) of the duodenal sweep at the junction of the second and third portion and a large extrinsic pressure defect along the

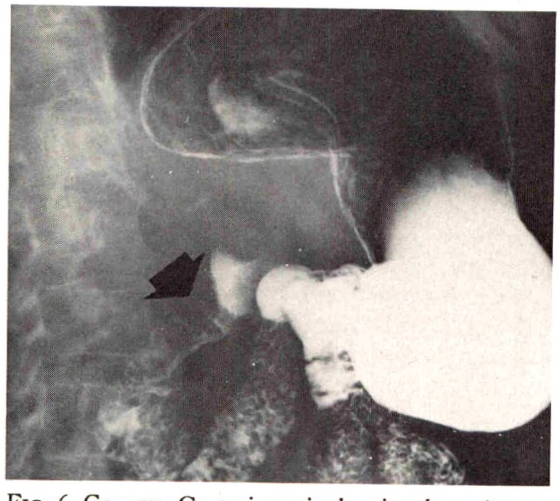

FIG. 6. Case III. Gastrointestinal series showed pressure in the postbulbar area (arrow) consistent with a dilated common duct.

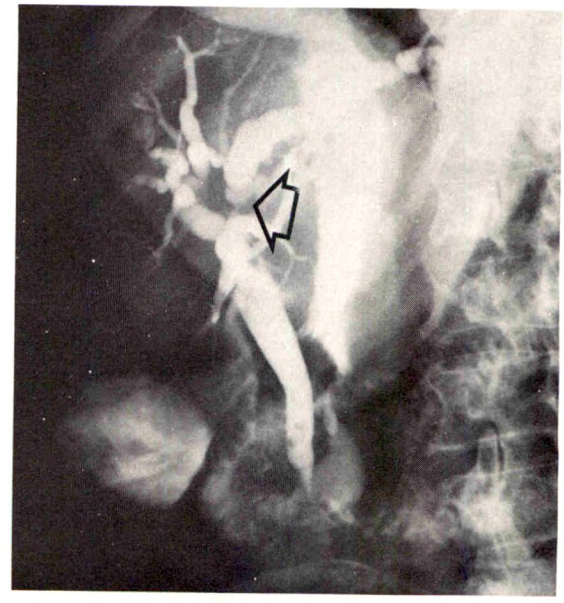

FIG. 7. Case III. Percutaneous transhepatic cholangiography showed a mild dilatation of the right hepatic radicles and more severe dilatation of the left hepatic ducts with a narrowed area at the proximal left hepatic duct (arrow). Multiple calculi in the distal dilated common duct were identified. Narrowing of the left main hepatic duct suggested the possibility of recurrent carcinoma of the gallbladder or a benign stricture.

greater curvature of the proximal second portion of the duodenum (Fig. 11 and 12). A percutaneous transhepatic cholangiogram demonstrated a massively dilated common duct (arrow) and gallbladder. Again contrast material did not reach the distal common duct suggesting a mid-duct block (Fig. 13).

At operation, a well localized carcinoma of the ampulla of Vater was found. This is another example which demonstrates the difficulty in establishing the site of block.

Better aspiration of the common bile duct undoubtedly would have allowed more distal filling of the duct. The clue here is the hazy interface between the contrast medium and the bile indicating mixing of the two, rather than contrast material against a solid obstruction. However, even more significant is the fact that the primary lesion was carcinoma of the ampulla of Vater, clearly demonstrated by gastrointestinal series. The presence of this lesion clearly indicated the level of the block. Gastrointestinal series should always precede a percutaneous transhepatic cholangiogram, since biliary disease is often secondary to primary gastrointestinal pathology. This is also seen in Case IX.

Fig. 9. Case IV. Percutaneous transhepatic cholangiography revealed an eccentrically tapered distal common duct which had the appearance of the meniscus sign on some films and an irregular defect suggesting tumor on others. The presence of a large stone in the cystic duct remnant (arrow) favored the presence of a second stone obstructing the common duct.

Case VI. This 63 year old woman was admitted with the diagnosis of recurrent extrahepatic biliary obstruction. Four and one half months prior to admission the patient had been admitted with a history of jaundice, dark urine, and light stools. She was explored, and fibrosis of the extrahepatic biliary tree was defined. The dense fibrotic process involved the common hepatic duct and common bile duct causing extremely narrowed areas which were

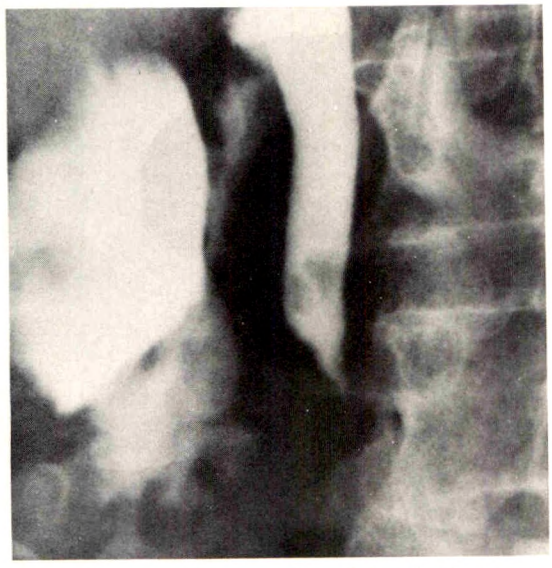

Fig. 8. Case III. Close-up view of the distal common duct showing the multiple calculi.

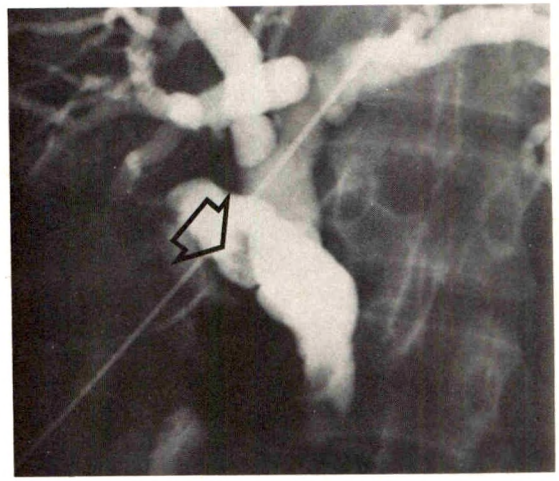

Fig. 10. Case IV. Close-up view of the distal common duct. Arrow marks position of a large stone in the cystic duct remnant.

FIG. 11. Case v. Gastrointestinal series showed a large lesser curvature ulcer (arrow) of the duodenal sweep at the junction of the second and third parts of the duodenum and a large extrinsic pressure defect along the greater curvature of the proximal second part of the duodenum.

freed with difficulty. Following the exploration, the bilirubin fell to the upper limits of normal. Shortly after discharge she required a second admission for recurrent jaundice. Intravenous pyelography showed a dilated right kidney collecting system as a result of ureteropelvic obstruction (Fig. 14). A percutaneous transhepatic cholangiogram revealed a complete obstruction just distal to the junction of the right and left hepatic ducts (arrow). Selective arteriograms were normal which pointed away from tumor. The appearance of the obstruction favored the diagnosis of tumor (Fig. 15). At laparotomy, the ureteropelvic obstruction of the right kidney was due to retroperitoneal fibrosis. The ureter was dissected from the dense fibrotic process and re-anastomosed to the kidney pelvis. The hyalinizing fibrosclerosis was also demonstrated surrounding the extrahepatic tree and was clearly the cause of the complete block of the common bile duct.

The history of fibrosis at previous exploration and the current involvement of the right kidney ureteropelvic junction should have pointed clearly to multifocal sclerosing fibrosis. Its presence in more than one organ system is characteristic and the roentgenologic picture often resembles the classic appearance of malignancy.

CASE VII. This 61 year old female presented with dizziness, blurred vision, markedly decreased appetite, scleral icterus, and increasing weakness. Elevated bilirubin and alkaline phosphatase levels were noted on admission.

A gastrointestinal series showed a smooth defect along the lesser curvature of the antrum

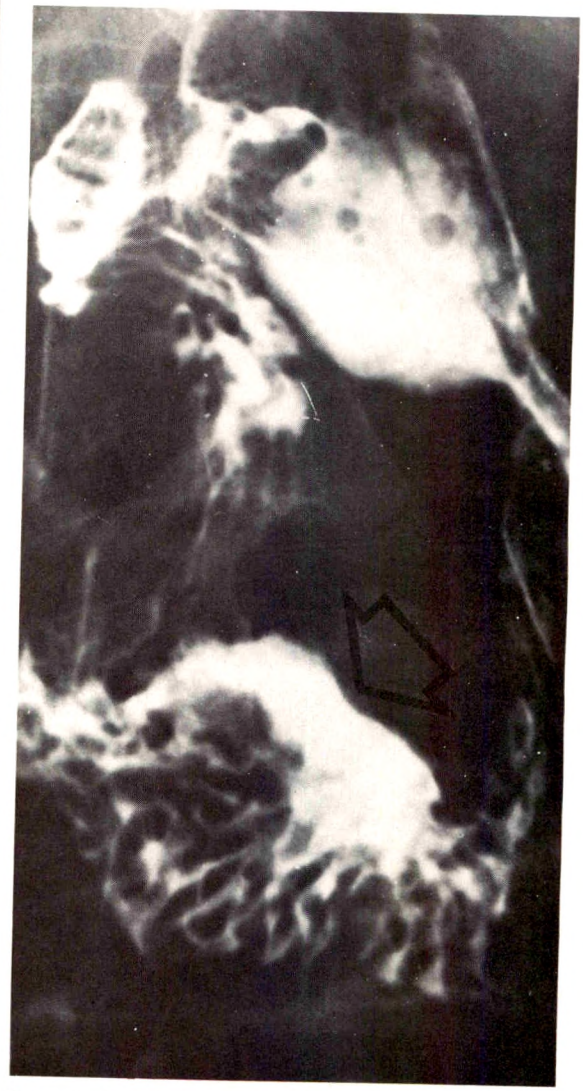

FIG. 12. Case v. Close-up view of the second part of the duodenum with the large extrinsic pressure defect along the greater curvature of the proximal duodenum and the lesser curvature ulcer (arrow) at the junction of the second and third parts of the duodenum.

(Fig. 16). Percutaneous transhepatic cholangiography showed a long area of stenosis of the mid common bile duct (arrows) extending to the junction of the right and left hepatic ducts strongly suggesting tumor (Fig. 17).

At operation carcinoma of the gallbladder was found with diffuse involvement of the regional lymph nodes causing marked narrowing of the common duct. The patient was decompressed via a drainage tube placed in the junction of the right and left hepatic ducts. When this tube was injected with contrast material, it was noted that the dilatation of the left biliary system (arrow) was greater than on the right (Fig. 18).

Here again the difficulty in dealing with a narrow area in the biliary system was demonstrated. Primary malignancy of the common duct or gallbladder, and metastases to regional lymph nodes were foremost in the differential diagnosis. However, benign tumor of the common duct, as seen in Case x, inflammatory disease, secondary effects of calculi in the biliary tree, and finally multifocal fibrosclerosis (sometimes referred to as sclerosing cholangitis) must all be considered in the differential of what can appear to be a relatively benign, smooth, narrowed area. Stricture formation in the biliary system probably represents the most difficult differential diagnostic problem in percutaneous transhepatic cholangiography.

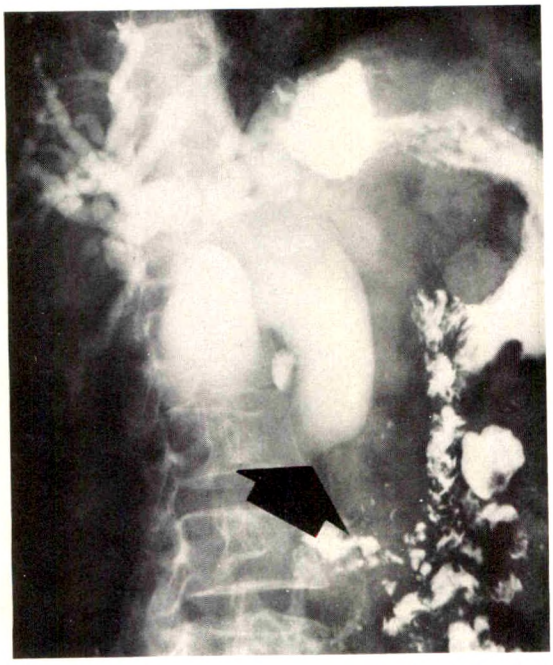

Fig. 13. Case v. Percutaneous transhepatic cholangiogram demonstrated a massively dilated common duct (arrow) and gallbladder. Contrast material did not reach the distal common duct suggesting a mid-duct block.

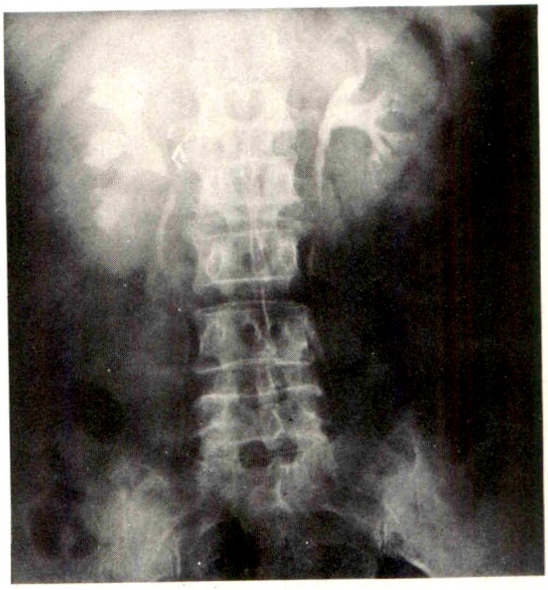

Fig. 14. Case vi. Intravenous pyelogram showed a dilated right kidney collecting system as a result of ureteropelvic obstruction.

Case viii. This 59 year old heroin addict was admitted for methadone detoxification with a history of 6 weeks of weight loss, vomiting, decreasing appetite, and increasingly dark urine. He was a thin jaundiced male with an enlarged, markedly nodular and tender liver. He had elevated bilirubin and alkaline phosphatase levels. Transhepatic cholangiography showed irregular areas of narrowing throughout the biliary tree with some dilatation of the distal left hepatic duct to a level 1 cm. from the porta hepatis (arrow, Fig. 19). The common hepatic duct and common bile duct were also markedly narrowed. Contrast material appeared to flow into the duodenum without significant delay. In view of the free flow of contrast medium into the duodenum, the jaundice was probably related to intrahepatic disease. The differential diagnosis on transhepatic cholangiography included sclerosing cholangitis and diffuse metastatic disease. In the hospital the patient developed a pleural effusion; his condition rapidly deteriorated and he died approximately a month following admission.

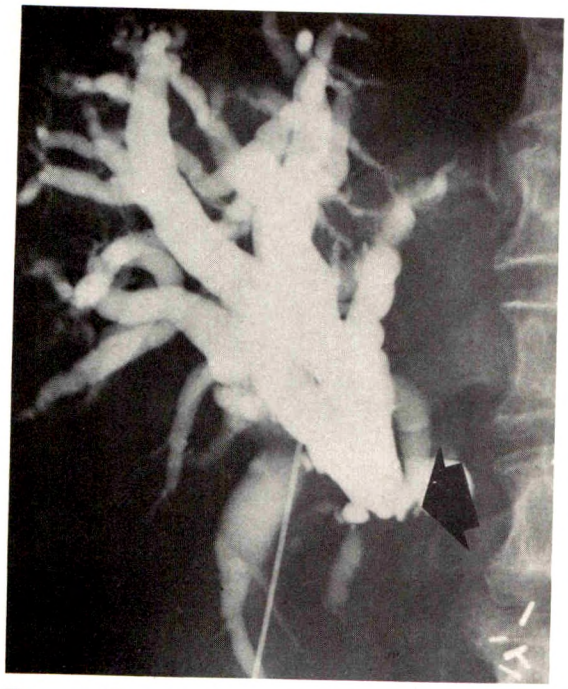

FIG. 15. Case VI. Percutaneous transhepatic cholangiogram revealed a complete obstruction just distal to the junction of the right and left hepatic ducts (arrow). The appearance of the obstruction favored the diagnosis of tumor.

Liver biopsy just before death revealed diffuse adenocarcinoma of the liver, explaining the multiple areas of narrowing in the biliary system. Because of the history of heroin addiction it was felt during his hospitalization that the best interpretation of the transhepatic cholangiogram was a form of fibrosing cholangitis rather than diffuse tumor. The problem of interpreting extensive areas of narrowing in the biliary system is again emphasized in this case.

FIG. 16. Case VII. Gastrointestinal series showed a smooth defect along the lesser curvature of the antrum.

CASE IX. This 29 year old man presented with a 2 day history of nausea, vomiting, and right upper quadrant pain. One year earlier he had had a cholecystectomy for cholecystitis secondary to cholelithiasis. On this admission he had marked right upper quadrant tenderness with elevated bilirubin and alkaline phosphatase levels.

A gastrointestinal series showed a large filling defect (arrow) in the second part of the duodenum in the region of the ampulla with marked mucosal effacement (Fig. 20 and 21). Extrinsic pressure was noted along the greater curvature of the second part of the duodenum (open arrow). Percutaneous transhepatic cholangiography revealed a dilated collecting system,

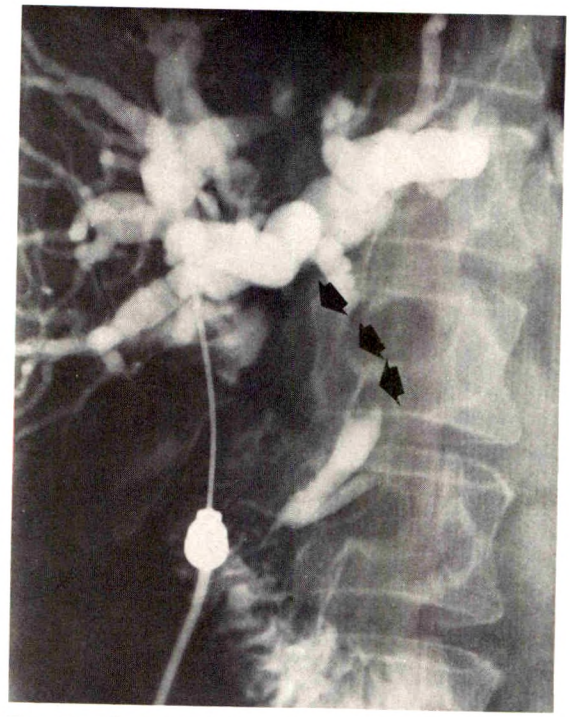

FIG. 17. Case VII. Percutaneous transhepatic cholangiography showed a long area of stenosis of the mid common bile duct (arrows) extending to the junction of the right and left hepatic ducts strongly suggesting tumor.

without evidence of obstruction at the time of the examination (Fig. 22). The differential diagnosis included an edematous ampulla of Vater secondary to an impacted stone associated with pancreatitis versus a tumor of the ampulla of Vater. At surgery a hard triangular mass measuring 1.5×2 cm. was found involving the ampulla of Vater and that part of the duodenal wall contiguous with the lateral edge of the pancreas. The ampulla appeared to be in the center of the tumor. An ulceration of the duodenal surface at the site of the tumor was demonstrated (Fig. 20 and 21). The tumor also involved the paraduodenal lymph nodes. One lymph node along the greater curvature of the duodenum measured 7×3 cm. (open arrow, Fig. 20). Pathologic examination revealed the tumor mass to be a carcinoid of the duodenum involving the ampulla.

Here, as in Case v, the cause of the abnormal percutaneous transhepatic cholangiogram was not clear from the cholangiogram, but rather from the gastrointestinal series. The interpretation of percutaneous cholangiography should always have the benefit of the preceding complete gastrointestinal work-up. If this is not practiced, then duodenal disease as a cause of biliary obstruction will elude the radiologist.

Case x. This 41 year old woman was admitted for treatment of a lower abdominal mass. Four months prior to this she had had 2 episodes of right upper quadrant pain associated with transient jaundice. She was told she had hepatitis. On admission her liver was enlarged. Percutaneous transhepatic cholangiography showed

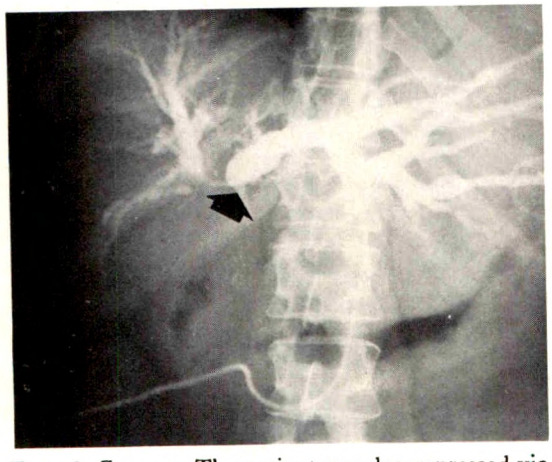

Fig. 18. Case vii. The patient was decompressed via a drainage tube placed in the junction of the right and left hepatic ducts. When this tube was injected with contrast material, it was noted that the dilatation of the left biliary system (arrow) was greater than the right.

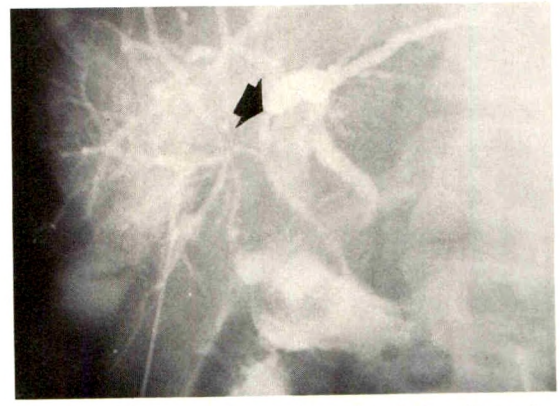

Fig. 19. Case viii. Transhepatic cholangiography showed irregular areas of narrowing throughout the biliary tree with some dilatation of the distal left hepatic duct to a level 1 cm. from the porta hepatis (arrow). The common hepatic duct and common bile duct were also markedly narrowed. Contrast material appeared to flow into the duodenum without significant delay.

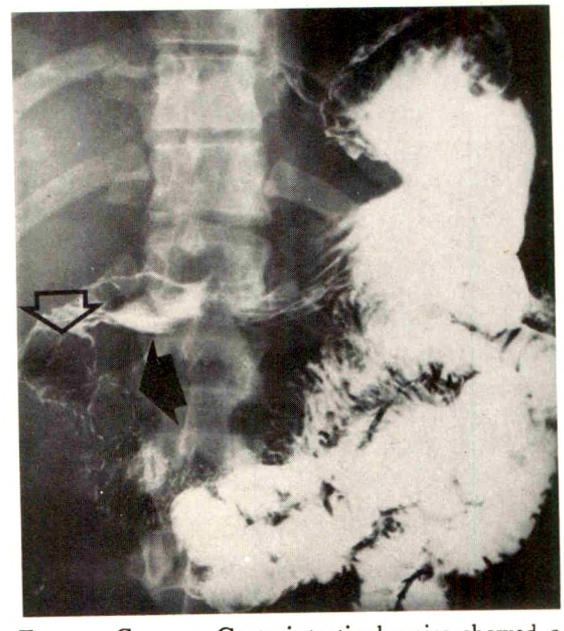

Fig. 20. Case ix. Gastrointestinal series showed a large filling defect (arrow) in the second part of the duodenum in the region of the ampulla with marked mucosal effacement. Extrinsic pressure was noted along the greater curvature of the second part of the duodenum (open arrow).

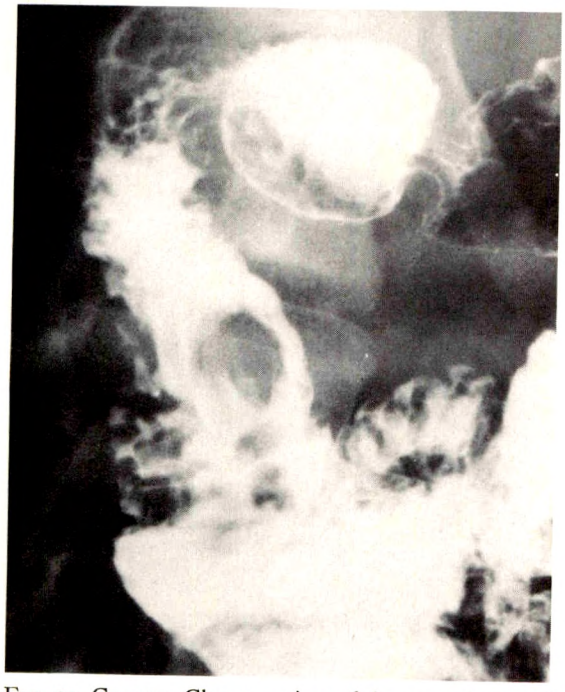

FIG. 21. Case IX. Close-up view of the second part of the duodenum showing the large filling defect with marked mucosal effacement.

partial obstruction of the common hepatic duct by a smooth eccentric defect measuring 1 cm. in length (arrow). The cystic duct and gallbladder were never visualized (Fig. 23). Only small amounts of contrast material entered the duodenum. At operation a well localized tumor mass was obstructing the cystic duct and partially obstructing the common duct. Biopsy of the area showed a granular cell myoblastoma. This is an uncommon tumor which may be encapsulated or, as in most instances, locally infiltrative. Because of the clear localized involvement (arrow, Fig. 23) and the relatively benign nature of the tumor a conservative procedure was performed. This case illustrates the broad range of pathology including relatively benign tumors that can cause partial obstruction of the common duct.

DISCUSSION

Reviewing the questions usually asked of percutaneous transhepatic cholangiography, *i.e.*, is there a mechanical extrahepatic block of the biliary tree, where is the extrahepatic block, and finally, what is causing the block, by far the most effectively answered question is the first.

With a well visualized biliary system following a successful puncture of a duct, the interpretation should be correct. However, if a duct is not successfully entered following several passes of the needle through the liver, what then is the proper interpretation of a failure to obtain visualization? There are those who would say that in experienced hands the inability to puncture a duct probably rules out dilatation of the ducts due to extrahepatic obstruction or at least makes it so unlikely that a conservative approach to the patient is indicated. We feel that although more often than not this approach is correct for the individual situation, it is far too presumptive to be safe. We have experienced inability to puncture markedly dilated ducts in the face of an excellent record of successful puncture of the normal undilated biliary tree. Our position is that failure to visualize the biliary tree by puncture is a technical failure, and no reliable information or assumptions should be drawn from this result. If this policy is adhered to, false diagnosis will not occur.

The first serious problem we have encountered is in answering the second question: where is the extrahepatic block? In

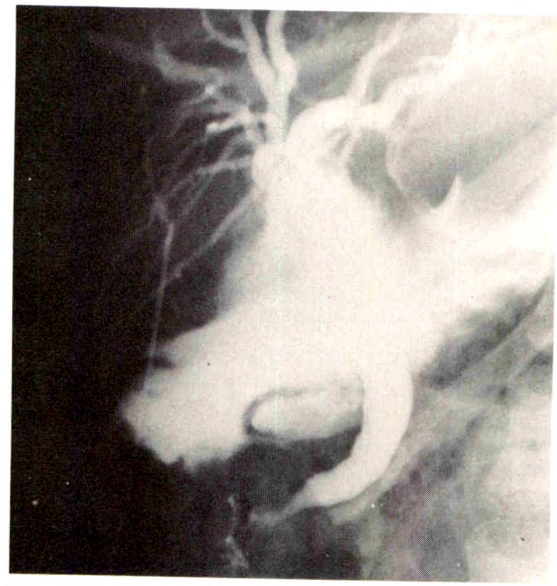

FIG. 22. Case IX. Percutaneous transhepatic cholangiography revealed a dilated collecting system without evidence of obstruction at the time of the examination.

the majority of cases the contrast material will fill the ducts to the most distal point of block and there create a typical defect that will suggest a specific diagnosis. Only in cases of complete block of the biliary tree is there a problem in interpretation of the percutaneous transhepatic cholangiogram. Incompletely blocked ducts show consistently accurate localization of the lesion. Perhaps the diagnostic clue is a hazy interface of contrast material which indicates diffusion of contrast medium through densely inspissated bile. This is illustrated by Cases I, II, and V in which contrast material failed to pass far enough to accurately identify the point of pathology. In Case I a block close to the porta hepatis was suggested. At operation the block in the distal common duct was just proximal to the ampulla of Vater. Dense inspissated bile prevented the contrast material from filtering through to the true point of block. Better and more complete aspiration of the biliary tree is the answer as indicated above.

This leads to the necessity to make certain choices. Some of our surgeons in the past have held that a more peripheral entrance into the biliary system, specifically intrahepatic in position, helps to minimize bile leakage, since liver parenchyma naturally tends to tamponade the potential leakage site. However, aspiration of the com mon bile duct from a small peripheral radicle is difficult. We feel that direct entrance into the central biliary system or main common duct has presented no problem in either the nonobstructed patient who returns to his bed without a laparotomy or the obstructed patient who is immediately operated upon. A more central puncture site allows for a better localization of the block and, therefore, a more reliable percutaneous transhepatic cholangiogram.

Case II again suggested high porta hepatic block. The needle was clearly in the peripheral left hepatic radicles and decompression was limited. A small pancreatic carcinoma involving only the most distal duct was found. In Case V the pathology was identified on a gastrointestinal series

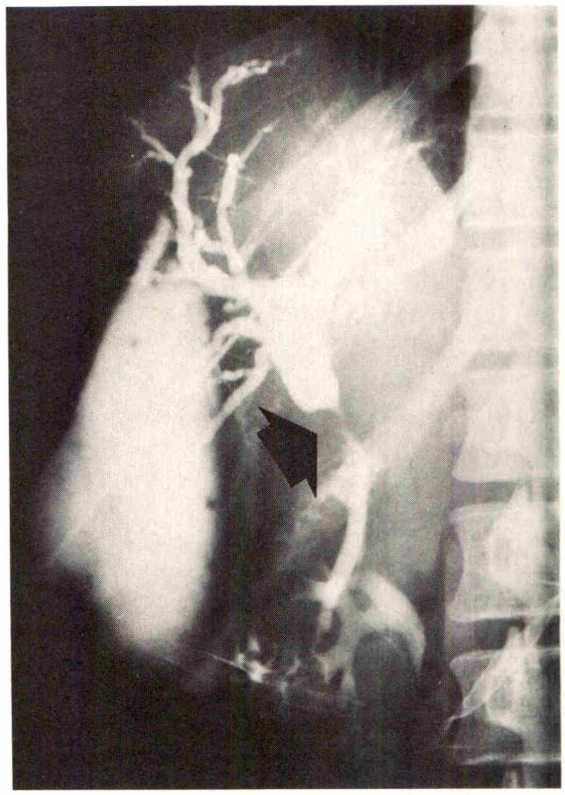

Fig. 23. Case X. Percutaneous transhepatic cholangiography showed partial obstruction of the common hepatic duct by a smooth eccentric defect measuring 1 cm. in length (arrow). The cystic duct and gallbladder were never visualized. Only a small amount of contrast medium entered the duodenum.

as a large lesser curvature ulcer of the duodenal sweep at the level of the ampulla of Vater. The percutaneous transhepatic cholangiogram demonstrated a mid-duct obstruction. At operation local pathology in the duodenal wall and ampulla was the only finding. The need for proper aspiration of the biliary tree is well established by the above 3 cases. Attention paid to a more central needle puncture site will attain better filling and localization.

A second point can be stressed in evaluating the level of block. All appropriate preliminary studies should be performed and be utilized during percutaneous transhepatic cholangiogram interpretation. A complete roentgenologic study of the gastrointestinal tract should be performed prior to transhepatic cholangiography. In Case V the roentgenologic diagnosis of ul-

cerating carcinoma involving the third part of the duodenum, and in Case IX a large filling defect in the second part of the duodenum which was a carcinoid were clearly identified by gastrointestinal series while definite problems arose on transhepatic cholangiography in interpreting the site of block in Case V and even as to whether significant block was present at the time of examination in Case IX. These lesions demonstrated by gastrointestinal series at the ampulla of Vater clearly identify the level of the problem in the biliary tree. In fact, the gastrointestinal series was in both cases the most direct way of establishing the cause of jaundice.

The most difficult problem is deciding the etiology of the block solely from the roentgenologic appearance of the blocked biliary system. As we have seen demonstrated in the above cases, stones should look fairly characteristic, but can create irregular defects suggestive of tumor or stricture secondary to long standing choledocholithiasis. Narrowing of the ducts is a nonspecific finding. Attempts to resolve this by combining percutaneous transhepatic cholangiography with selective visceral angiography have enjoyed considerable success.[1,2,6,8–10] Angiographic demonstration of a well circumscribed mass of tumor vessels at, or just distal to, the site of occlusion or narrowing seen on percutaneous transhepatic cholangiogram tends to establish tumor in that case. Angiography in this type of case may reveal the extent of the tumor growth and liver metastasis if present. However, absence of tumor vessels is less helpful since the many benign diseases mentioned in the above cases and avascular malignant tumors can show strikingly few angiographic findings. Gastrointestinal series may be diagnostic in certain cases by defining pathology of the duodenum or the ampulla of Vater.

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REFERENCES

1. BOIJSEN, E. Selective hepatic angiography in primary and secondary tumors of liver. *Rev. Int. Hepat.*, 1965, *15*, 385–395.
2. BOIJSEN, E., and REUTER, S. R. Combined percutaneous transhepatic cholangiography and angiography in evaluation of obstructive jaundice. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *99*, 153–161.
3. CARTER, R. F., and SAYPOL, G. M. Transabdominal cholangiography. *J.A.M.A.*, 1952, *148*, 253–255.
4. EVANS, J. A., GLENN, F., THORBJARNARSON, B., and MUJAHED, Z. Percutaneous transhepatic cholangiography: discussion of method and report of 25 cases. *Radiology*, 1962, *78*, 362–370.
5. FERRIS, E. J., JOISON, J., SHAPIRO, J. H., and BYRNE, J. J. Percutaneous transhepatic cholangiography: preliminary report describing new approach to examination. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *92*, 1131–1138.
6. GÖTHLIN, J., MOHAMMAD, M., and TRANBERG, K. G. Combined percutaneous transhepatic cholangiography (PTC) and selective visceral angiography (SVA) in obstructive jaundice. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *117*, 419–425.
7. KITTREDGE, R. D., and FINBY, N. Percutaneous transhepatic cholangiography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *101*, 592–604.
8. LUNDERQUIST, A. Angiography in carcinoma of pancreas. *Acta radiol.*, 1965, Suppl. 235.
9. MILLER, H., DEL GUERCIO, L. R. M., COHN, J. D., FEINS, N. R., and COOMARASWAMY, R. P. Transhepatic cholangiography combined with splenoportography in biliary pancreatic carcinoma. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 468–476.
10. REUTER, S. R., REDMAN, H. C., and BOOKSTEIN, J. J. Angiography in carcinoma of biliary tract. *Brit. J. Radiol.*, 1971, *44*, 636–641.
11. WASTE, M. L., and CUNNINGHAM, I. G. E. Roentgenologic findings in recurrent pyogenic cholangitis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *119*, 71–77.
12. WIECHEL, K. L. Percutaneous transhepatic cholangiography. *Acta chir. scandinav.*, 1964, Suppl. 330.
13. YOUNG, W. B. Obstructive jaundice, radiologist, surgeon and patient: Caldwell Lecture, 1971. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *119*, 4–39.
14. ZUHEIR, M., and EVANS, J. A. Percutaneous transhepatic cholangiography. *Radiol. Clin. North America*, 1966, *4*, 535–545.

VOL. 125, NO. 1

GRAY SCALE ULTRASOUND DIAGNOSIS OF OBSTRUCTIVE BILIARY DISEASE*

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ABSTRACT:

B-mode ultrasound examinations have been useful in demonstrating cholelithiasis and obstructive dilatation of the gallbladder. It is now possible with gray scale ultrasound technique to demonstrate dilatation of the common bile duct, as differentiated from the gallbladder, and also to show fluid containing spaces within the liver. Discrimination between solid and fluid filled intrahepatic structures is readily made, and dilated biliary radicles are frequently discernible.

THE clinical applications of B-mode ultrasound examination of the gallbladder, "cholecystosonography," have been recently documented by several investigators.[1–4] Cholelithiasis has been demonstrated in patients with nonvisualization at oral cholecystography, and obstructive dilatation of the gallbladder—the Courvoisier phenomenon—has been identified when hyperbilirubinemia precluded conventional intravenous cholangiography. First Lehman[3] and later Suruga *et al.*[5] discussed the possibility of recognizing biliary duct dilatation by B-mode ultrasound. Recently, using a specially constructed device for liver scanning, Taylor and others[6,7] were successful in demonstrating intrahepatic abnormality. To date these findings have not been demonstrated with commercially available ultrasound equipment capable of total abdominal evaluation. This report describes the appearance of obstructive ectasia of intra- and extrahepatic biliary ducts by gray scale ultrasound technique.

MATERIAL AND METHOD

Studies were performed with an ultrasound unit using a 13 mm., 2.25 mHz barium titanite nonfocused transducer. The wave length at this frequency is 0.75 mm. Contact scanning in the axial plane was performed using a water soluble aqueous gel as a coupling agent. Gray scale images were recorded on a non-storage oscilloscope. In the latter instance, the image was photographed as a time exposure. Scanning technique was an exaggerated sectoring motion in which the probe is maintained tangentially to the abdominal wall and moved in a continuously sweeping arc. This method provides a display of greater dynamic range which is more directly dependent upon the strength of echo return than conventional leading edge storage techniques. Each echo is demonstrated as a brightness modulated spot of variable intensity dependent upon actual reflected amplitude at varying tissue interfaces. Approximately 5 intermediate shades of gray can be recognized by this method.

REPORT OF CASES

CASE 1. A 74 year old female entered the hospital with a chief complaint of abdominal pain. Thirteen years prior to admission cholecystectomy and common duct exploration were performed for cholecystitis and cholelithiasis. In recent months episodes of increasingly severe pain occurred at frequent intervals accompanied by jaundice and dark urine. Physical examination on admission was not revealing. Laboratory studies disclosed an alkaline phosphatase level of 13.7 Bodansky units (normal 2.0–4.5 Bodansky units). The bilirubin was normal. Ultrasound examination was per-

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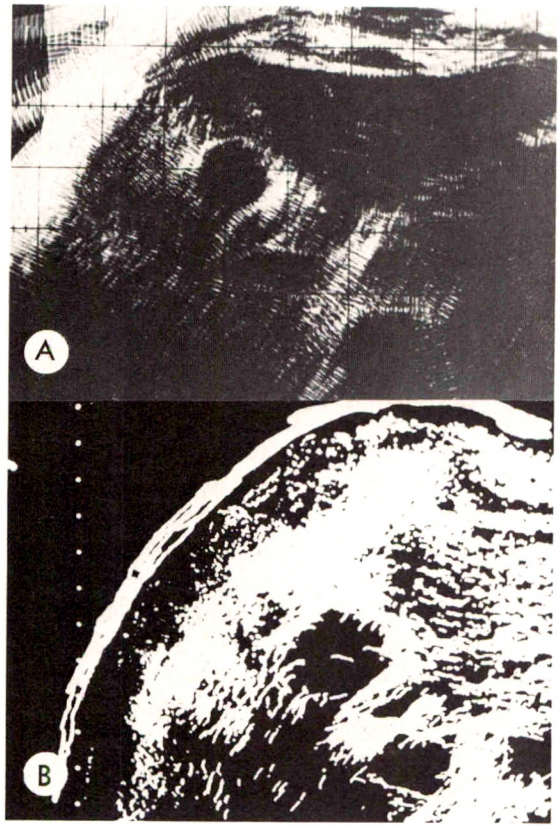

FIG. 1. Case 1. (*A*) Transverse gray scale section at level of porta hepatis disclosing a round echo-free space representing the dilated common duct. (*B*) High gain leading edge section 3 cm. below (*A*) revealing a single linear echo within the dilated duct.

formed to exclude associated pancreatic abnormality. This revealed a dilated common duct containing a large calculus (Fig. 1, *A* and *B*). Intravenous cholangiography disclosed a 1 cm. common duct stone (Fig. 2). At exploration the calculus was removed and a choledochoduodenostomy performed.

CASE II. A 70 year old male entered the hospital with a chief complaint of severe low back pain. Approximately 1 year prior to admission an operation was performed at another hospital for jaundice at which time an apparently obstructed common bile duct was found and bypassed. A poorly defined mass was palpated during that procedure but a biopsy was not obtained. The patient did well following hospitalization until 3 months prior to this admission when the onset of back pain became severe. Jaundice recurred, and intermittent temperature elevation was noted. Physical examination disclosed a grossly jaundiced, chronically ill man in no acute distress. Abdominal examination revealed a tender, enlarged liver without other abnormality. The direct bilirubin was 17 mg. per cent with an alkaline phosphatase level of 15.3 Bodansky units and an amylase level of 2.0 units. The white blood cell count was 26,000 with 95 per cent polys and 5 per cent band forms. Blood cultures were positive on 3 occasions for *E. coli* species. An abdominal ultrasound examination indicated the presence of a large irregular solid mass in the head of the pancreas. In addition, well-defined small ductal structures were noted to occupy a significant portion of the right lobe of the liver (Fig. 3). A diagnosis of obstructive jaundice and cholangitis secondary to carcinoma of the pancreas was made. Treatment with appropriate antibiotics was initiated, but, prior to anticipated bypass surgery, the patient expired.

At necropsy a large carcinoma of the head of the pancreas causing common duct obstruction was found. The liver was enlarged and con-

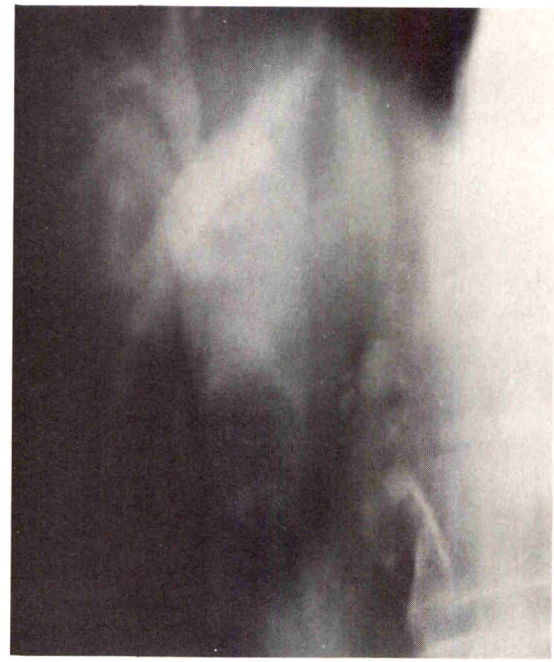

FIG. 2. Case 1. Intravenous cholangiogram. Tomographic section demonstrating marked common duct dilatation with a single large radiolucent calculus.

tained grossly ectatic biliary radicles measuring 0.5–0.1 cm. in diameter.

DISCUSSION

Ultrasound examination in Case 1 disclosed a distinct 3.5 cm. cystic structure with sharply defined margins located within the hepatic parenchyma. The round transonic space having the appearance of a dilated duct in cross-section appears more laterally placed than the usually observed gallbladder echo.

Differentiation of anatomic structures within the porta hepatis (*e.g.*, portal vein, common bile duct, hepatic artery) may be difficult to elucidate inasmuch as they are in close relation. This is complicated by the fact that the gallbladder is a highly variable organ both in location and morphology. Certain features, however, aid in discrimination and warrant further comment. The echotomographic appearance of the gallbladder is that of a more medially placed structure having an oblique axis towards the midline in the sagittal plane. The dilated ductus choledochus noted above does not exhibit this orientation and in fact follows an oblique axis away from the midline in the sagittal plane. In addition, this structure does not appear to manifest an interface with the inferior margin of the liver. This latter factor has not been previously stressed, but has been of use in identification. Figure 4 is an axial section of the typical appearance of the gallbladder utilizing the gray scale technique. Here it is noted as a cystic oval mass "suspended" from the inferior margin of the liver in which the usual hepatic internal echoes are noted. The appearance of both liver and gallbladder are accentuated in this instance by the presence of ascitic fluid. Differentiation from primary intrahepatic fluid with abnormalities such as hematoma or abscess can be made on the basis of the lack of adjacent hepatic parenchymal compression echoes and the presence of a very smooth, well-defined, regular margin of the duct itself.

Fig. 3. Case II. Gray scale transverse section of the upper abdomen revealing an irregular solid pancreatic mass and multiple oval transonic spaces within the liver indicative of dilated bile ducts.

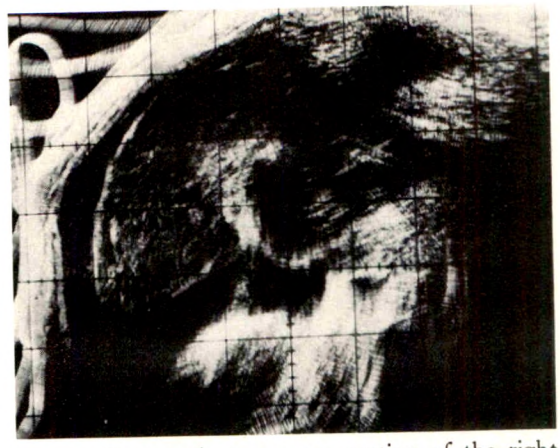

Fig. 4. Gray scale transverse section of the right upper abdominal quadrant revealing an elliptical echo-free space representing an enlarged gallbladder. The transonic crescentic space separating liver from abdominal wall is produced by ascitic fluid.

Case II is an illustration of the appearance of dilated intrahepatic biliary ducts. This clearly indicates that the degree of intrahepatic biliary ectasia does not have to be as extensive as noted in the prior example of a dilated common duct. As seen in Figure 3, multiple small intrahepatic ducts were defined which ranged in size from 0.75 to 1.0 cm. This approached the limit of resolution obtainable with the 2.25 mHz crystal used at that time. The image readily

discriminates between the low amplitude gray echoes which are produced by the solid lesion of the pancreas and the rather bright, higher amplitude echoes at the duct interfaces. This difference is the result of a greater change in acoustic impedance which occurs between fluid-soft tissue boundaries.

The above examples indicate that fluid containing intrahepatic pathology can be clearly resolved by gray scale ultrasound technique. In part, this is due to the fact that a gray scale liver scan can generally be completed within one pass of the transducer. This in itself prevents the considerable degradation of resolution of the hepatic image caused by multiple scan paths and respiratory variation which accompany the production of a leading edge ultrasound scan. The greater range of decibel information produced by this method directs more visual information to the observer. This usually allows discrimination of fluid from solid lesions without sequential gain changes. In addition, the quality of the image is improved by the intrinsically smaller spot size utilized by the non-storage oscilloscope. These factors can be readily appreciated by comparison of the storage oscilloscope image seen in Figure 1*B* with any of the gray scale examinations reproduced in this communication.

Although reported experience of gray scale hepatic scanning is at the moment limited, these early studies seem to indicate that it is not an unusual circumstance to observe ectatic biliary radicles in the presence of obstructive jaundice. In part, this is a manifestation of the substantial intrahepatic mass effect which may be produced by the decompensated intrahepatic biliary tree as demonstrated by other less specific imaging techniques such as technetium liver scanning. Recognition that this space occupying lesion can be resolved in patients with obstructive disease should warrant more frequent search for its presence; as frequently, an etiologic diagnosis can also be rendered by this non-invasive modality.

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REFERENCES

1. DOUST, B. D., and MAKLAD, N. F. Ultrasonic B-mode examination of gallbladder. *Radiology*, 1974, *110*, 643–647.
2. HUBELITZ, U. F., KAHN, P. C., and SELL, L. A. Cholecystosonography: approach to non-visualized gallbladder. *Radiology*, 1972, *103*, 645–646.
3. LEHMAN, J. Ultrasound in diagnosis of hepatobiliary disease. *Radiol. Clin. North America*, 1966, *4*, 605–623.
4. LEOPOLD, G., and SOKOLOFF, J. Ultrasonic scanning in diagnosis of biliary disease. *Surg. Clin. North America*, 1973, *53*, 1043–1052.
5. SURUGA, K., NAGUSHIMA, K., HARAI, Y., WAGAI, T., and INUI, M. Clinical and pathological study of congenital biliary atresia. *J. Pediat. Surg.*, 1967, *2*, 558–564.
6. TAYLOR, K. J. W., CARPENTER, D. A., and McCREADY, V. R. Grey scale echography in diagnosis of intrahepatic disease. *J. Clin. Ultrasound*, 1973, *1*, 284–287.
7. TAYLOR, K. J. W., CARPENTER, D. A., and McCREADY, V. R. Ultrasound and scintigraphy in differential diagnosis of obstructive jaundice. *J. Clin. Ultrasound*, 1974, *2*, 105–116.

Vol. 125, No. 1

HEPATIC ECHOGRAPHY*

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ABSTRACT:

There are 2 kinds of echographic investigations: those which are only complementary and those which are specific by themselves. To improve this latter category, we would like to emphasize two useful echographic signs. First, the attenuation sign, which indicates the fibrotic nature, therefore practically speaking, the cirrhotic nature of a diffuse echogenic pattern. Secondly, the posterior echo accumulation sign, which indicates the liquid nature, and therefore in general the cystic nature of a localized sonolucent pattern.

SINCE the first reports of Holmes,[10] Lehman,[13] and Freimanis *et al.*,[5] the abdominal echographic investigation has been gradually accepted as a valuable diagnostic method. However, there are relatively few publications pertaining to upper abdominal lesions. We have found that the echographic investigation of the liver is usually a complementary study, but that it may sometimes be very specific and quite diagnostic by itself.

METHOD

The examinations are performed with a 2 mHz transducer, embedded in a contact scanner. The patient needs no special preparation, and lies in a supine position. His abdomen is covered with an oily material to assure proper sonic transmission. The views are recorded while the patient holds his breath in deep inspiration, to avoid superimposition of ribs, as much as possible. Sometimes, subcostal views with a cephalad angled probe may be necessary.

THE NORMAL LIVER

The normal liver is an echo-free structure, at low to moderate sensitivity levels, and it becomes echogenic only at high sensitivity levels. In other words, a normal liver is sonolucent at usual sensitivity levels (Fig. 1). This anechoic character comes from the fact that the normal liver parenchyma is homogeneous, so it harbors almost no interfaces reflecting echoes. The only exceptions, at usual sensitivity levels, to this anechoic character come from the capsule and the hilus, which structures constitute highly reflecting interfaces.

THE PATHOLOGIC LIVER

Cirrhosis. The outstanding characteristic of a cirrhotic liver is its great echogenicity, even at low sensitivity levels (Fig. 2). This occurs because such a partially necrotic and partially regenerating liver is no longer homogeneous but contains many interfaces for reflection.

A further characteristic of a cirrhotic liver is the miliary phenomenon (Fig. 2),

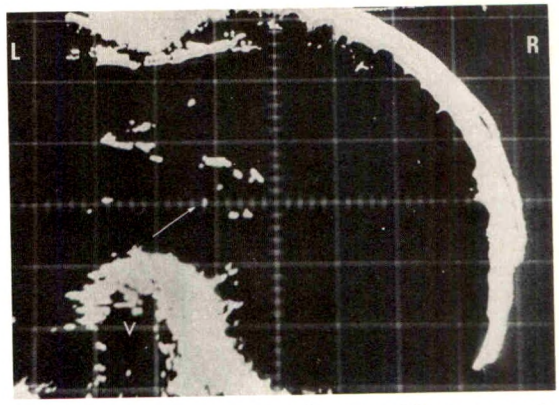

FIG. 1. Transverse section of a normal liver, at moderate sensitivity. The liver is seen as an anechoic area. The only exceptions are the capsule at the periphery and the hilus (arrow) centrally, in front of the vertebral column (V).

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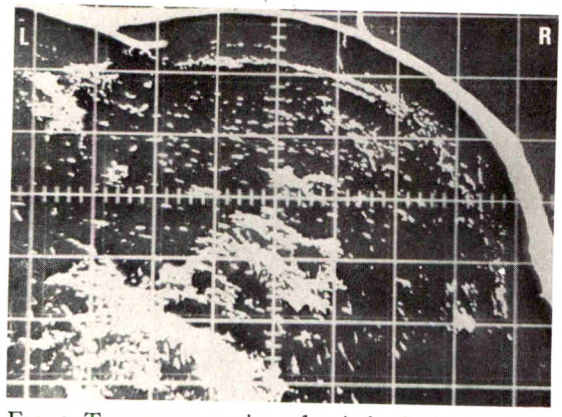


FIG. 2. Transverse section of a cirrhotic liver, at low sensitivity. The liver is now seen as an echogenic area, and the echoes are diffusely scattered.

where the echoes recorded are fine and diffusely scattered, without any tendency for particular grouping. The explanation of this miliary phenomenon is simply the generalized aspect of the cirrhotic process.

The cirrhotic liver may display a third sign, the ascitic sign. This is seen as an absolutely sonolucent area anteroinferior to the liver (Fig. 3), even at high sensitivity, representing fluid which has no interfaces to reflect the ultrasound beam. Such a sonolucent ascitic area must be distinguished from the sonolucent band very often seen coming from the subcutaneous fat. The distinction is easily made by considering these 3 criteria: (1) The ascitic band is absolutely sonolucent, while the subcutaneous fat band becomes echogenic at high sensitivity (Fig. 4, *A* and *B*). (2) In the dorsal recumbent position, the ascitic band widens inferiorly, while the subcutaneous fat band maintains its width (Fig. 4, *A* and *B*). (3) The width of an ascitic band will vary according to the patient's position, while the width of a subcutaneous fat band will not change (Fig. 5, *A* and *B*).

Finally, there is another possible sign with cirrhosis, the attenuation sign. This sign, when present, means that the echoes are found mostly in the superficial parts of the liver, while the deepest portions seem to be less echogenic (Fig. 6, *A* and *B*). This has a double explanation. First, the great heterogeneity of cirrhotic livers, by creating

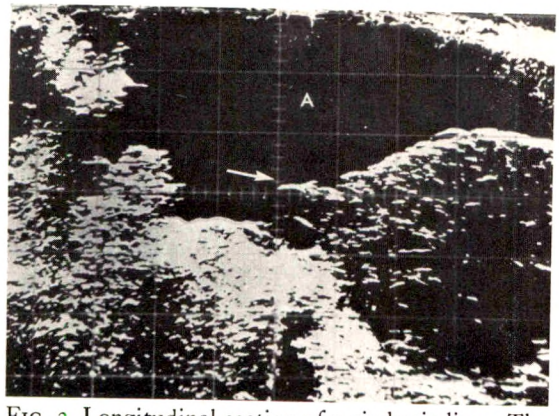


FIG. 3. Longitudinal section of a cirrhotic liver. The absolutely sonolucent area (A) anteroinferior to the liver tip (arrow) represents the ascites (caudal is left).

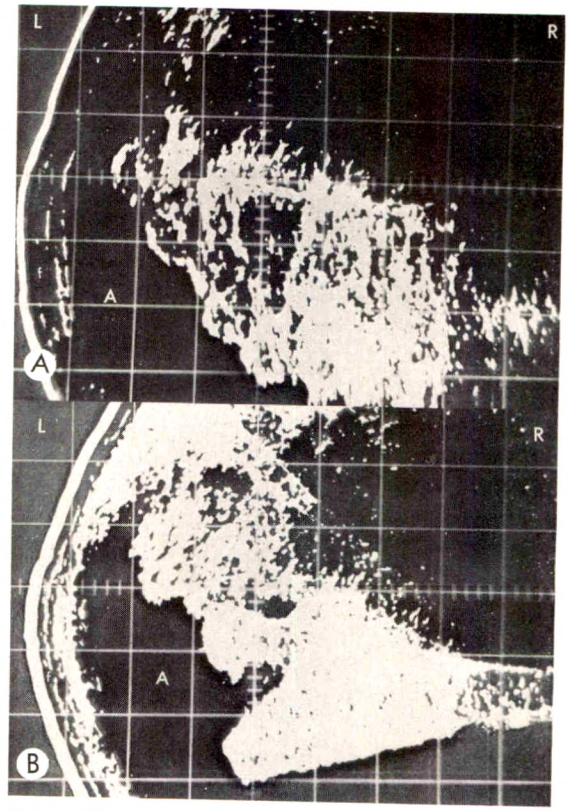


FIG. 4. Transverse sections of an ascitic abdomen, in dorsal recumbent position, at low (*A*) and high (*B*) sensitivity, displaying both the subcutaneous fat band (f) and an ascitic area (A). In contrast to the subcutaneous fat band, the ascitic area widens inferiorly and resists the gain increase.

many reflecting interfaces, prevents good transmission of the beam toward the deepest portions. Secondly, the fibrosis of advanced cirrhosis greatly absorbs the beam and also prevents good transmission of the beam toward the deepest portions. Thus, the attenuation sign, being partially due to fibrosis, is a sign of severity, and when present means that we are dealing with an advanced stage of cirrhosis.

Myeloid Metaplasia. The echographic appearance of this infrequent liver disease resembles mild cirrhosis with no fibrosis. Thus, the echographic signs are a great echogenicity and a miliary phenomenon, but a negative attenuation sign (Fig. 7). The great echogenicity again comes from the heterogeneity of the liver, where hematopoietic foci give many reflecting interfaces, seen even at low sensitivity. The miliary phenomenon is again due to the generalized aspect of the process. Finally, the negative attenuation sign means that the deepest parts are easily explored by the ultrasound beam, even at low sensitivity. This is mainly explained by the low density of such a nonfibrosing process, therefore less absorbing for the ultrasound energy which easily reaches and discloses the echogenicity of the deepest portions.

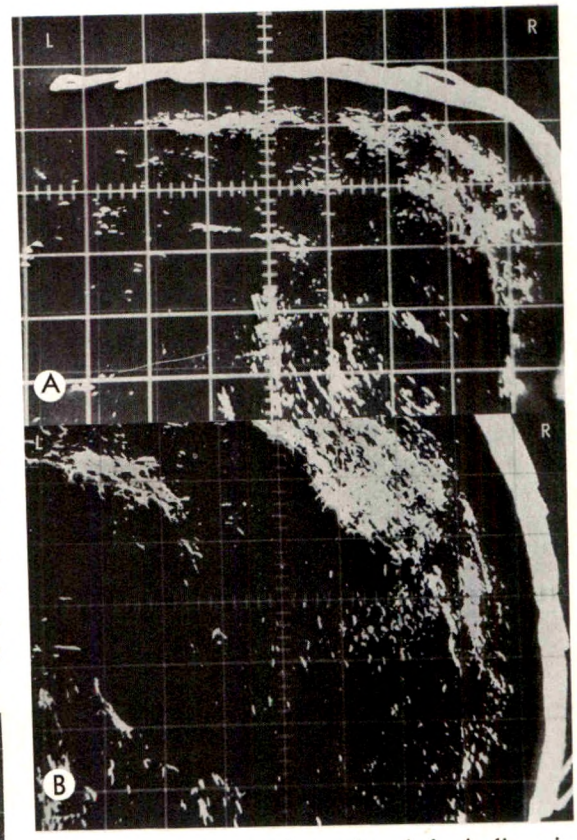


Fig. 6. Transverse sections of a cirrhotic liver in dorsal recumbent (*A*) and right posterior oblique recumbent (*B*) positions. The sonolucent band at the periphery represents subcutaneous fat: it does not widen inferiorly and does not shift. The liver itself displays a positive attenuation sign: the deepest portions seem to be less echogenic than the superficial.

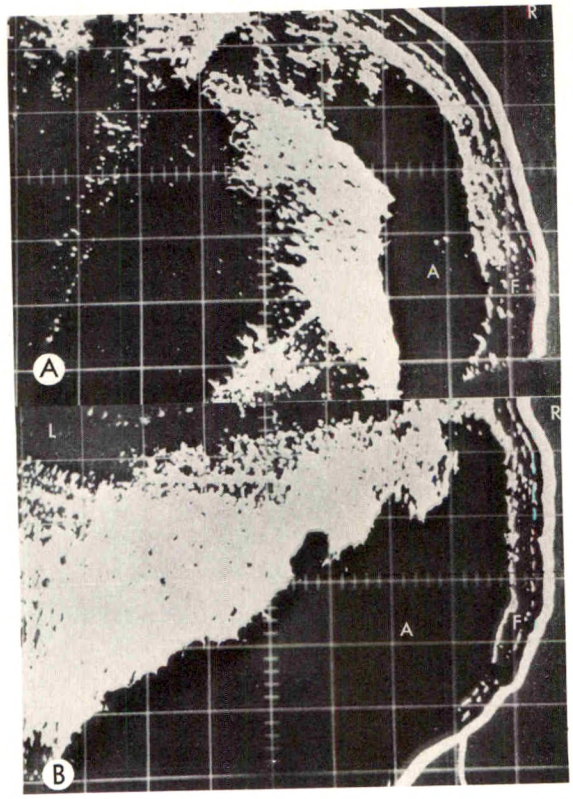


Fig. 5. Transverse sections of an ascitic abdomen in left posterior oblique recumbent (*A*) and right posterior oblique recumbent (*B*) positions. These sections display that ascites (A), in contrast to the subcutaneous fat band (F), is mobile.

Liver Carcinomatosis. In liver carcinomatosis, there are two kinds of echographic patterns: the miliary and the nodular. The first consists of many diffusely scattered echoes and corresponds to an extensive involvement (Fig. 8). Unfortunately, this pattern is not specific and resembles the

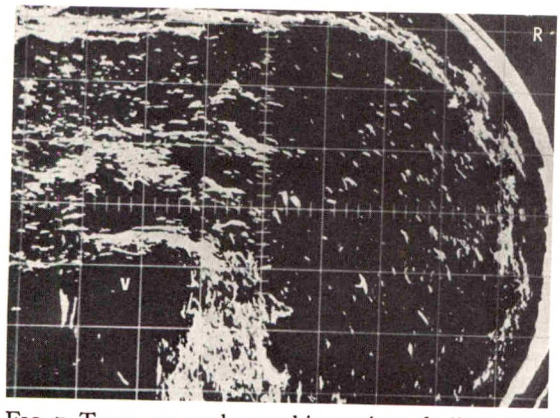


FIG. 7. Transverse echographic section of a liver with myeloid metaplasia. The pattern is echogenic and miliary. The attenuation sign is negative: the echoes are found equally in the superficial and in the deep portions of the liver (V: vertebra).

one we have seen for cirrhosis and myeloid metaplasia.

The second pattern for carcinomatosis is nodular where the echoes are no longer diffusely scattered, but show a tendency for particular groupings. Two kinds of nodule can be seen. The first is echogenic, seen as a localized accumulation of echoes (Fig. 9, *A* and *B*). This pattern is obtained from heterogeneous metastasis which, being necrotic, contains many interfaces reflecting the ultrasounds.

The second kind of metastatic nodule is sonolucent and appears as a ringlike structure. This ringlike pattern is produced by metastasis, the texture of which is rather homogeneous, so that there are almost no interfaces reflecting the ultrasound in echoes (Fig. 10). Such a ringlike pattern for metastasis must be differentiated from simple cystic disease. This is done by increasing the gain setting. When the gain is increased, the ringlike patterns produced by metastasis gradually fill in with echoes, while fluid-filled cysts, which are homogeneous, resist a gain increase. This is shown in Figure 11.

Hepatoma. The echographic appearance of this disease is shown in Figure 12.

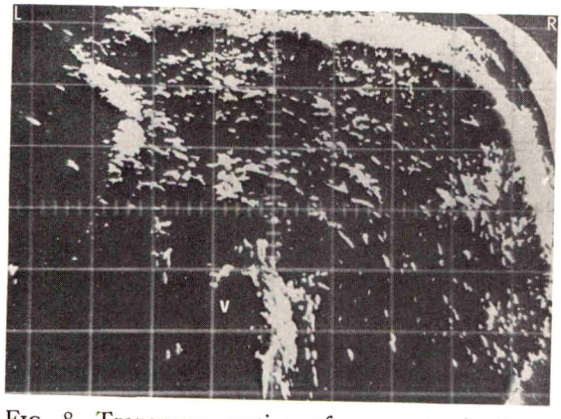


FIG. 8. Transverse section of a metastatic liver, showing the miliary pattern of carcinomatosis: the pattern is echogenic, and the echoes are diffusely scattered (V: vertebra).

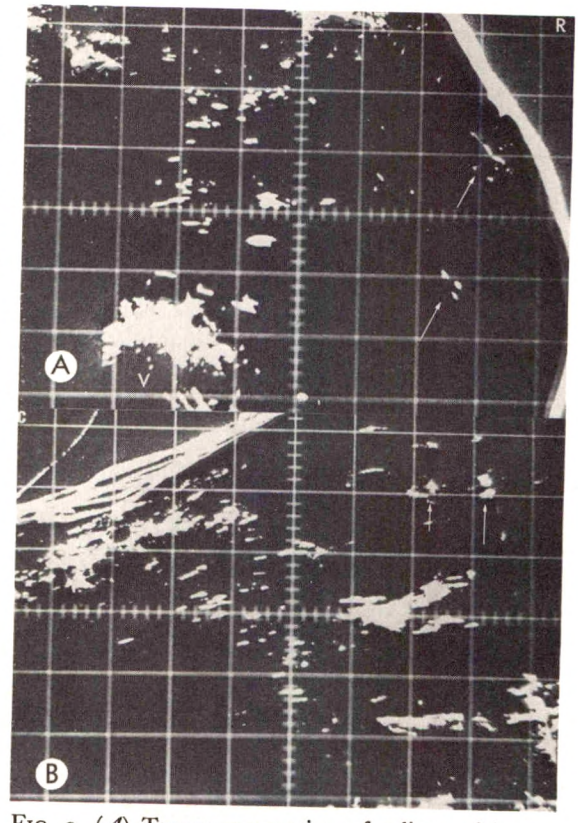


FIG. 9. (*A*) Transverse section of a liver with metastases showing the echogenic nodular pattern of carcinomatosis: the lesions (arrows) are spontaneously seen, at low sensitivity, as localized accumulations of echoes (V: vertebra). (*B*) Longitudinal section showing the echogenic nodular pattern of carcinomatosis: the lesions (arrows) are spontaneously seen again as localized accumulations of echoes (C: caudal end).

Hepatoma presents as an echogenic mass with ill-defined contours. The echogenicity is due to its necrotic and hemorrhagic characteristics producing many reflecting interfaces, while the ill-defined contours are a manifestation of the invading aspect of such a pathology.

Multicystic Liver. The echographic picture of any cystic process rests on 3 signs. The first is a great sonolucency. Cysts are anechoic structures, even when the gain setting is increased (Fig. 13). This sonolucency comes from the fact that the liquid content gives to cysts a homogeneous, interface-free, internal structure.

The second echographic sign is a sharp boundary. Cysts have typical, precise contours which come from the great impedance difference between their walls and the surrounding tissues on the one hand and their fluid content on the other hand (Fig. 13). Finally, cysts classically display a positive posterior echo accumulation sign. The latter is explained by the fact that liquids, as opposed to solids, do not attenuate the ultrasound beam. Thus the beam is strongly reflected by the interfaces beyond the cyst (Fig. 13 and 14).

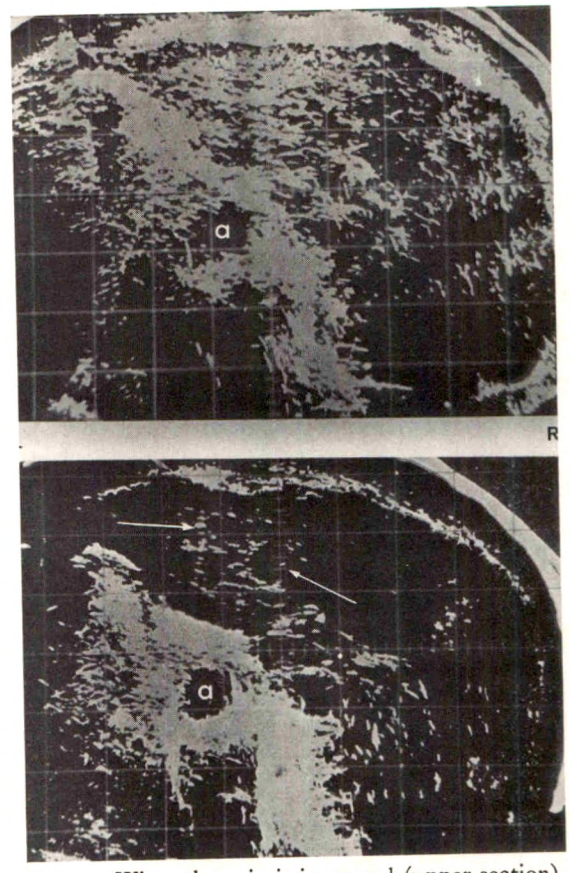


Fig. 11. When the gain is increased (upper section), the ringlike patterns coming from metastasis do not keep their sonolucency, while the ringlike pattern coming from the aorta (a), a fluid filled structure, resists and keeps its sonolucency.

DIFFERENTIAL ECHOGRAPHIC APPROACH

By utilizing all the signs described previously, it is possible to build a synthetic differential echographic approach to the liver pathology. For this approach, we simply divide the echographic patterns into diffuse or localized, and each of these patterns may be echogenic or sonolucent. This creates 4 categories.

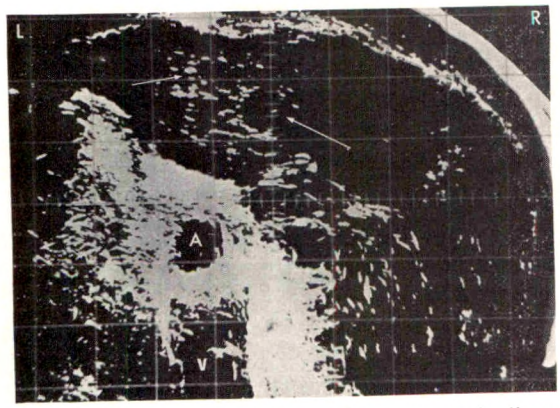


Fig. 10. Transverse section of a metastatic liver showing the sonolucent nodular pattern of carcinomatosis: the lesions appear as ringlike areas (arrows) particularly in the pre-aortic area (A: aorta, V: vertebra).

Diffuse Echogenic Presentation. In this first category (Table 1), the type of distribution is important. If it is nodular, it means that the diffuse heterogeneous involvement we are dealing with is coarse, and in these circumstances, carcinomatosis must be considered. On the other hand, if the diffuse heterogeneous involvement is of the miliary type, we are left with many possibilities including cirrhosis, extensive

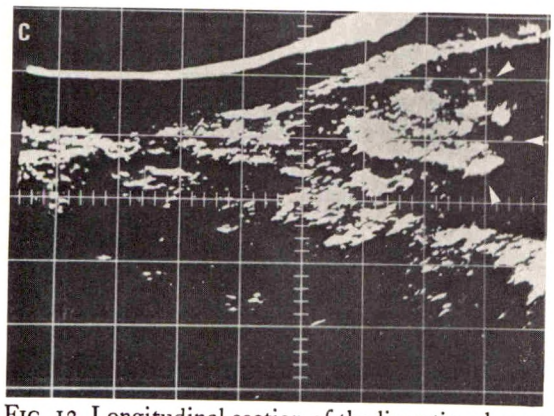


FIG. 12. Longitudinal section of the liver tip where a hepatoma is seen as an echogenic mass (arrowheads) with ill-defined contours (C: caudal end).

carcinomatosis, hepatitis, storage diseases, and myeloid metaplasia. But these numerous possibilities can be divided into 2 groups by the useful attenuation sign, which, when positive, indicates fibrosis, and circumscribes the diagnosis strongly in favor of moderate to advanced cirrhosis.

Diffuse Initially Sonolucent (ringlike) Presentation. In this second category (Table I), we consider whether the ringlike structures we have represent homogeneous or only pseudohomogeneous lesions. This is done by increasing the gain. If the ringlike patterns do not persist, the lesions are only pseudohomogeneous, and carcinomatosis is to be considered. On the other hand, if the ringlike patterns resist a gain increase we have true homogeneous lesions, and a multicystic liver disease is present.

Localized Echogenic Presentation. In this third category (Table II), we are dealing with a single heterogeneous process. Such a presentation is, in most instances, a malignant one, and represents either a hepatoma or a single necrotic metastasis.

Localized Initially Sonolucent (ringlike) Presentation. In this last category (Table II), we determine if the initial sonolucency resists a gain increase. If it does not, then the localized lesion is a pseudohomogeneous one, and we consider either an abscess with necrotic fragments or a single metastasis. On the other hand, if the initial sonolucency remains despite a gain increase, we can conclude that the lesion is truly homo-

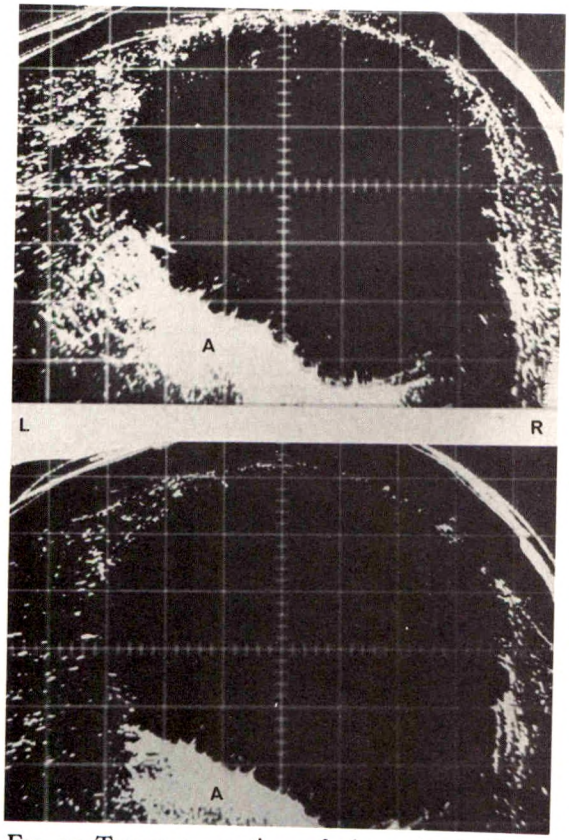


FIG. 13. Transverse sections of a huge simple biliary cyst. The sonolucency resists the gain increase (upper section). Its contours are sharp. There is a positive posterior echo accumulation sign (A).

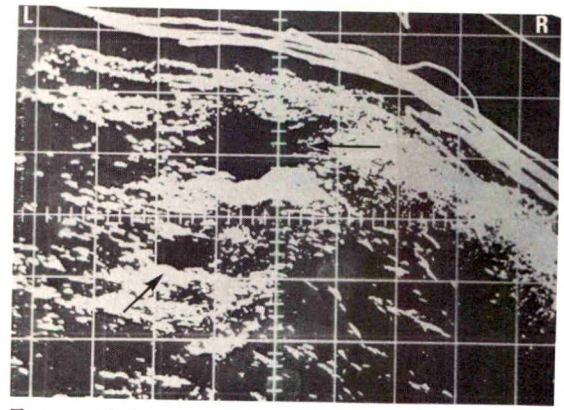
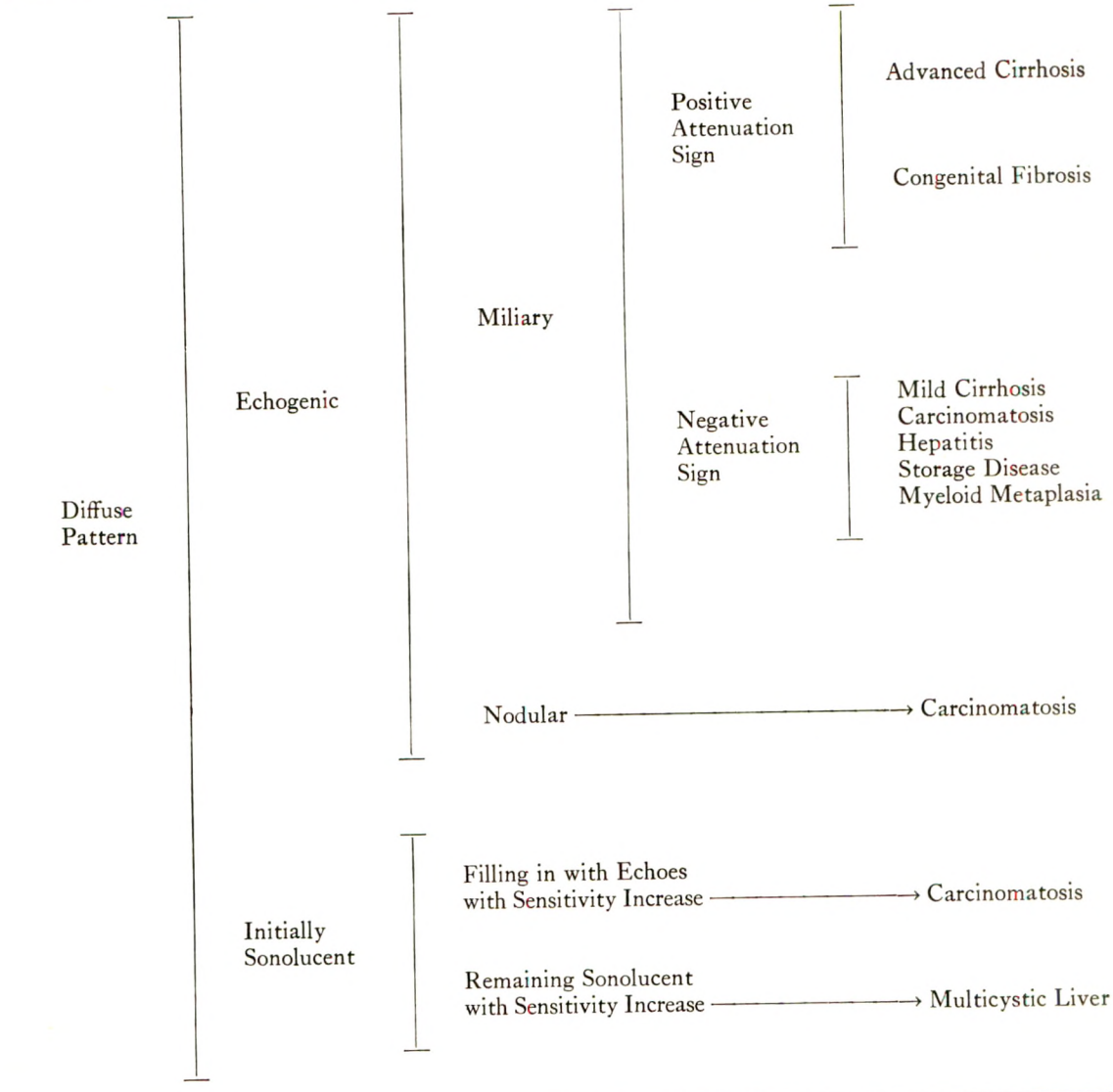


FIG. 14. Subcostal oblique section of a multicystic liver, at high sensitivity, illustrating 2 cysts (arrows) appearing as absolutely sonolucent areas, with echoes accumulating posteriorly.

TABLE I

DIFFUSE PATTERNS

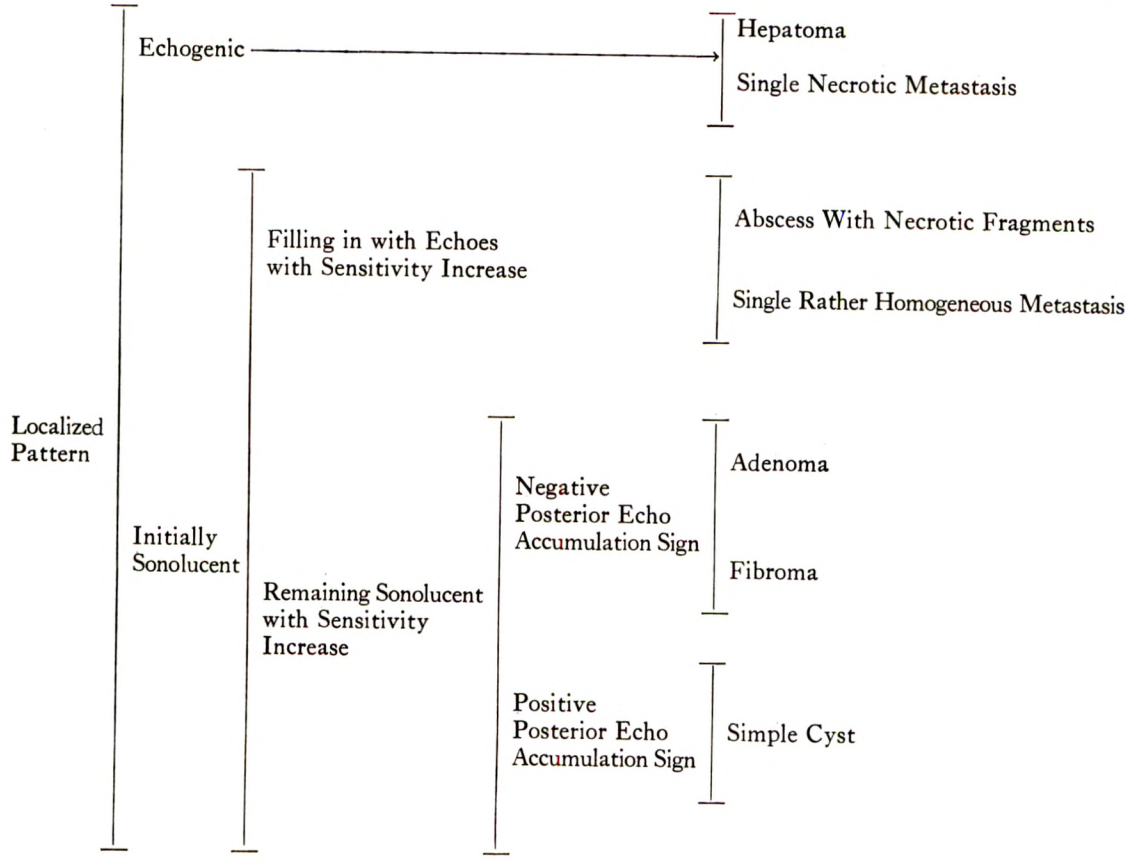


geneous, and is, for practical purposes, liquid. But a further test is available, and we use the posterior echo accumulation sign to discriminate between a liquid lesion and a homogeneous solid lesion.

If this last sign is negative, that is, if there is no echo accumulation posteriorly, it means that the very homogeneous lesion we are dealing with is also an absorbing one for the beam, and thus a solid one. The diagnosis to propose in this instance is a homogeneous and solid lesion such as an adenoma or a fibroma. On the other hand, if the posterior echo accumulation sign is positive, that is, if there is echo accumulation posteriorly, it means that the homogeneous lesion we are dealing with is also non-absorbing for the beam, and thus liquid.

TABLE II

LOCALIZED PATTERNS



The diagnosis is then a fluid filled cystic lesion.

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REFERENCES

1. BECKER, J. C. Ultrasonic symptomatology of liver diseases. *J. de radiol., d'électrol. et de méd. nucléaire*, 1972, *53*, 741–742.
2. CHARBONNIER, A. Differential diagnosis of solid liquid tumors of liver by ultrasonic B echotomography. *Arch. Fr. Mal. App. Dig.*, 1971, *60*, 151–152.
3. COHEN, W. N. B-scan ultrasonography of abdominal mass lesions. *Radiology*, 1969, *93*, 589–591.
4. DAMASCELLI, B. Two-dimensional ultrasound in liver diseases. *J.A.M.A.*, 1968, *204*, 963–968.
5. FREIMANIS, A. K., and ASHER, W. M. Development of diagnostic criteria in echographic study of abdominal lesions. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *108*, 747–755.
6. GOLDBERG, B. B. Evaluation of ascites by ultrasound. *Radiology*, 1970, *96*, 15–22.
7. GOTTLIEB, S. Quantitation of hepatic abscess by A-mode ultrasound: report of case. *Ann. Surg.*, 1972, *38*, 292–294.
8. GROS, C. Echography and liver pathology. *J. de radiol., d'électrol. et de méd. nucléaire*, 1972, *53*, 740–741.
9. HOLM, H. H. Ultrasonic scanning in diagnosis of space-occupying lesions of upper abdomen. *Brit. J. Radiol.*, 1971, *44*, 24–36.
10. HOLMES, J. H. Ultrasonic diagnosis of liver diseases. *Diagnostic Ultrasound*, 1965, 249–263.
11. HOWRY, D. H. Brief atlas of diagnostic ultrasonic radiologic results. *Radiol. Clin. North America*, 1965, *3*, 433–452.
12. IGAWA, K. I. Use of scintillation and ultrasonic

scanning to disclose polycystic kidneys and liver. *J. Urol.*, 1972, *108*, 685–688.

13. Lehman, J. S. Ultrasound in diagnosis of hepatobiliary disease. *Radiol. Clin. North America*, 1966, *4*, 605–623.
14. Mathews, A. W. Use of combined ultrasonic and isotope scanning in diagnosis of amoebic liver disease. *Gut*, 1973, *14*, 50–53.
15. McCarthy, C. F. Comparison of ultrasonic and isotope scanning in diagnosis of liver disease. *Brit. J. Radiol.*, 1970, *43*, 100–109.
16. McCarthy, C. F. Use of ultrasound in diagnosis of cystic lesions of liver and upper abdomen and in detection of ascites. *Gut*, 1969, *10*, 904–912.
17. Melki, G. Hepatic and pancreatic investigation using contact echotomography. *Ann. Radiol.*, 1972, *15*, 567–579.
18. Monroe, L. S. Ultrasonic scan in management of amebic hepatic abscess. *Am. J. Digest. Dis.*, 1971, *16*, 523–528.
19. Mountford, R. A. Quantitative analysis of ultrasonic liver scan. *Gut*, 1971, *12*, 862–863.
20. Rasmussen, S. N. Liver volume determination by ultrasonic scanning. *Brit. J. Radiol.*, 1972, *45*, 579–585.
21. Weill, F. Tomoechographic exploration of liver. *J. de radiol., d'électrol. et de méd. nucléaire*, 1971, *52*, 221.
22. Weill, F. Tomoechographic hepato-biliary elementary semiology. *Ann. Radiol.*, 1970, *13*, 567–578.,
23. Wells, P. N. Comparison of A-scan and compound B-scan ultrasonography in diagnosis of liver disease. *Brit. J. Radiol.*, 1969, *42*, 818–823.

EVALUATION OF PELVIC MASSES DURING INFUSION EXCRETORY UROGRAPHY*

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ABSTRACT:

Infusion excretory urography not only clearly delineates the urinary tract structures, but provides additional information about the type of pelvic mass present. Benign uterine fibroids show contrast enhancement in a significant number of cases, whereas endometrial and cervical malignancies give no specific pattern. Cystic and mixed ovarian neoplasms show rim opacification in over 50 per cent of cases, and the presence of this rim sign points strongly to an ovarian lesion. The opacification of solid ovarian tumors cannot be differentiated from that of uterine fibroids unless other identifying features such as specific calcification or visualization of a normal uterus are present. Pelvic inflammatory disease shows no specific type of opacification. Excretory urography is part of the preoperative evaluation in most patients with pelvic mass lesions. By utilizing the infusion method, high quality excretory urograms are obtained, and, in addition, helpful information is obtained regarding the type of pelvic mass present.

DURING the past 18 months at this institution, infusion urography has been performed routinely in all nonhypertensive adult excretory urograms. Definite opacification of both normal pelvic structures and pelvic masses was noted, prompting the review of all available infusion urograms in females with surgically and pathologically documented pelvic masses. Uterine fibroids gave a rather typical solid to mottled opacification. Cystic ovarian tumors showed an opacified rim following infusion of contrast medium, whereas solid ovarian lesions often showed a mottled opacification.

MATERIAL AND METHOD

For the past 18 months at this institution, infusion excretory urography has been the routine mode of adult urinary tract examination. All infusion urograms are monitored and individualized according to the problems encountered, although a minimum routine filming sequence is followed in all patients.

Following preliminary roentgenograms of the abdomen, a dose of 300 cc. of commercially prepared 30 per cent Methylglucamine diatrizoate is infused *via* a 19 gauge needle. A film localized to the kidneys is obtained at 5 minutes, and a roentgenogram of the entire abdomen is obtained at 10 minutes after the beginning of infusion (usually the time required to complete the infusion). From that point on, we tailor all studies, obtaining oblique, upright, coned and tomographic films as indicated. A final prone roentgenogram is routine at the completion of the examination.

Records and roentgenograms of 126 patients with proved pelvic masses were reviewed. Of these patients, 80 had sufficient preoperative information available to qualify for this study. Patients suspected of renovascular hypertension were not included in this study. All infusion urograms were reviewed regarding: (1) the roentgenographic presence or absence of a pelvic mass; (2) the presence of contrast enhancement; and (3) the type of opacification noted.

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Table I

	No.	Rim	Solid or Mottled	No Opacification
Uterine Tumors				
A. Leiomyomata	47	0	40 (85 per cent)	7
B. Endometrial Neoplasm	6	0	1	5
C. Carcinoma of Cervix	3	0	0	3
Ovarian Tumors				
A. Cystic	10	5 (50 per cent)	0	5
B. Mixed (cystic and solid)	4	3 (75 per cent)	1	1
C. Solid	6	0	3 (50 per cent)	3
Pelvic Inflammatory Disease	6	1	1	4

RESULTS

The 80 cases reviewed were divided into 3 major categories: uterine tumors; ovarian tumors; and pelvic inflammatory disease. The presence of contrast enhancement and the type of opacification obtained were tabulated for each group (Table 1).

Forty-seven cases with uterine leiomyomata were studied. None showed rim opacification. Forty showed definite contrast enhancement, with either a solid or mottled appearance (Fig. 1, *A* and *B*). Seven cases with uterine fibroids showed no opacification. In 4 these fibroids were quite small, and no soft tissue mass except an apparently normal uterus could be identified. Two others had large, densely calcified uterine fibroids, and no opacification could be detected, possibly because of the presence of calcium. A total of 10 of the 47 cases had uterine fibroids containing calcifications, which varied from focal punctate calcifications to dense calcification of the entire fibroid. Even in the presence of calcification, contrast enhancement could be detected in 8 cases. Frequently, margins of the uterine fibroids were better delineated allowing determination of actual size.

Uterine malignancies showed opacification in only 1 of the 6 cases reviewed. This patient had a large endometrial carcinoma which had caused moderate enlargement of the uterus. The tumor extended through the uterine wall to involve several adjacent loops of small bowel with fistula formation. The mass showed mottled opacification and was roentgenographically identical to the contrast enhancement seen in many cases of uterine leiomyomata.

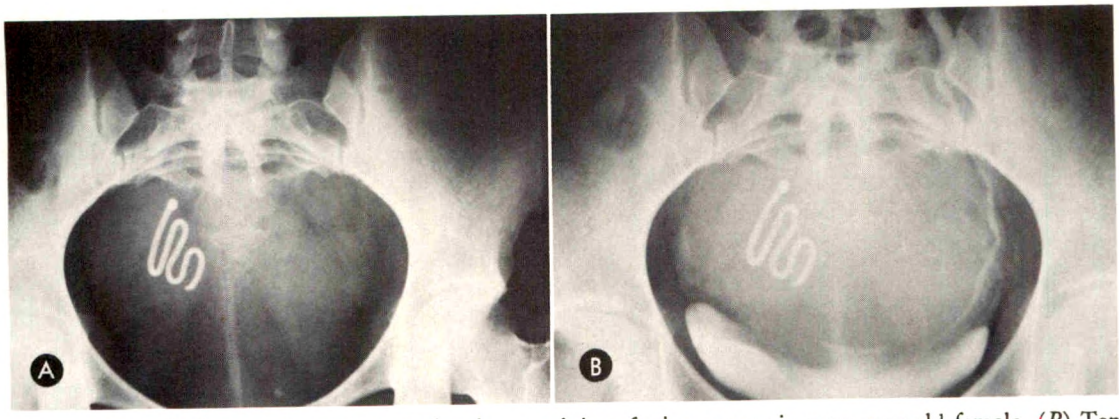

Fig. 1. (*A*) Preliminary roentgenogram showing a pelvic soft tissue mass in a 33 year old female. (*B*) Ten minutes after infusion there is homogeneous opacification of the pelvic mass. An intrauterine contraceptive device is present. Pathologic diagnosis was multiple uterine leiomyomata.

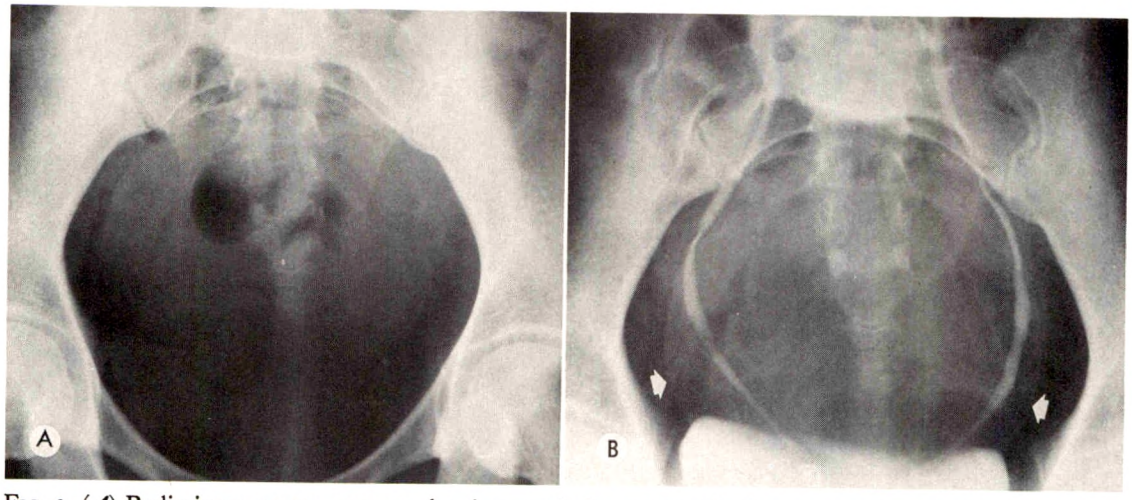

FIG. 2. (*A*) Preliminary roentgenogram showing a soft tissue mass clearly separate from the bladder in a 21 year old female. (*B*) Twenty minutes after infusion there is definite opacification of the cyst wall (arrows). Pathologic diagnosis was mucinous cystadenoma, right ovary.

Of the 4 cases of carcinoma of the cervix reviewed, 1 showed opacification of a roentgenographically normal uterus. No abnormal opacification could be detected.

Ovarian tumors are divided into 3 groups according to their gross pathologic characteristics: (1) cystic, (2) solid, and (3) mixed (*i.e.*, those tumors having both cystic and solid elements).

A total of 20 ovarian masses was studied, 10 of which were cystic. Fifty per cent of these cystic ovarian tumors showed definite contrast enhancement of the cyst wall during infusion urography (Fig. 2, *A* and *B*). In 1 of these lesions, a multilocular cystic malignant teratoma, the septa were enhanced by contrast in addition to the cyst wall (Fig. 3, *A* and *B*). Another cystic ovarian tumor occurred in association with a myomatous uterus. The preliminary film revealed a single large pelvic mass, eccentric in configuration, but homogeneous in density. However, the infusion urogram showed definite contrast enhancement of the myomatous mass which could be clearly separated from the cystic ovarian tumor. One of the cystic tumors that did not show contrast enhancement was a dermoid cyst. This tumor contained no calcium, but the radiolucent characteristic of the cyst contents could be appreciated on the preliminary study. The cyst wall appeared radiopaque, lying between the fatty cyst fluid and the fat covering other adjacent pelvic structures. The cyst wall was clearly visible both before and after infusion. Since no definite change could be detected, this case was rated as no enhancement. Of the remaining 4 cases of cystic ovarian tumors that did not show opacification of the cyst wall, one which was later revealed to be a small (3 cm. in its greatest diameter) serous cystadenoma showed no mass on any study. In the other 3, the cystic ovarian tumors could be identified, but no cyst wall opacification could be detected.

Six solid ovarian tumors were encountered; none showed rim opacification. Three lesions showed solid or mottled contrast enhancement (Fig. 4, *A* and *B*). The opacification could not in itself be differentiated from that seen in uterine fibroids. The 3 remaining solid ovarian tumors showed no contrast enhancement. A soft tissue mass could, however, be identified on the infusion urograms in all 3 cases.

Four ovarian tumors were placed in the mixed category, *i.e.*, those containing both solid and cystic elements. Three of these lesions showed contrast enhancement of the cyst walls, and 1 lesion showed mottled

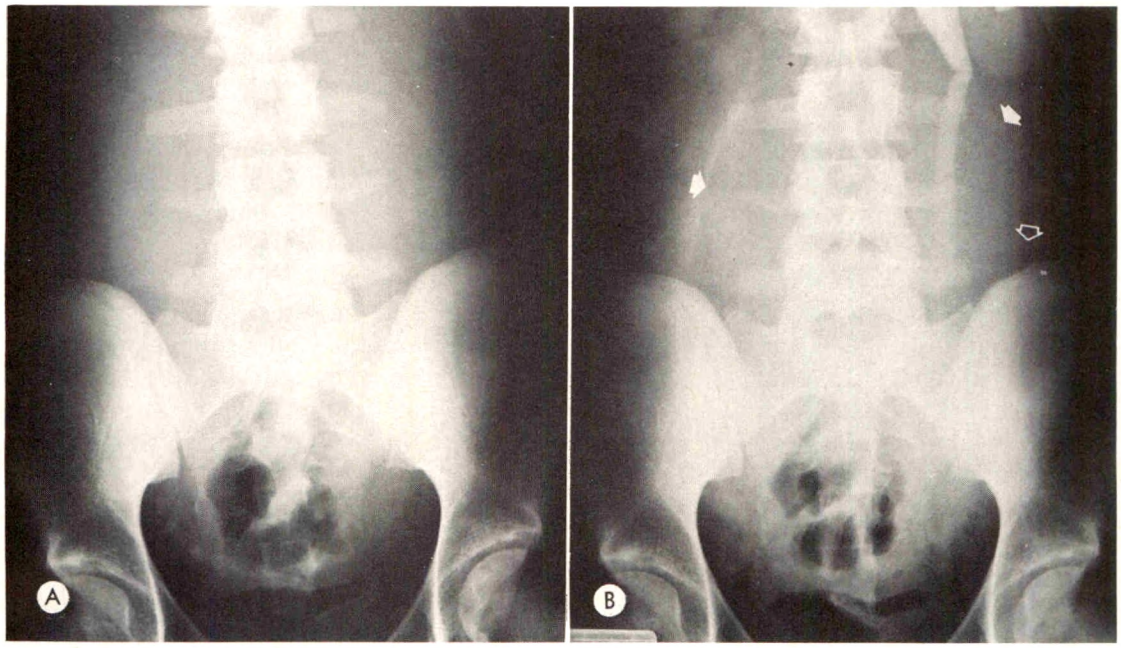

Fig. 3. (*A*) Preliminary roentgenogram shows a poorly defined pelvic and abdominal mass in a 16 year old female. (*B*) Twenty minutes after infusion, opacification of the wall of the abdominal mass (solid arrows) and opacification of septa within the mass (open arrow) are shown. Pathologic diagnosis was malignant teratoma of the ovary with multiple cystic areas.

opacification of the solid portions of the tumor in addition to revealing contrast enhancement of the cyst wall. This lesion was a Brenner cell tumor of the ovary with both cystic and solid elements. There were 2 dermoid cysts in this group, both having typical calcifications present. In one instance, the cyst wall showed contrast enhancement. Neither showed any opacification of the solid elements. The remain-

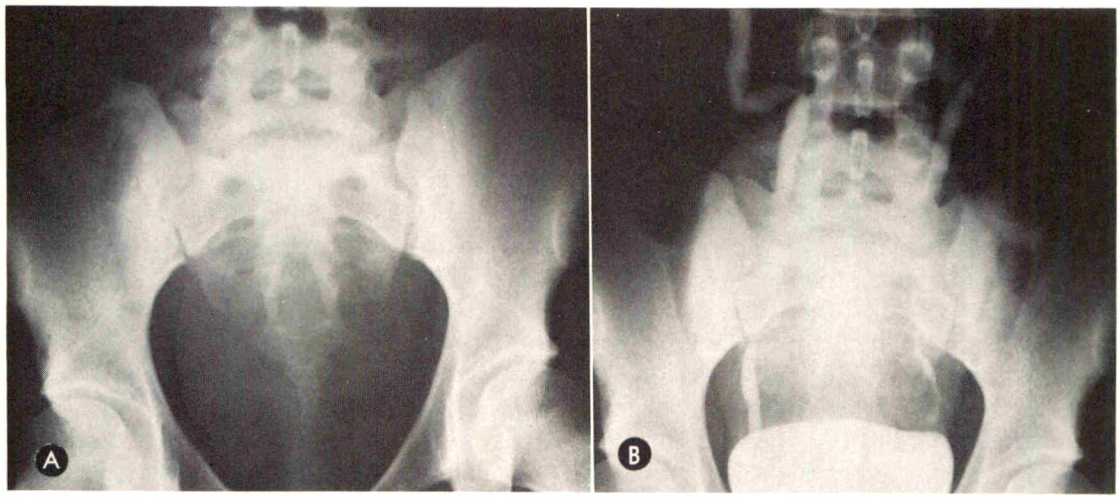

Fig. 4. (*A*) Preliminary roentgenogram of an 18 year old female with amenorrhea and masculinizing signs and symptoms for 18 months. (*B*) Twenty minutes after infusion there is homogeneous opacification of the pelvic soft tissue mass. Pathologic diagnosis was arrhenoblastoma, left ovary.

ing case was a patient with bilateral granulosa cell ovarian tumors which showed a small area of cyst wall opacification in the right ovarian mass. There was also opacification of a normal sized uterus, clearly separating it from the bilateral adnexal masses.

Pelvic inflammatory disease was the primary etiology in 6 cases of pelvic masses. Four of these patients showed no evidence of contrast enhancement. One patient showed definite rim opacification due to a 6 cm. serous cyst of the left fallopian tube associated with chronic salpingitis. One other case showed mottled enhancement of a pelvic soft tissue mass, similar in appearance to a myomatous uterus, except that the borders of the mass were very indistinct. The patient had no uterine leiomyomata, only chronic salpingitis with multiple tubo-ovarian adhesions.

DISCUSSION

Total body opacification and its value in the evaluation of abdominal masses was first described by O'Connor and Neuhauser in 1963.[9] The authors noted that following high doses of intravenous contrast media, opacification of liver and bowel wall as well as abnormal abdominal masses occurred.[5,8,12] Cystic intraabdominal masses could be identified. In addition, opacification of the cyst wall following intravenous injection of contrast medium was noted in this and other reports.[2,4,7,10]

The contrast enhancement of the cyst wall seen in pelvic neoplasms is the result of the opacification of the blood vascular pool. The opacification of the cyst wall is due to the circulating contrast medium in the capillary bed of the cyst wall which can be visualized as a rim around the nonopacified cyst fluid. Because 1 to 2 cm. of cyst wall are tangential to the roentgen beam, this rim can be visualized, thus giving a 1 to 2 cm. effective tissue thickness. In almost all cases, the best opacification of the cyst wall occurred on roentgenograms taken at the time of completion of the infusion, the time of maximum plasma contrast medium levels. The opacification of the cyst wall continues about 30 minutes after completion of infusion, with diminishing intensity. The persistence of this opacification corresponds to the sustained plasma contrast medium levels obtained by the infusion method of excretory urography.[3] It is highly unlikely that there is any active or passive accumulation of contrast in the cyst wall. The walls of ovarian cysts are in general not hypervascular, and therefore the opacification is not due to increased circulation of the cyst wall, which is the explanation offered for the visualization of the gallbladder wall during infusion cholecystography.[11]

The cyst wall was best visualized in the dermoid cysts of the ovary. But since the cyst wall could often be seen prior to the administration of contrast medium, it was quite difficult to evaluate contrast enhancement. In the 4 dermoid cysts studied, only 2 showed definite contrast enhancement of the cyst wall. However, the cyst wall was visible in all 4 cases prior to and following infusion. This ease of visualization of the teratoma wall is due to the radiolucent fat within the cyst and the surrounding fat in the pelvis. Opacification of solid and mixed ovarian lesions is also best explained by the sustained high plasma contrast medium levels. This explanation is supported by the fact that the best opacification occurs at the completion of infusion. The type of opacification was quite similar to that seen in uterine fibroids. These 2 lesions could be differentiated only if: (1) a normal uterus could be distinguished from the ovarian tumor; (2) typical psammomatous calcifications of ovarian carcinoma were present; or (3) typical calcifications of uterine fibroids or an ovarian dermoid were present.

Uterine opacification was seen often during this study. Frequently the normal uterus could be differentiated from the ovarian mass because of the homogeneous uterine opacification. Such opacification of a normal uterus is probably a consequence of the rich capillary bed of the uterus.

The high incidence of contrast enhancement found in this study is in keeping with observations in other studies.[1,10] Birnholz[1] postulated that contrast enhancement was due to passive interstitial accumulation of the contrast agent, thus giving the hysterogram effect. Although Birnholz' theory may be valid, this opacification is more likely to be due to the opacification of the intravascular and interstitial spaces of the uterine fibroids.

An attempt to differentiate solid from mottled opacifications was abandoned because making a clear distinction between these 2 patterns was often not possible. Large and small bowel were frequently superimposed on the pelvic mass. Also feces in the rectosigmoid was projected over most pelvic masses, giving a mottled appearance.

The time of maximum opacification, as mentioned previously, was usually at completion of infusion; however, problems existed in evaluating this finding. First, all excretory urograms are tailored, so that comparable films were not obtained in all cases. Second, because of the amount of contrast agent used, a brisk diuresis occurs. The bladder becomes moderately distended during the course of the study, altering the position of pelvic masses. As the bladder distends, the mass is elevated over the sacrum, thus increasing the apparent radiodensity of the mass. Third, any change in technical factors will alter the apparent radiodensity of all soft tissue masses. Fourth, ureteral compression devices decrease the anteroposterior diameter of the lower abdomen, therefore enhancing the visualization of pelvic masses by displacing overlying bowel. Fifth, prone films also alter relationship of the bladder to the pelvic mass, and the bladder will often partially or completely obscure the mass.

Tomography, which has been used for the visualization of the gallbladder wall, has proved useful in the evaluation of pelvic masses.[1] Although additional information can be obtained by this method, we do not routinely use pelvic tomography because of the additional time and pelvic irradiation encountered.

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REFERENCES

1. Birnholz, J. C. Uterine opacification during excretory urography: definition of previously unreported sign. *Radiology*, 1972, *105*, 303–307.
2. Carlson, D. H., and Griscom, N. T. Ovarian cysts in newborn. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 664–672.
3. Cattell, W. R. Excretory pathways for contrast media. *Invest. Radiol.*, 1970, *5*, 473–486.
4. Griscom, N. T., and Neuhauser, E. B. D. Total body opacification. *J. Pediat. Surg.*, 1966, *1*, 76–79.
5. Kittredge, R. D., Kanick, V., and Finby, N. Value of nephrotomography in differential diagnosis of abdominal masses. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 935–947.
6. Love, L., Moncada, R., Melamed, M., and Cooper, R. Infusion tomography of the female pelvis. Scientific Exhibit at the 59th Scientific Assembly and Annual Meeting of the Radiological Society of North America, Chicago, Ill., Nov. 26–30, 1973.
7. Martin, D. J., Griscom, N. T., and Neuhauser, E. B. D. Further look at total body opacification effect. *Brit. J. Radiol.*, 1972, *45*, 185–192.
8. Mitty, H. A., and Schapira, H. E. Total body opacification in adult. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 630–635.
9. O'Connor, J. F., and Neuhauser, E. B. D. Total body opacification in conventional high dose intravenous urography in infancy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 63–71.
10. Phillips, J. C., Easterly, J. F., and Langston, J. W. Contrast enhancement of pelvo-abdominal masses: rim sign. *Radiology*, 1974, *112*, 17–21.
11. Rabushka, S. E., Love, L., and Moncada, R. Infusion tomography of gallbladder. Scientific Exhibit at the 58th Scientific Assembly and Annual Meeting of the Radiological Society of North America, Chicago, Ill., Nov. 26–Dec. 1, 1972.
12. Salzman, E., and McClintock, J. Opacification of small bowel with intravenously administered contrast medium. Exhibit, Tenth International Congress of Radiology, Montreal, Canada, Aug., 1962.

THE UTILIZATION AND EFFICACY OF PELVIMETRY*

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ABSTRACT:

Data from non random groups of deliveries with some identical clinical factors were compared and certain conclusions are suggested.

Most x-ray examinations which cause significant fetal exposure are pelvimetries. There appears to be no uniform set of indications for performing pelvimetry. Instead, the criteria seem to vary from hospital to hospital and even from physician to physician within the same hospital. A normal pelvimetry gives the physician little confidence that a cesarean section will not be necessary. The duration of labor is not significantly altered by pelvimetry in those cases needing a cesarean section for cephalopelvic disproportion or following unsuccessful oxytocin stimulation.

ROENTGENOGRAPHIC evaluation of the relative sizes of the fetal head and maternal pelvis has been used clinically almost since the advent of medical radiography. The technique was considerably refined by Colcher and Sussman in 1944.[5] Further refinements and variations have been instituted since that time.[2,4,6,7,9,12]

Even though pelvimetry has been widely used over the years and has been generally accepted as a clinical tool, its efficacy has been questioned recently by some authorities.[8,11,13,14] Any doubts about the usefulness of this procedure assume additional significance in light of evidence that irradiation of the fetus, even at the low levels used in diagnostic radiology, may carry with it an increased risk of leukemia and other malignant tumors in childhood.[3,10,16,17]

Because of these questions of risk and efficacy, a study of selected information from 67,078 single deliveries in the years 1969 and 1970 was undertaken to evaluate the utilization and efficacy of pelvimetry. This information was collected and provided by the Obstetrical Statistical Cooperative based at Downstate University in Brooklyn, New York. The Cooperative is an association of teaching hospitals which collects data on all deliveries at these hospitals for later use in retrospective investigation by any member institution.

MATERIAL AND METHOD

This study analyzed clinical information from 67,078 single deliveries of 1,000 gm. or greater from 16 hospitals. Pelvimetries were performed during 6.9 per cent (4,599) of these deliveries, a rate which is similar to the 5.1 per cent found in a 1963 U.S.P.H.S. national survey.[19] The cases, although constituting about 1 per cent of the deliveries in the United States for 1969 and 1970, do not constitute a representative sample in a number of ways. As illustrated in Table 1, the population studied differed considerably from what would be expected in a random national sample in many geographical, economic and racial factors. Also, all of the hospitals providing data in this study were training institutions; consequently, a greater proportion of the deliveries were assisted by interns and residents than would be expected on a national basis.

The Cooperative case record consists of approximately 75 datum elements, the most pertinent of which are listed in Table

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TABLE I

BIAS OF DATA

	Obstetrical Statistical Cooperative Population	National Population (1, 18)
1. Geographical	90 per cent deliveries from New York City Area 69 per cent deliveries from inner cities	5.3 per cent <30 per cent
2. Economic	70 per cent ward patients	est. 29 per cent
3. Racial	43 per cent non-white	17.2 per cent
4. Type of Care	100 per cent teaching hospitals 69 per cent trainee deliveries	41.7 per cent est. 29 per cent
5. Parity	100 per cent single births	97.5 per cent

11. The attending physicians abstracted the clinical data onto a data form. This form was checked for completeness by one person, generally a medical secretary, at each participating hospital and the information transferred to magnetic computer tape.

GOALS AND LIMITATIONS OF THE STUDY

The data accumulated through this study provide direct information on the frequency of pelvimetry use, at least among the participating hospitals. But the more important questions of "Why is pelvimetry done?" and "Is there any benefit from the procedure?" cannot be answered directly by this study, since the data collection system did not ascertain the attending physician's indications for the pelvimetry, nor his own direct evaluation of its usefulness in each case. However, the study was used to extract information indirectly on the indications for and benefits from pelvimetry. With respect to indications, certain clinical conditions associated with high rates of pelvimetry were identified by computer, enabling us to infer that these conditions were commonly employed indications for the x-ray procedure. We were able to derive answers to the question of benefit by observing whether some of the commonly assumed *goals* of pelvimetry (a shortening of nonproductive labor in certain clinical circumstances, for example) were actually accomplished in x-rayed women as compared with those who had not had pelvimetry.

TABLE II

PARTIAL LIST OF DATA AVAILABLE ON PATIENTS IN THE STUDY

1. Pelvimetry
2. Other X-ray Examinations
3. Method of Delivery
4. Complications of Delivery
5. Complications of Pregnancy
6. Position of Fetus
7. Type of Pelvis
8. Prior Pregnancies
9. Prior Abortions
10. Prior Fetal Deaths
11. Prior Medical Diseases
12. Prior Surgery
13. Present Medical Diseases
14. Reason for Operative Intervention
15. Age of Mother
16. Weight of Mother
17. Height of Mother
18. Race of Mother
19. Weight Gain of Mother
20. Weeks of Pregnancy
21. Birth Weight
22. Anomalies
23. Resuscitation
24. Infant Diseases
25. Reason for Neonatal Death

One further word of caution is necessary. This study is an evaluation of the effect of pelvimetry on groups and not on individuals. As in the case of any statistical investigation, some individual patients may prove to be exceptions to the generalizations drawn from the study.

DISCUSSION

1. *Pelvimetry as a contributor to fetal irradiation.* Simple tabulation of the data showed that pelvimetries accounted for 72 per cent of all the examinations which delivered direct x-ray exposure to the fetuses in this study. In other words, nearly three-fourths of the pelvic and abdominal x-ray procedures in the study were pelvimetries. This is considerably higher than the 50 per cent reported in the 1963 U.S.P.H.S. study.[19] This difference may be explained by the substitution of ultrasound for many nonpelvimetric x-ray examinations of pregnant women since 1963. *Consequently, pelvimetry may now have emerged as the greatest single source of fetal irradiation by far.* As ultrasound becomes more and more widely available, this phenomenon can be expected to continue.

2. *Indications for pelvimetry.* As mentioned above, the question of "Why is pelvimetry done?" was not directly answerable because the physicians' reasons for requesting pelvimetry were not recorded in the original data. However, a decision analysis computer program[15] was used to weigh the relative importance of the recorded clinical factors associated with pelvimetry. This generated a decision tree which revealed some of the reasons for the pelvimetries, as shown in Figure 1. The computer program divided the deliveries first into 2 subgroups; each subgroup was then divided into 2 others, etc. This division process continued until little additional variation in pelvimetry rates could be explained by further subdivision. Each division was based on the clinical factor that would give the highest possible pelvimetry rate in the subgroup with that factor and the lowest rate in the subgroup without it.* For example, uterine dysfunction was the first determinant chosen by the computer program because it was the clinical factor which best divided the deliveries into high and low pelvimetry rate subgroups. The program then treated each of these two subgroups separately and divided them again on the next most important factor (fetal position), and so on.

The reasons for pelvimetries which were discovered by the process are summarized in Table III. They can be divided into 3 categories: (1) patient-related indications important in most cases; (2) physician-related factors important in most cases; and (3) patient-related indications important in only a limited portion of the study population. The fact that the computer program selected physician-related factors (the experience of the attendant physician at delivery and the particular hospital in which the pelvimetry was done) seems to indicate that pelvimetries may not be ordered solely on uniform and objective clinical grounds. In addition, it was particularly disquieting that only 20 per cent of the variation in whether or not pelvimetries were ordered could be explained by the decision analysis program. The remaining variation appeared to be due to a lack of consistency within the data themselves. In other words, the most important determinants in the decision to use pelvimetry seem not to be consistent clinical factors or combinations of factors, but rather the variation of attending physicians in identical clinical circumstances.

3. *Pelvimetry and cesarean section rates.* The pelvimetry rate varied from 1.8 per cent to 15.7 per cent among the 16 hospitals studied. Since pelvimetric examinations serve primarily as an aid in deciding whether or not to perform a cesarean section, it might be expected that there would be some relationship between pelvimetry

* More accurately, the program uses a standard least squares method to maximize the explained variation. With pelvimetry taking on values 1 (yes) or 0 (no), the explained variation is the ratio of the sums of the squares of the deviations from the final group means to the sum of the squares of the deviations from the original group mean.

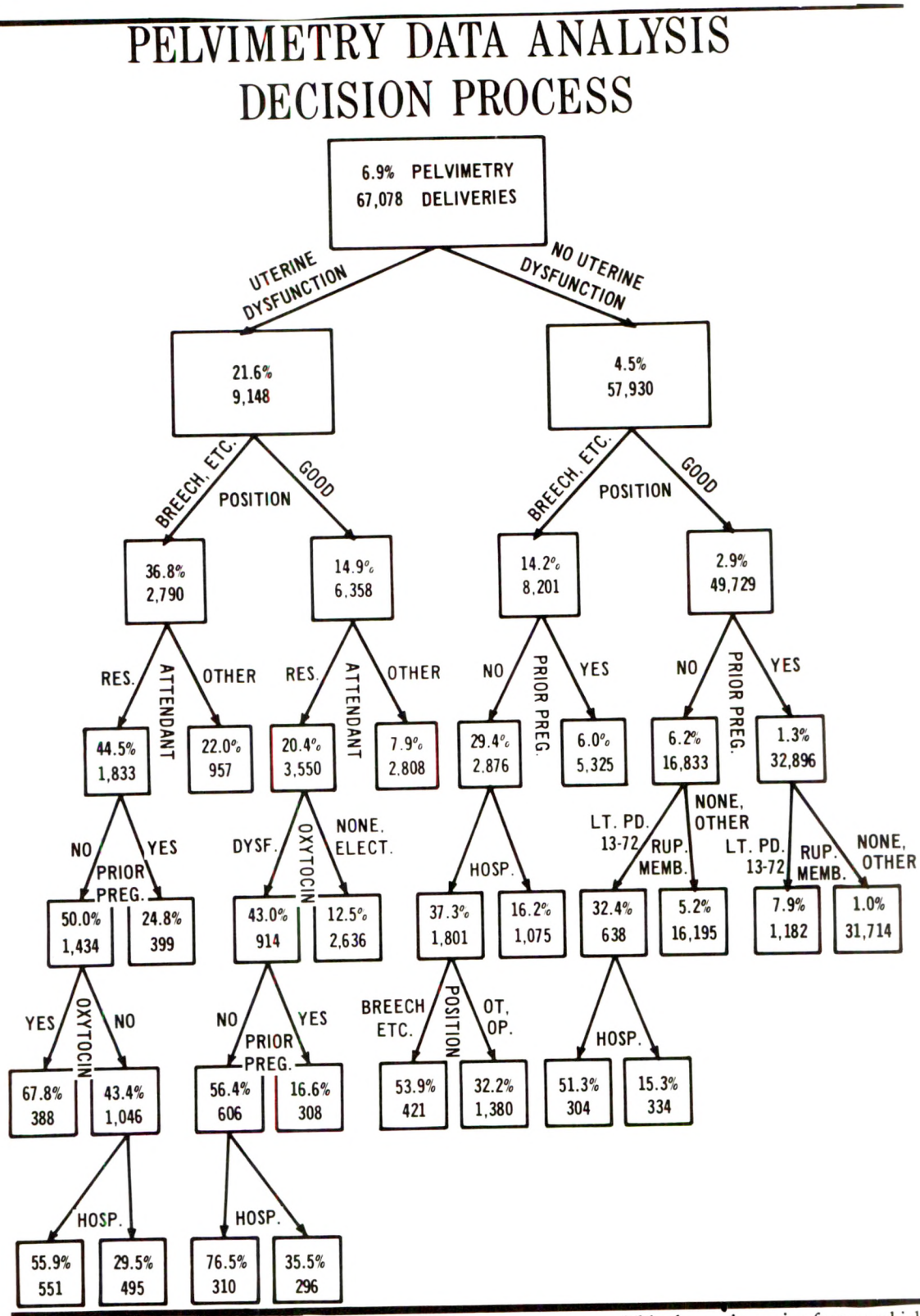

Fig. 1. Decision tree, generated by decision analysis computer program. This shows the major factors which accounted for some of the variation in the use of pelvimetry in the study hospitals.

TABLE III

IMPORTANT FACTORS IN THE DECISION TO DO A PELVIMETRY

1. Patient related factors important in most cases
   (A) Strength of uterine contractions
   (B) Position of the fetus
   (C) Any prior term pregnancies
2. Physician related factors important in most cases
   (A) Experience of the attending physician
   (B) Hospital or departmental practice
3. Patient related factors important in select cases
   (A) Premature rupture of the membranes
   (B) Length of labor
   (C) Use of oxytocin
   (D) Toxemia
   (E) Weeks of pregnancy

rates and cesarean section rates at the various hospitals. In fact, the correlation coefficient was 0.28, which is not significantly different from 0. This shows that there was little or no consistent relationship between the pelvimetry and cesarean section rates at the hospitals in the study. This is graphically demonstrated in Figure 2.

Since there was considerable variation among the hospitals in the rate of primagravid women coming to delivery, it might be argued that a hospital with a higher percentage of primagravid women might have a relatively high rate of pelvimetry which would contribute to lack of correlation shown above. However, when the pelvimetry rate was compared to the cesarean rate for all 16 hospitals, for those women who had never previously had a term pregnancy, the correlation coefficient was even lower (0.11).

4. *Effectiveness of pelvimetry.* The marked variability in the use of pelvimetry among physicians in similar clinical situations, along with the lack of correlation between pelvimetry use and cesarean rate, raised the question of whether pelvimetries were beneficial at all. Since pelvimetry should be used only for comparative measurements of the fetal head and maternal pelvis in order to determine whether safe vaginal delivery is possible, the fundamental question is whether the physician can make this decision more accurately and more quickly with the aid of pelvimetry than would be possible using clinical judgment alone.

a. Prognostic value of the normal pelvimetry. The data show that 41 per cent of patients undergoing cesarean section received a pelvimetry. Among those who had a pelvimetry which was interpreted as normal, 27 per cent nonetheless went on to have a section. One of the arguments in favor of pelvimetry has been that when the clinician has roentgenographic confirmation of a normal pelvis, he is able more judiciously to allow labor to progress. However, these findings contradict this. The decision to do a pelvimetry puts a woman in a class with a high rate of cesarean sections, about 4 out of 10. The finding that her pelvis is normal reduces her chance of cesarean section slightly (to about 3 out of 10). In other words, because a significant chance of cesarean section still exists in women with a normal pelvimetry, the clinician still has to make the same decision about cesarean section on clinical grounds that he would have had to make even if he had not done the pelvimetry in the first place.

b. Prognostic value of pelvimetry in cephalopelvic disproportion. If pelvimetry helps to provide an early and accurate diagnosis of cephalopelvic disproportion and thus allows for timely intervention with cesarean section, then women with disproportion who have had pelvimetry should undergo, on the average, a shorter labor prior to section than those with disproportion who have not received pelvimetry. With this in mind, the duration of labor was compared for 2 groups of patients in the study: those diagnosed as having cephalopelvic disproportion and undergoing cesarean section who had received a pelvimetry, and those with the same diagnosis and section who had not had a pelvimetry. To make the groups more alike,

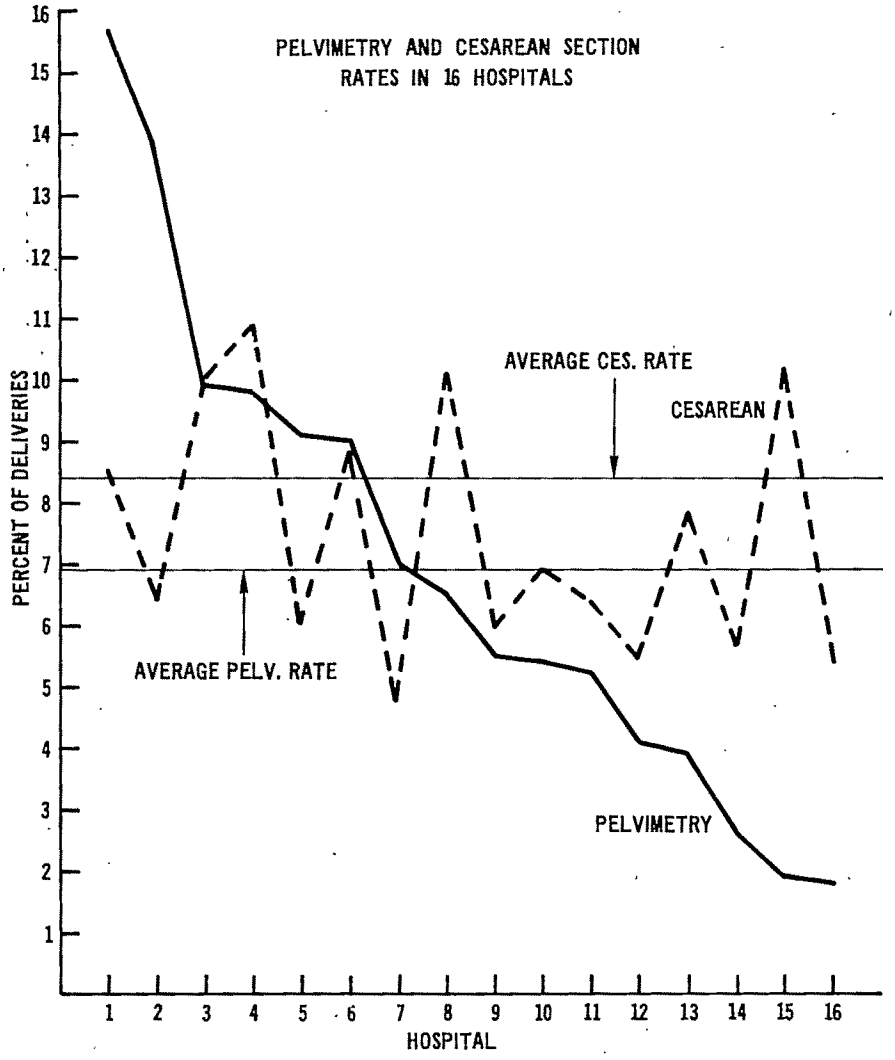

FIG. 2. Comparison of pelvimetry and cesarean section rates found in 16 member hospitals of the Obstetrical Statistical Cooperative.

only women both delivering for the first time and having infants greater than 2,500 gm. were included. As shown in Figure 3, there was essentially no difference between the two groups with respect to hours of labor. In other words, as it was used in the 16 hospitals, pelvimetry did not significantly shorten labor in those women with cephalopelvic disproportion when compared to the use of clinical judgment alone.

c. Prognostic value of pelvimetry on oxytocin failures. Since a pelvimetry is done to prevent those with a pelvis inadequate for vaginal delivery from having an unnecessarily long labor, only data concerning the oxytocin failures are presented here (Fig. 4 and 5). Two separate groups, those having elective stimulation and those having stimulation for dysfunction were considered. First, patients having an unsuccessful elective oxytocin stimulation, with or without pelvimetry, were compared to see if pelvimetry shortened the duration of labor prior to cesarean section. Again, only those women with at least a 2,500 gm. fetus and who had not had a

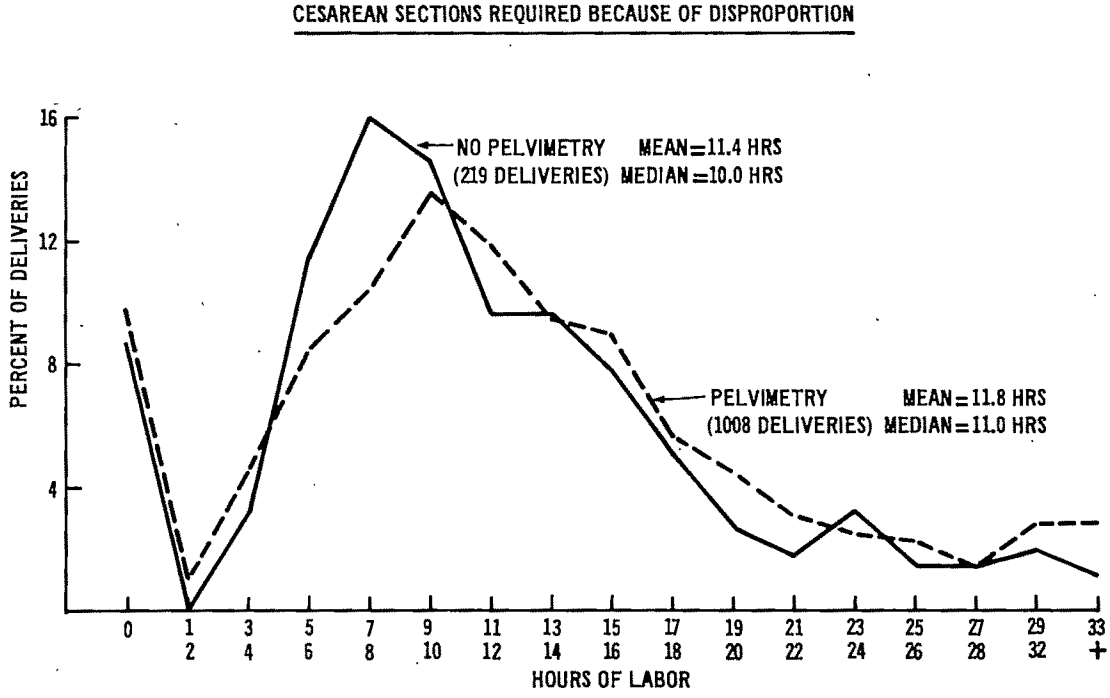

FIG. 3. Comparison of duration of labor experienced by patients, with and without pelvimetry, who had cesarean sections because of cephalopelvic disproportion.

previous term pregnancy were used in this comparison so that the groups would be composed of similar individuals. Figure 4 reveals no significant difference between the 2 groups.

Next, the patients with unsuccessful oxytocin stimulation for dysfunction were considered. Duration of labor for these women is shown in Figure 5. The mean and median labor lengths for the women with pelvimetry were about 2 hours longer than those without pelvimetry. A number of factors could account for this. For example, the longer length of labor among the x-rayed women might be related to the time consumed in ordering, performing, and interpreting a pelvimetry; in many hospitals it may take up to an hour or more to transport the patient to the x-ray department, carry out the procedure, and return her to the delivery suite. Some of the delay in performing necessary cesarean sections among the x-rayed women may be related to indecisiveness or procrastination on the part of those physicians who most often use pelvimetries. This suggestion is supported by the computer decision analysis which showed a greater use of pelvimetry by the less experienced attendant physicians.

It should be noted that any difference between the pelvimetry and non-pelvimetry groups in this study could possibly be accounted for by a basic difference between the groups beyond the fact that some were x-rayed and others were not; retrospective studies such as this are particularly prone to such difficulties. For example, differences in duration of labor among the pelvimetry and non-pelvimetry groups could be due to a greater proportion of difficult cases, *i.e.*, those in which a clinical decision is harder to make, within the pelvimetry group. But it is difficult to imagine enough variation in the distribution of "difficult cases" among the 16 hospitals in the study to account for the ninefold variation in the use of pelvimetry observed among them.

In any case, as can be seen from Figures

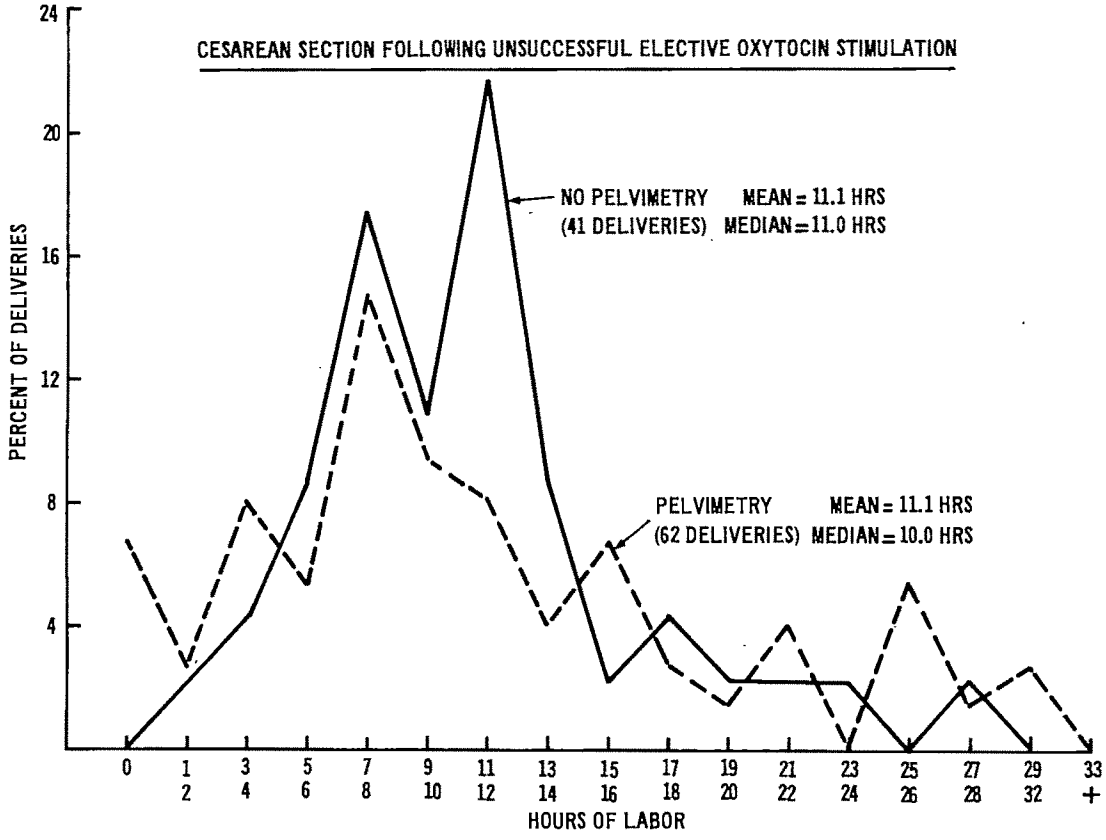

FIG. 4. Comparison of duration of labor experienced by patients, with and without pelvimetry, who had cesarean sections following unsuccessful oxytocin stimulation. The difference at 0 hours between the 2 groups is due only to 3 patients and is not statistically significant.

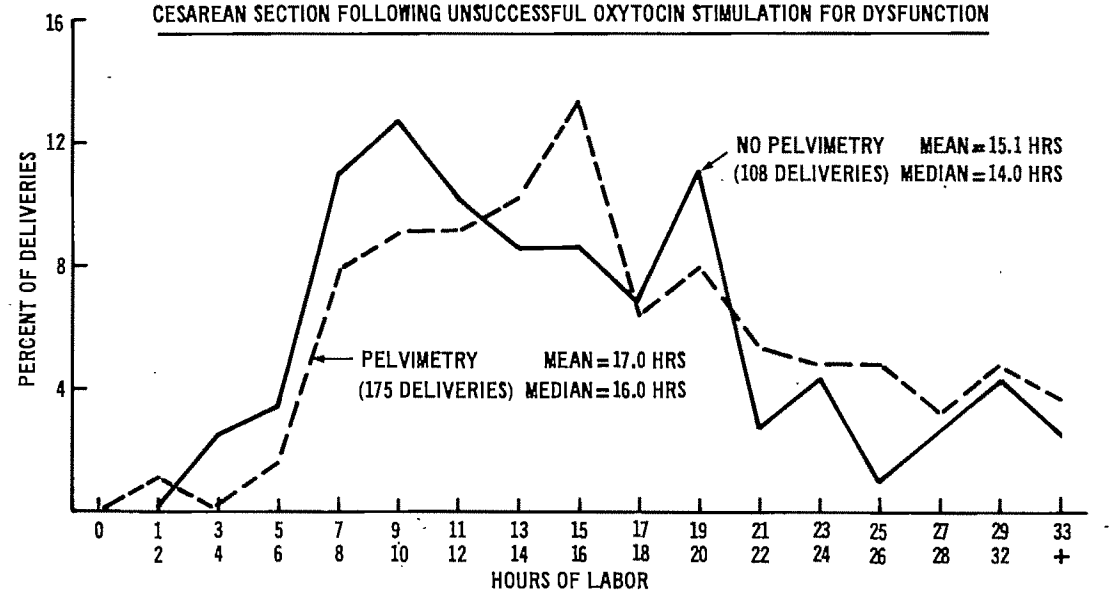

FIG. 5. Comparison of duration of labor experienced by patients, with and without pelvimetry, who had cesarean sections, following unsuccessful oxytocin stimulation for uterine dysfunction.

4 and 5, pelvimetry did not decrease the length of labor in those who ultimately went to cesarean section following unsuccessful oxytocin.

The authors feel that the findings from this study warrant a prospective investigation of the efficacy of pelvimetry.

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REFERENCES

1. American Hospital Association, Chicago, Illinois. Personal correspondence concerning statistics for 1970.
2. BORELL, U., and RADBERG, C. Orthodiagraphic pelvimetry with special reference to capacity of distal part of pelvis and pelvic outlet. *Acta radiol.* (Diag.), 1964, *2*, 273–282.
3. BROSS, I. D. J., and NATARAJAN, N. Leukemia from low-level radiation, identification of susceptible children. *New England J. Med.*, 1972, *287*, 107–110.
4. BROWN, R. C. Modification of Colcher-Sussman technique of x-ray pelvimetry. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *115*, 623–629.
5. COLCHER, A. E., and SUSSMAN, W. Practical technique for roentgen pelvimetry with new positioning. AM. J. ROENTGENOL. & RAD. THERAPY, 1944, *51*, 207–214.
6. DIEHL, J., and FERNSTROM, I. Radiologic pelvimetry with special reference to widest transverse diameter of pelvic inlet. *Acta radiol.* (Diag.), 1966, *4*, 557–568.
7. FRIEDMAN, E. A., and TAYLOR, M. B. Modified tomographic aid for x-ray cephalopelvimetry. *Am. J. Obst. & Gynec.*, 1969, *105*, 1110–1115.
8. HANNAH, W. J. X-ray pelvimetry—critical appraisal. *Am. J. Obst. & Gynec.*, 1965, *91*, 333–340.
9. JOHNSON, T. H., JR., and REAGAN, T. A. Roentgenologic rule and pelvimetry technique. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *105*, 850–893.
10. MACMAHON, B. Prenatal x-ray exposure and childhood cancer. *J. Nat. Cancer Inst.*, 1962, *28*, 1173–1191.
11. MCGRUDER, C. E. X-ray pelvimetry at teaching hospital: ten year evaluation. *J. Nat. M. A.*, 1968, *60*, 213–214.
12. MURRAY, J. P. Semi-orthometric pelvimetry: appraisal. *Brit. J. Radiol.*, 1971, *44*, 524–530.
13. RUSSELL, J. G. B., and RICHARDS, B. Review of pelvimetry data. *Brit. J. Radiol.*, 1971, *44*, 780–784.
14. SCHWARZ, G. S. Radiation hazards to human fetus in present day society. *Bull. New York Acad. Med.*, 1968, *44*, 388–399.
15. SONQUIST, J. A., BAKER, E. L., and MORGAN, J. N. Searching for structure (Alias-Aid-III): approach to analysis of substantial bodies of micro-data and documentation for computer program. Institute for social research. The Univ. of Michigan, Ann Arbor, Michigan, 1971.
16. STEWART, A. M., and KNEALE, G. W. Age-distribution of cancers caused by obstetric x-rays and their relevance to cancer latent periods. *Lancet*, 1970, *2*, 4–8.
17. STEWART, A., WEBB, J., and HEWITT, D. Survey of childhood malignancies. *Brit. M. J.*, 1958, *2*, 1495–1508.
18. U.S. Dept. of H.E.W. Health Resources Adm. Final natality statistics 1970, Monthly Vital Statistics 1970 (HRA) 74–1120, vol. 22, 12, p. 6, 10.
19. U.S. Dept. of H.E.W. National Center for Health Statistics. Medical x-ray visits and examinations during pregnacy—United States 1963, Public Health Serv. Publication No. 1000, Series 22, No. 5, 1968.

VOL. 125, NO. 1

INTESTINAL PERFORATION AS A COMPLICATION OF THE NEONATAL SMALL LEFT COLON SYNDROME*

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ABSTRACT:

Intestinal perforation can occur as a complication of the neonatal small left colon syndrome. Four such infants are reported.

Early diagnosis and treatment, utilizing water soluble contrast enema examination, are recommended to reduce the frequency of this complication.

THE neonatal small left colon syndrome (SLCS) was recently reported by Davis *et al.*,[3] as a condition producing signs and symptoms of low colonic obstruction. Affected infants are often the offspring of diabetic mothers. Roentgen examination of the abdomen shows multiple dilated small bowel loops and sometimes visualization of dilated ascending and transverse portions of the colon. Contrast enema study demonstrates a characteristic pattern of a small left colon to the level of the splenic flexure where a sharp transition zone exists with the proximal colon being dilated.

This syndrome has been considered a benign condition in which contrast enema examination was curative by stimulating meconium evacuation. We wish to supplement these concepts by reporting 4 cases with intestinal perforation as a complication of this condition. The case histories and roentgen findings in 3 patients, all infants of diabetic mothers, recently seen at Primary Children's Medical Center will be reported. A fourth case, recently contributed by Dr. Davis will also be reviewed.

REPORT OF CASES

CASE 1. This 4,500 gram female infant was transferred to Primary Children's Medical Center at age 2 days because of abdominal distention. She was the product of a 38 week gestation and the pregnancy was complicated by toxemia. The mother was an insulin dependent diabetic. Mild cyanosis secondary to persistent fetal circulation syndrome and hypoglycemic cardiomyopathy was present during the first 24 hours. Only a minimal amount of meconium had been passed prior to her transfer to this hospital.

Abdomen roentgenograms demonstrated numerous dilated loops of bowel and a pneumoperitoneum.

Perforation due to abdominal distention had not occurred as a complication of SLCS in our previous experience with infants of diabetic mothers. We felt that SLCS with perforation was an unlikely cause for the pneumoperitoneum and therefore elected to do a water soluble contrast enema to evaluate the possibility of aganglionosis and to rule out the colon as the site of perforation. This study showed characteristic findings of SLCS without extravasation of contrast medium into the peritoneal cavity (Fig. 1).

Exploratory laparotomy revealed multiple small perforations with necrotic margins in the ileum approximately 10 cm. proximal to the ileocecal valve. This segment of bowel was resected and an ileostomy done.

The patient survived and is doing well at 1 year of age. Her ileostomy has been closed several months. A follow-up barium enema done at age 5 weeks showed the entire colon to appear normal with no disparity in size between the right and left sides (Fig. 2).

* Presented at the Seventeenth Annual Meeting of the Society for Pediatric Radiology, San Francisco, California, September 23, 1974.

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FIG. 1. Case I. A large pneumoperitoneum outlines the falciform ligament. The appearance of the colon is typical of the neonatal small left colon syndrome. The colon is small to the splenic flexure with dilatation proximal to the transition zone.

CASE II. This 2,400 gram female infant of an insulin dependent diabetic mother was transferred to Primary Children's Medical Center after intestinal perforation was identified. She was the product of a 36 week gestation which was complicated by polyhydramnios.

Mild respiratory distress and cyanosis felt to be secondary to hypoglycemic cardiomyopathy and persistent fetal circulation syndrome were initially present, and were controlled by supportive medical measures. At 24 hours of age the patient had not passed meconium, and abdominal distention was noted. Small saline enemas were unsuccessful in relieving this condition. Roentgen examination of the abdomen at this time revealed very mild dilatation of the cecum with the remainder of the intestinal gas pattern appearing normal.

At 48 hours of age a pneumoperitoneum was present on an abdomen film (Fig. 3). A gastrografin enema done at another hospital was reported to show transition zone at the splenic flexure with the proximal colon of larger caliber. Contrast medium entered the peritoneal cavity after reaching the cecum (Fig. 4). A subsequent postoperative abdomen roentgenogram confirmed the splenic flexure transition zone (Fig. 5).

Exploratory laparotomy confirmed the cecal perforation, and a cecostomy was done. Attempts to control her chemical abnormalities, cardiovascular problems and peritonitis were unsuccessful and the patient died of sepsis and disseminated intravascular coagulation at age 11 days.

Postmortem examination revealed candida abscesses of the liver, lung and kidney and a large subarachnoid hemorrhage. Changes associated with maternal diabetes and the bowel perforation were also noted.

Tissue specimens from the right and left colon were reviewed by Blaise E. Favara, M.D., co-author of the original paper describing SLCS. The ratio of multipolar ganglion cells to small cells in the intermyenteric plexus in this patient was reduced, as in their first reported cases of this syndrome.

CASE III. This 4,400 gram female infant was transferred to Primary Children's Medical Center at 24 hours of age because of abdominal distention and failure to pass meconium. She was born to a diabetic mother after a 36 week gestation with the pregnancy complicated by toxemia. Hypoglycemic cardiomyopathy and persistent fetal circulation syndrome resulted

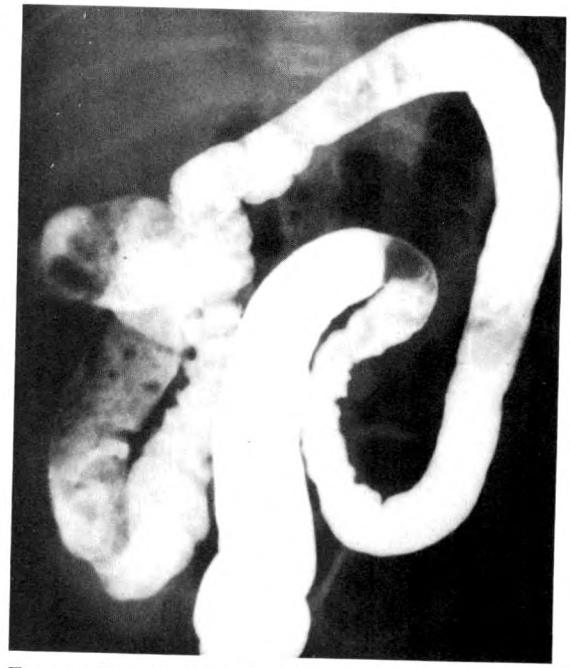

FIG. 2. Case I. Normal appearance of a follow-up barium enema colon examination done at 5 weeks of age.

in cyanosis during the first day.

Abdomen roentgenograms demonstrated dilated bowel loops proximal to the splenic flexure region of the colon (Fig. 6). A contrast enema (Fig. 7) showed the distal colon to be moderately small to the level of the mid-descending colon with some dilatation proximal to the transition zone. Contrast medium refluxed into the terminal ileum. No definite extravasation of contrast medium into the peritoneal cavity was noted. Following this enema study, a large amount of meconium was expelled, but despite continued meconium passage the abdominal distention progressed and her condition deteriorated. An abdomen roentgenogram done 6 hours after the contrast enema study showed a pneumoperitoneum and contrast medium in the peritoneal cavity (Fig. 8).

Exploratory laparotomy revealed a large cecal perforation. The peritoneum was lavaged and the cecum exteriorized at the perforation site. Postoperatively, the patient's condition continued to deteriorate and she died at 48 hours of age. Autopsy permission was denied.

Hirschsprung's disease was not completely ruled out in this patient. The facts that the patient was female, premature by gestation duration, and the infant of a diabetic mother strongly support the diagnosis of SLCS rather than Hirschsprung's disease.

Case IV. This 2,820 gram male infant was the product of a 37–40 week gestation. Although there was a positive family history, the mother was not diabetic. At 24 hours of age abdominal distention was noted.

An abdomen roentgenogram revealed dilated bowel loops with the colon appearing involved to the region of the splenic flexure. Contrast

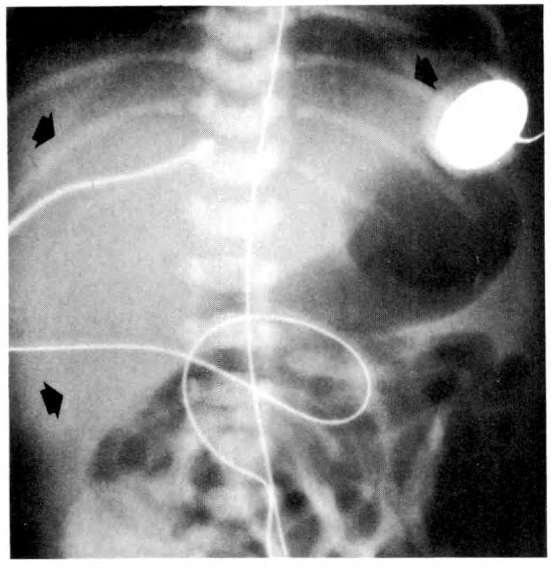

Fig. 3. Case II. Supine abdomen roentgenogram demonstrates a large pneumoperitoneum (arrows).

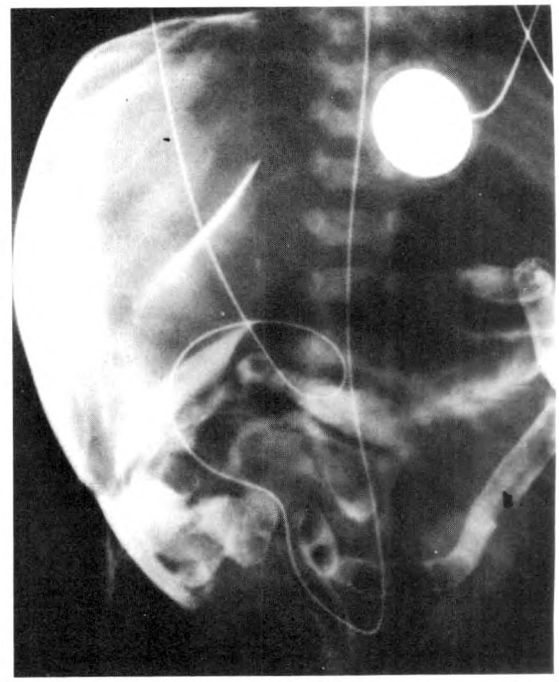

Fig. 4. Case II. Water soluble contrast enema reveals extensive extravasation into the peritoneal cavity.

Fig. 5. Case II. Follow-up postoperative abdomen roentgenogram confirms the transition zone in the splenic flexure area of the colon.

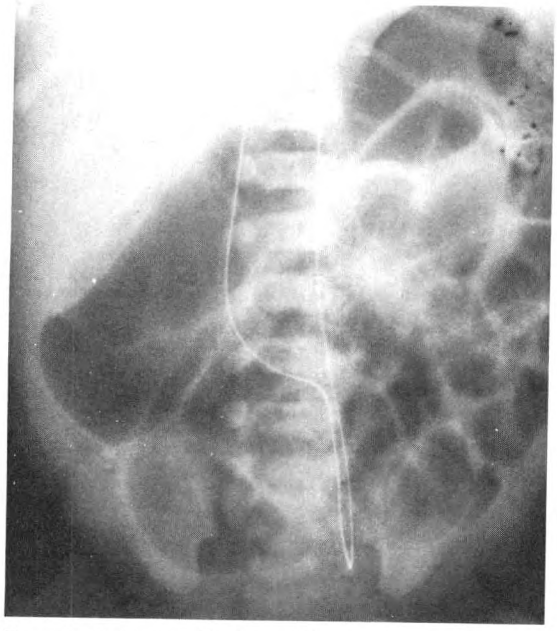

FIG. 6. Case III. Supine abdomen roentgenogram shows numerous dilated loops of small bowel and colon to the splenic flexure region. This pattern is typical of the neonatal small left colon syndrome.

enema examination at 2 days of age demonstrated a small left colon to the mid-descending colon region with proximal dilatation (Fig. 9).

Abdominal distention progressed and a repeat contrast enema was performed at 3 days of age again showing changes of SLCS. An abdomen roentgenogram done later that day demonstrated contrast medium in the peritoneal cavity (Fig. 10).

Following the diagnosis of intestinal perforation this baby was transferred to Denver Children's Hospital. Laparotomy revealed the perforation site to be in the cecum. A cecostomy was done and a rectal biopsy showed ganglion cells to be present. This patient survived and has done well, currently being 3 months of age.

DISCUSSION

There are multiple etiologies of gastrointestinal perforation in the newborn. The more frequently reported causes include gastric perforation of the newborn, meconium ileus, small bowel or colon atresia, necrotizing enterocolitis, Hirschsprung's disease, imperforate anus, trauma, and the meconium plug syndrome. Intestinal perforation associated with the neonatal small left colon syndrome has not been previously reported. It should be noted, however, that previous cases reported as idiopathic or spontaneous perforations of the small bowel or proximal colon certainly

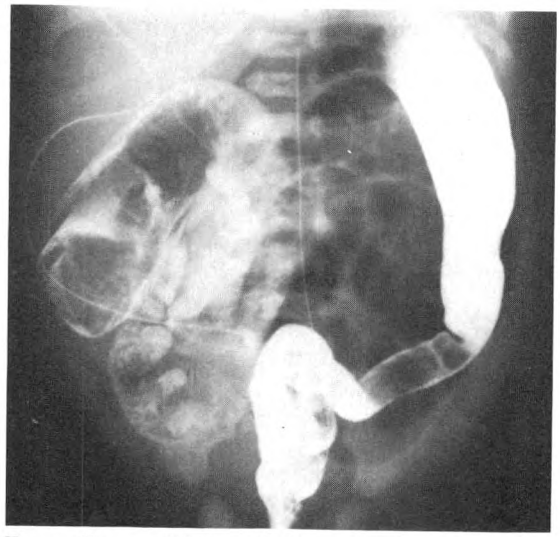

FIG. 7. Case III. Water soluble contrast enema examination reveals the left colon to be small to the mid-descending region without apparent extravasation or pneumoperitoneum.

FIG. 8. Case III. Pneumoperitoneum and extravasated contrast medium are demonstrated on this follow-up abdomen roentgenogram.

may have occurred secondary to this syndrome.[4,7]

Intestinal perforation secondary to dilatation and focal necrosis can occur proximal to a mechanically or functionally obstructed colon. Factors other than dilatation and pressure necrosis may contribute to intestinal perforation in infants with the neonatal small left colon syndrome (Fig. 11). It has been postulated that selective intestinal circulatory ischemia ("the diving reflex") occurs in neonates with hypoxia or shock.[6] Infants of diabetic mothers commonly have respiratory disease, hypoglycemic cardiomyopathy and persistent fetal circulation syndrome (PFCS)[5] which could trigger this asphyxial defense mechanism.

Perforation secondary to an obstructive colon lesion usually occurs in the cecum. This was the location in 3 of these 4 cases. With associated bowel ischemia, other sites may be involved as in our other case where the perforation occurred in the distal ileum.

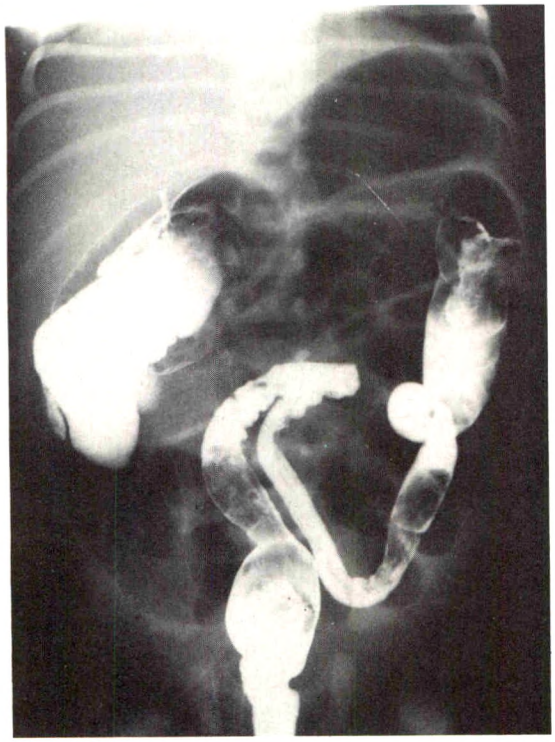

Fig. 9. Case IV. Contrast enema roentgenogram shows a small left colon with an abrupt transition zone near the splenic flexure.

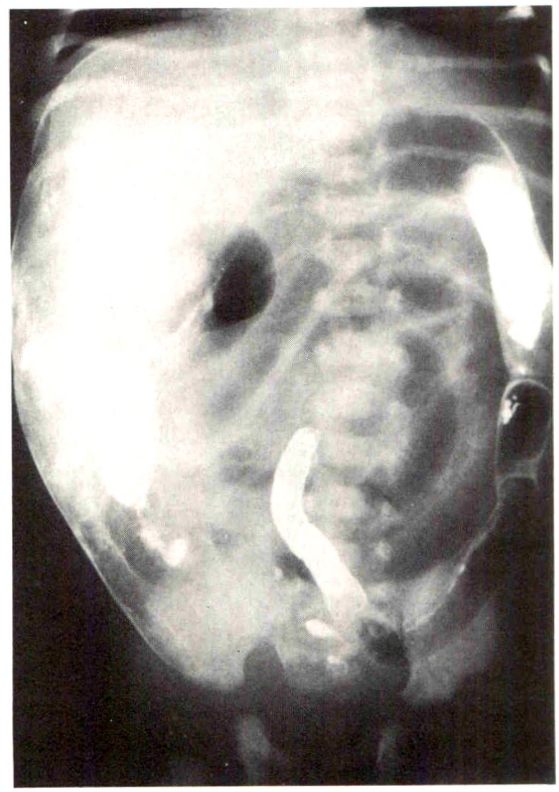

Fig. 10. Case IV. Follow-up abdomen roentgenogram reveals extravasation of contrast medium into the peritoneal cavity.

These cases of the neonatal small left colon syndrome with perforation demonstrate the potentially serious nature of this condition and the need for early diagnosis

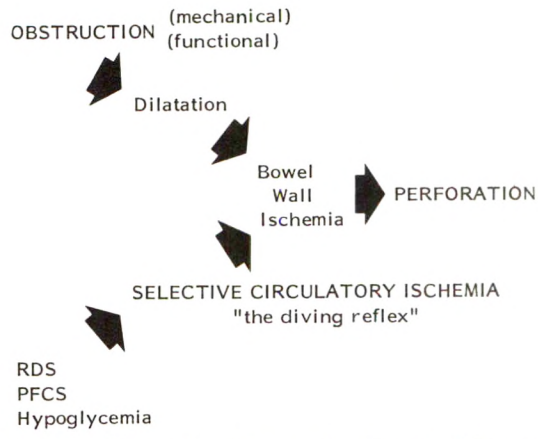

Fig. 11. Schematic diagram showing possible contributing factors resulting in intestinal perforation in SLCS.

and treatment. The mortality rate in newborns with gastrointestinal perforation is high.[4,7] This is especially true in infants of diabetic mothers with associated serious complicating medical problems.

Early diagnosis and treatment are accomplished with a water soluble contrast enema examination. We recommend that this study be done immediately in newborns who develop clinical findings of colon obstruction or fail to pass significant meconium within 24 to 48 hours. This is particularly critical in infants of diabetic mothers. This aggressive approach hopefully should reduce the incidence of intestinal perforation as a complication of the neonatal small left colon syndrome.

Repeat contrast enema examinations may occasionally be necessary to relieve the obstructive signs in these babies.[2] Also, undiluted or 1:1 diluted water soluble contrast medium may result in irritability and spasm of the distal colon. Further dilution to 1:4 has subsequently been successful in diagnosis and treatment.[2]

Although we recommend the use of water soluble contrast media in the evaluation and management of SLCS it must be noted that these agents are less accurate than barium in diagnosing aganglionosis.[1] Clinicians must be informed that if signs and symptoms of obstruction persist, a follow-up examination using barium is essential to evaluate more accurately the possibility of long segment Hirschsprung's disease.

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We wish to express our appreciation to Dr. William S. Davis, Denver Children's Hospital, Denver, Colorado, for Case IV.

REFERENCES

1. BERDON, W. E., and BAKER, D. H. In: Pediatric X-Ray Diagnosis. Sixth edition. By J. Caffey. Year Book Medical Publishers, Inc., Chicago, 1972, pp. 1515.
2. DAVIS, W. S. Personal communication.
3. DAVIS, W. S., ALLEN, R. P., FAVARA, B. E., and SLOVIS, T. L. Neonatal small left colon syndrome. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1974, *120*, 322–329.
4. FREEARK, R. J., RAFFENSPERGER, J. G., and CONDON, J. B. Pneumoperitoneum in infancy. *Surg., Gynec. & Obst.*, 1961, *113*, 623–630.
5. GERSONY, W. M. Persistence of fetal circulation: commentary. *J. Pediat.*, 1973, *82*, 1103–1106.
6. LLOYD, J. R. Etiology of gastrointestinal perforations in newborn. *J. Pediat. Surg.*, 1969, *4*, 77–84.
7. THOMAS, C. C., JR., and BROCKMAN, S. K. Idiopathic perforation of colon in infancy. *Ann. Surg.*, 1966, *164*, 853–858.

VOL. 125, NO. 1

ANGIOGRAPHY IN EXPANSILE LESIONS OF THE SPLEEN*

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ABSTRACT:

The angiographic findings in a relatively large number of expansile lesions of the spleen are presented. In cysts our findings correspond to those in earlier reports, the hallmark being stretching of intrasplenic arteries and veins around an avascular expansile lesion. As to neoplasms (primary as well as secondary), however, our findings suggest that the criteria of malignancy, that is, neovascularity, vascular encasement, and arteriovenous shunting, may be more frequent than is evident from earlier reports.

"SPLENIC tumors are rare. The experience that any individual angiographer can accumulate in the various manifestations of splenic neoplasms, therefore, is small."[21] To be able to review a larger sample, cases were collected from 2 University Hospitals. The findings do not always correspond to those of earlier reports and are thus presented.

MATERIAL AND METHOD

The 22 cases will be presented in groups: cysts, 8; primary neoplasms, 4; metastases, 4; and secondary tumor invasion, 6. Selective celiac angiography was performed in all cases, followed by selective examination of the splenic artery in some cases.

At angiography the interest has often been focused on the liver, sometimes resulting in poor quality splenograms. The angiographic findings in all cases were verified by histological diagnosis at operation or autopsy soon after the examination.

RESULTS

CYSTS

Age and sex of the patients, together with the angiographic findings in this group, are given in Table 1.

In 6 patients only 1 lesion was evident while in 2 cases multiple cysts were present. The spleen was enlarged in 7 patients and of normal size in 1. Calcifications in the wall of the cysts were seen in 2 cases. In 1 of

TABLE I

AGE, SEX, AND ANGIOGRAPHIC FINDINGS IN THE PRESENT MATERIAL OF SPLENIC CYSTS

Case	Age	Sex	Size of Spleen (cm.)	Size of Lesion (cm.)	Diameter of Splenic Artery (mm.)	Diameter of Hepatic Artery (mm.)	Ratio Splenic /Hepatic Artery	Calcification
1	46	M	17×15	14×13	5.0	7.0	0.7	0
2	7	F	21×12	15×10	5.0	5.0	1.0	0
3	26	F	26×18	23×18	8.0	7.0	1.1	0
4	18	M	26×19	24×19	8.0	6.0	1.3	0
5	25	F	28×17	25×17	5.5	5.0	1.1	0
6	35	M	23×17	16×17, 3×3	9.0	6.0	1.5	++
7	44	F	14× 9	multiple, <2	7.0	3.5	2.0	0
8	63	M	10× 5	1×1	8.0	9.0	0.9	+

* From the Department of Diagnostic Radiology, University Hospital, Lund, Sweden† and the Department of Diagnostic Radiology, Allmänna Sjukhuset, Malmö, Sweden.‡

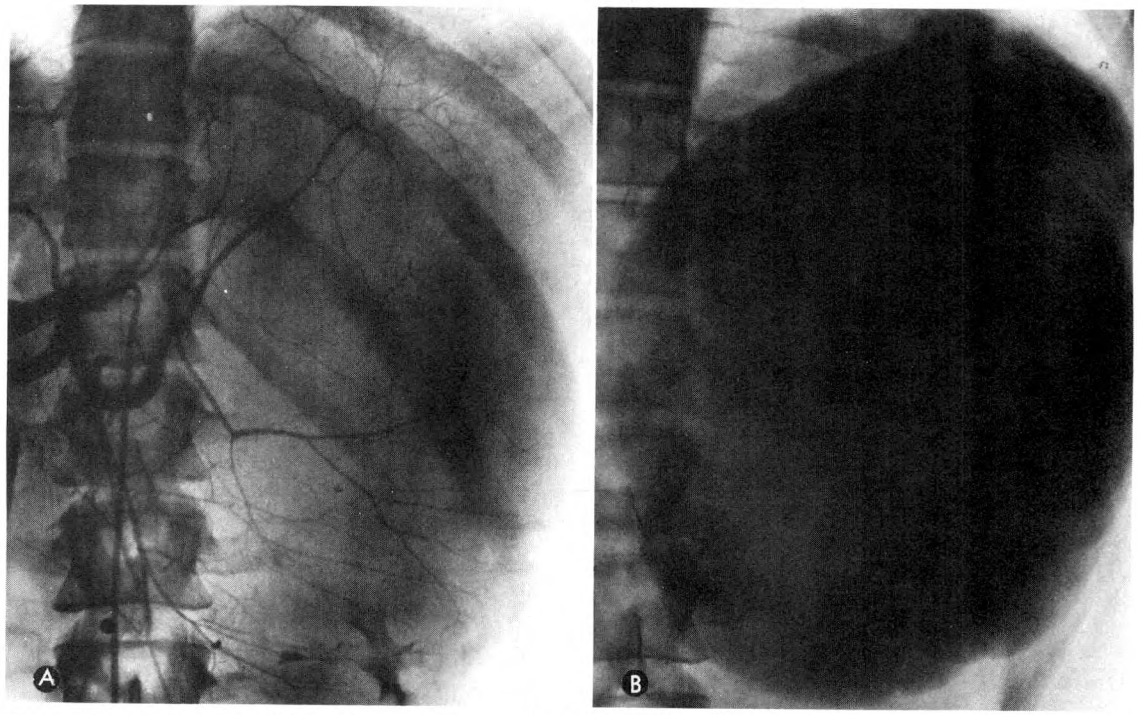

FIG. 1. Traumatic cyst of the spleen in man, aged 18. (*A*) Celiac angiography, arterial phase: intrasplenic arteries smoothly stretched around an avascular mass. Left kidney displaced downward. (*B*) Following cyst puncture with withdrawal of chocolate-coloured fluid and injection of water-soluble contrast medium.

these plain roentgenograms of the abdomen revealed a tiny, semilunar shaped calcification in a normal size spleen. Angiography revealed normal arteries and veins and a 2 mm. thick staining around an avascular area measuring 1×1 cm. The histological diagnosis was a partly calcified cyst, etiology unknown.

The remaining 7 cases had similar angiographic findings (Fig. 1*A*). In each there was caudal dislocation of the splenic artery and vein, and normal intrasplenic arteries were smoothly stretched around the mass. In some cases displacement of intrasplenic veins could be demonstrated. During the capillary phase the lesions were quite translucent, compared to the normal parenchyma stain. In all 7 patients the remaining parenchyma was localized in the most caudal part of the spleen. The demarcation between the lesion and the parenchyma was sharp and smooth, with occasional lipping.

Displacement of adjacent organs was present in 7 cases. The left inferior phrenic artery was 25–50 per cent wider than the right one. The ratio of the diameter of the splenic artery compared with that of the hepatic artery varied from 0.7 to 2.0. Percutaneous puncture of 2 of the cysts was made with withdrawal of chocolate-coloured fluid and injection of water-soluble contrast medium (Fig. 1*B*).

In Cases 1 through 6 the cysts were probably traumatic in origin, while in Case 7 multiple cysts were also present in liver and kidneys, thus indicating a congenital origin.

PRIMARY NEOPLASMS

Four cases have been examined. The first, a 64 year old female with a primary spindle-cell sarcoma displayed a lesion which was hypovascular in the cranial part of the spleen, whereas the caudal part contained multiple neoplastic vessels. Extensive arteriovenous shunting and irregular staining were evident as well as pronounced

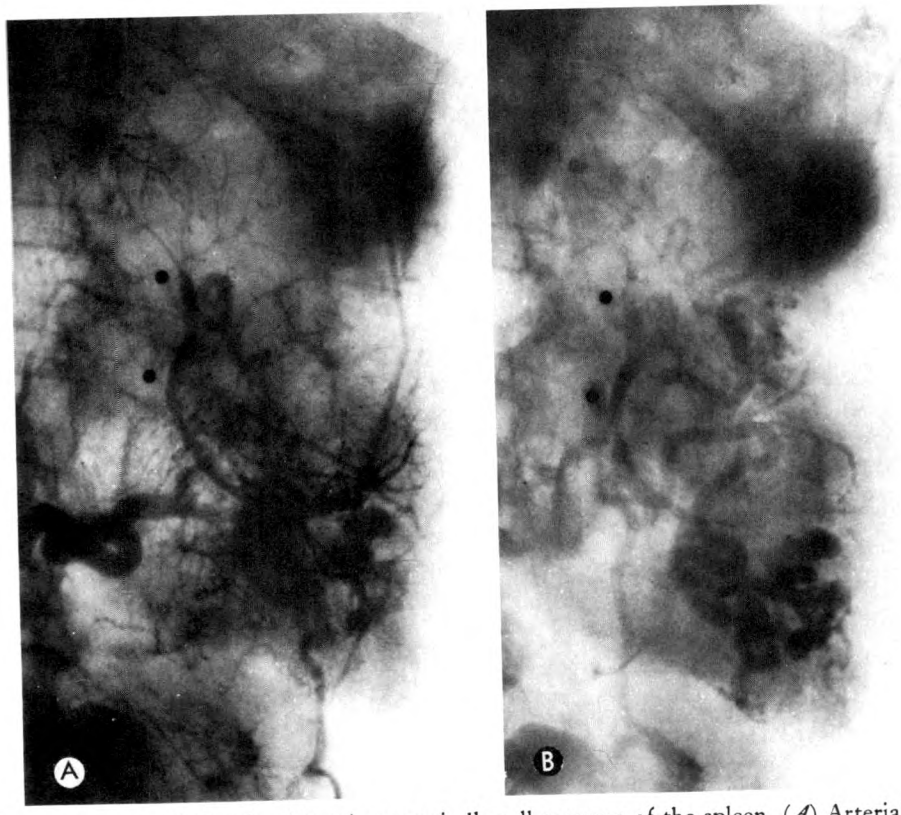

Fig. 2. Sixty-four year old female with primary spindle-cell sarcoma of the spleen. (*A*) Arterial phase. The tumor is hypovascular in its cranial part and contains multiple neoplastic vessels in the caudal part. Displacement as well as encasement of arteries and marked arteriovenous shunting. (*B*) Venous phase. Occlusion of splenic vein with multiple collateral veins. Irregular tumor staining. Kidney displaced caudally.

displacement of arteries and veins surrounding the lesion (Fig. 2*A*). The splenic vein was occluded (Fig. 2*B*).

The following 3 cases of reticulum-cell sarcoma showed different angiographic patterns. In one 76 year old female, whose spleen measured 23×11 cm., the entire spleen was involved. There was encasement of the main splenic artery and of many of its branches. Tiny tumor vessels were present as was pronounced central tumor staining. Also noted were compression of intrasplenic veins and occlusion of the main splenic vein, causing collateral drainage (Fig. 3).

The pattern was similar in the case of a 66 year old female, whose spleen measured 16×11 cm., but the lesion was limited to the lower pole of the spleen. The border between the lesion and the normal parenchyma was rather sharp but slightly irregular. Some arteriovenous shunting was evident (Fig. 4, *A* and *B*). The splenic vein was compressed, but not occluded.

In our third case of reticulum-cell sarcoma no tumor vessels were demonstrable. The patient was a 47 year old female with a splenic size of 29×15 cm. Arteries and veins in the cranial pole of the spleen were displaced by a hypovascular mass measuring approximately 15×15 cm. with no arteriovenous shunting demonstrable. Histologic examination revealed sarcomatous infiltration in the upper pole with central necrosis. In some places infiltrated vessels were noted.

METASTASES

Four cases of metastases were encountered. In 2 instances the primary lesion was

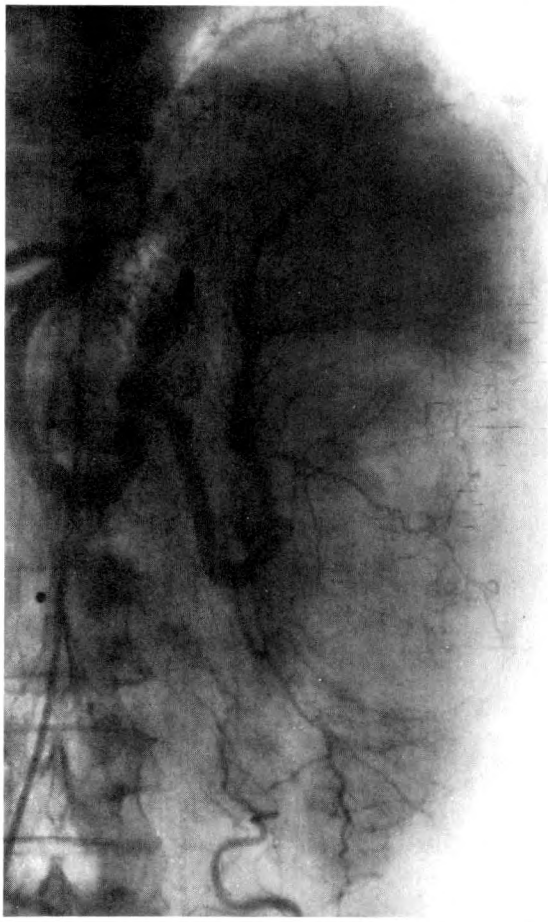

FIG. 3. Seventy-six year old female with primary reticulum-cell sarcoma of the spleen. Celiac angiography. Enlarged spleen (23×11 cm.). Encasement of multiple intrasplenic arteries, especially in upper pole and tiny tumor vessels throughout the spleen. Slight encasement of main splenic artery. Tumor stain medially. The splenic vein was occluded.

situated in the kidney, while in the other it was localized in the uterus and urinary bladder, respectively.

One 60 year old female had been operated upon for uterine carcinoma 7 years prior to the examination. The splenic size was 20×15 cm. Celiac angiography revealed an avascular lesion in the cranial part of the spleen. In the lower pole there were abundant neoplastic vessels (Fig. 5) and moderate tumor staining. There was pronounced displacement of intrasplenic veins, but the main splenic vein was open. Histological examination disclosed old bleeding in the upper pole of the spleen and metastasis from uterine carcinoma in the lower.

Two males, aged 66 and 69, respectively, had primary renal carcinomas. At celiac angiography multiple hypervascular lesions with a diameter up to 4.5 cm. were demonstrated in the spleen. There was displacement of arteries but no encasement was found. The splenic vein was normal in both

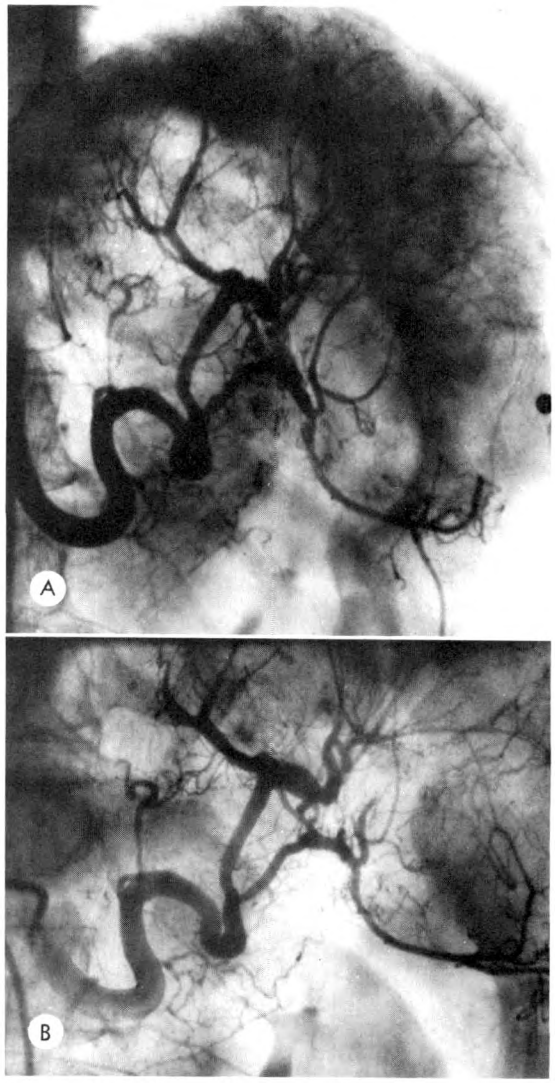

FIG. 4. Reticulum-cell sarcoma in female, aged 66. Encasement of distal part of splenic artery and of arteries in hilum and caudal part of the spleen. Neoplastic vessels in these areas. Some arteriovenous shunting was evident on a later film. (*A*) Celiac and (*B*) selective splenic artery injections.

cases. These patients also had hepatic metastases.

Finally, in a 70 year old male with carcinoma of the urinary bladder, celiac angiography showed a slightly enlarged spleen with 3 poorly vascularized lesions measuring 8×6, 5×5, and 3×3 cm., respectively, in the upper pole, Displacement of intrasplenic arteries and veins was found as well as arterial encasement.

SECONDARY GROWTH FROM ADJACENT ORGANS

One 57 year old woman had been operated upon because of a retroperitoneal leiomyosarcoma 6 years prior to the examination. At angiography a 12×10 cm. lesion in the spleen was found with neoplastic vessels and extensive arteriovenous shunting. Pronounced tumor staining was evident in the capillary phase. There was compression of the splenic vein with secondary collateral vessels (Fig. 6). The stomach and pancreas were involved as well.

Fig. 5. Sixty year old female with metastasis to the spleen from uterine adenocarcinoma. Avascular mass in upper pole with stretched arteries. Displaced, encased arteries and multiple neoplastic vessels in lower pole of the spleen. Histology: Old bleeding in upper pole, metastasis from uterine carcinoma in lower pole.

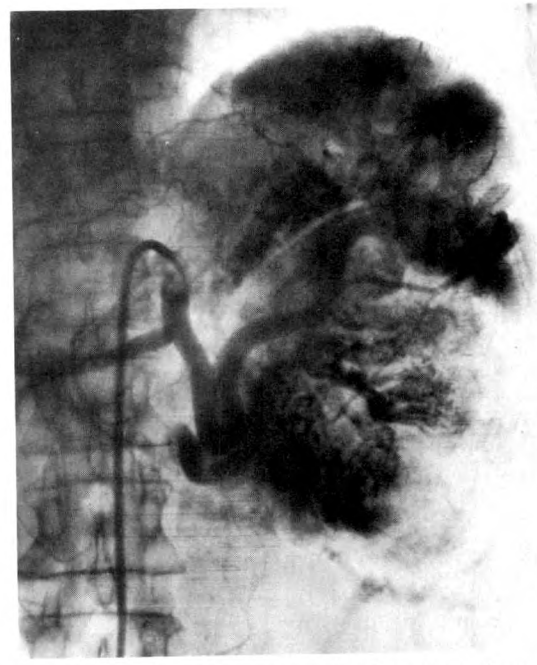

Fig. 6. Female, aged 57, previously operated upon because of a retroperitoneal leiomyosarcoma. Large tumor occupying lower part of the spleen with abundant neoplastic vessels and pronounced arteriovenous shunting. Compression of splenic vein and venous collaterals from caudal part of the lesion. At operation stomach and pancreas were invaded as well.

Celiac angiography in a 51 year old male with a large carcinoma of the pancreas revealed encasement of the splenic artery and intrasplenic branches in the hilum and several other places in the spleen. There were compression and displacement of the splenic vein in the hilum and of many intrasplenic veins. Encasement of the superior mesenteric artery and middle colic artery was also noted.

Invasion of the spleen from a left renal carcinoma was found in 2 females, aged 69 and 62, respectively. In both cases encased arteries and tumor staining immediately beneath the hilum of the spleen were demonstrated at celiac angiography. Arterial as well as venous displacement in the region

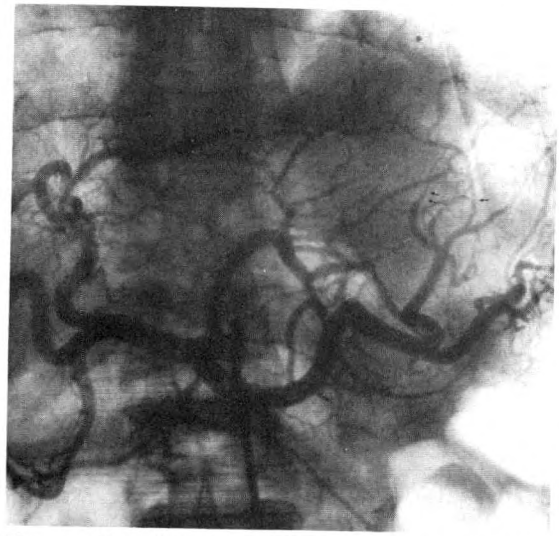

FIG. 7. Sixty-five year old male with gastric carcinoma invading the spleen. Celiac angiography: The carcinoma is supplied from a widened left gastric artery. Encasement and displacement of arteries in upper pole of the spleen (arrows).

of the lesion was evident and in 1 of the cases there was parasitic blood supply to the renal tumor from the splenic artery.

In one 65 year old male invasion of the spleen from a large gastric carcinoma was demonstrated at celiac angiography. The splenic size was 11×9 cm., and the lesion size was 6×6 cm. Encasement and displacement of arteries in the cranial pole of the spleen, as well as tumor staining in parts of the mass and displacement of intrasplenic veins, were encountered (Fig. 7).

Finally, in a 60 year old man with a carcinoma of the splenic flexure of the large bowel, celiac angiography revealed encasement and occlusion of the splenic artery in the hilum of the spleen and compression of the splenic vein, which was not occluded, however. No tumor stain could be demonstrated. At operation invasion of the hilum of the spleen was found.

DISCUSSION

CYSTS

There are mainly 3 types of benign cysts, the congenital, the post-traumatic and the parasitic. In northern Europe hydatid cystic disease is very uncommon and we cannot present any case. Rizk *et al.*[22] in 1971 described their experiences in 6 patients with this disease. In 5, a rim of increased density around the cyst was seen in the capillary phase, probably representing the ectocyst.

Congenital cysts may now and then be encountered. The angiographic findings do not differ from those of traumatic origin[4] with the exception that the lesions are multiple and smaller and often present in other organs. The findings in traumatic cysts in the present material correspond to those reported previously.[2,5,15,20,23,24]

Important features are the displacement of vessels around an avascular lesion and the sharp, smooth demarcation toward the remaining parenchyma, which in all of our cases was localized in the most caudal part of the spleen. There is no infiltration or occlusion of vessels. Displacement of adjacent organs and of the main splenic artery and vein may occur as in any enlargement of the spleen. We first expected the diameter of the splenic artery to be reduced when only small amounts of parenchyma remained. We therefore calculated a ratio between the diameter of the splenic and hepatic arteries, and found that the range and size of the ratio are about the same as in normal spleens. This suggests that the blood flow through the small remaining part of parenchyma may be close to normal. If that reasoning is also applicable to the function is impossible to judge, as the absence of the spleen does not disturb the patient.

Two cystic lesions in our material were punctured percutaneously with injection of water-soluble contrast medium. This diagnostic method was reported by Denneen[6] in 1942, Wicht *et al.*[27] in 1953, Ellis[9] in 1958, and Eban[7] in 1959. The procedure is very easy to perform, but obviously the examiner must be quite sure of not puncturing an echinococcus cyst where the danger of spread of the parasite as well as of an anaphylactic reaction is pronounced. The puncture discloses a typical cyst with smooth rounded walls.

Delorme *et al.*[5] gave as differential diag-

noses myxoma, fibroma, lipoma and teratoma, without showing angiographically proved examples of these diseases. Other rare conditions resembling cysts are osteomas, chondromas and lymphangiomas.[15,24] Infiltrative diseases such as leukemia, lymphoma and reticuloses have also been considered as differential diagnostic possibilities.[15,26]

PRIMARY NEOPLASMS

The first malignant tumor of the spleen diagnosed by angiography was described by Edsman[8] in 1954. He reported "many pathological vessels" in a tumor with the histological diagnosis of probable malignant endothelioma. Rösch[23] in 1966 described a hemangiosarcoma of the liver also involving the spleen, where at angiography the arterial phase showed multiple irregular foci which remained filled in the capillary phase. Delorme *et al.*[5] in 1971 described 1 case of anaplastic epithelial tumor and 1 lymphosarcoma. They state that generally in malignant tumors of sarcomatous type, changes of arterial caliber, neovascularity, thrombosis of the arteries and veins, and irregular staining may be seen.

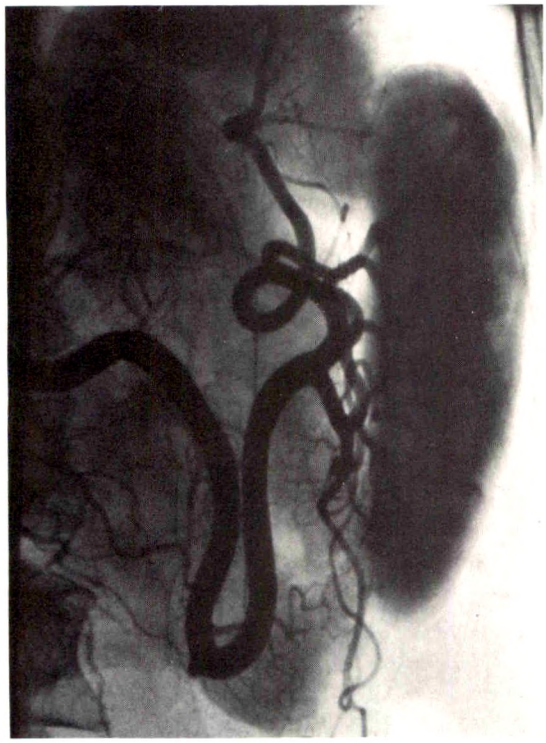

FIG. 8. Primitive spleen.

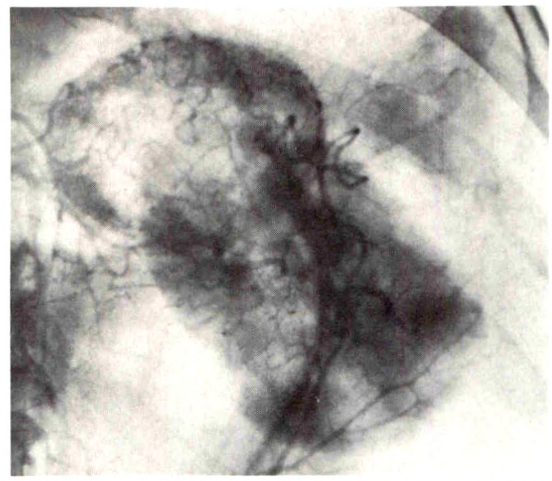

FIG. 9. "Pseudotumor" of the spleen caused by axial projection of fundus ventriculi.

Angiography in a case of spindle-cell sarcoma of the spleen was described by Morissette *et al.*[19] in 1973 (the same case also described by Baltaxe *et al.*[2] in 1972). Draping of the vessels around a mass and sparse vessels were found in this case, in which the splenic vein was open. Our case of spindle-cell sarcoma presents a more malignant angiographic pattern with neovascularity, extensive arteriovenous shunting, tumor staining, and occlusion of the splenic vein.

A case of reticulum-cell sarcoma of the stomach with invasion of the spleen was investigated by Rösch[23] in 1966, who found compressed and amputated arteries, minute newly formed vessels in places, and several irregular defects in the capillary phase. Gould *et al.*[10] in 1970 reported another case of reticulum-cell sarcoma with stretched vessels in the arterial phase and sharply demarcated radiolucent lesions in the capillary phase. Two of our reticulum-cell sarcomas displayed a somewhat different picture with more prominent neovascularity, encasement of arteries, and compression of veins, the splenic vein in 1 case being occluded. It seems justified to state that the diagnosis of malignant tumor can be established in these 2 cases, but not the nature of the lesion. In our third case of

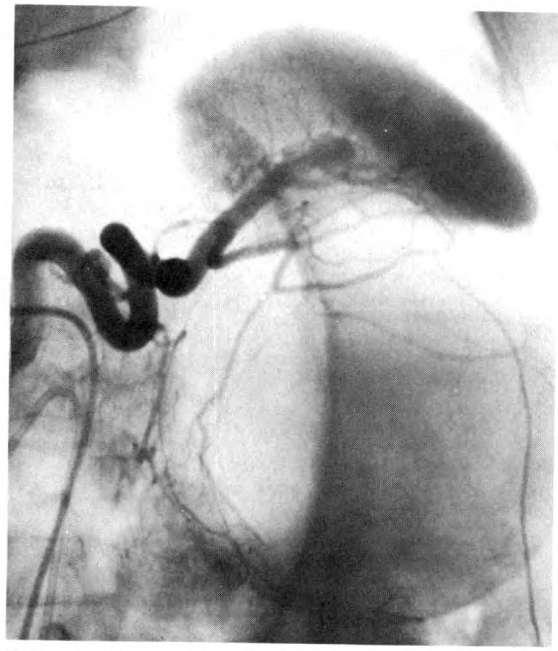

FIG. 10. Pseudocyst of the pancreas with smooth impression of the spleen.

reticulum-cell sarcoma no tumor vessels were demonstrable, the only finding being displacement of vessels.

METASTASES

Splenic metastases were reported to occur in 2–4 per cent in a large series of metastatic tumors.[11,18,25] Abrams[1] in 1952, however, reported an incidence of 9 per cent from neoplasms of epithelial origin and 45 per cent from those of nonepithelial origin. No further confirmation of this high incidence has been found in a review of the literature.

Kupic *et al.*[15] in 1967 and Gould *et al.*[10] in 1970 reported angiographically examined cases of metastatic melanomas showing neovascularity. Baltaxe *et al.*[2] in 1972 had 1 case with splenic metastases from a carcinoma of the stomach, where no neovasculature could be demonstrated.

Delorme *et al.*[5] in 1971 state that the most common primary tumors giving splenic metastases are localized in breast or ovary, or are chorionepitheliomas.

In our cases with metastases there are 2 renal carcinomas and 1 each of carcinoma of the urinary bladder and of the uterine body. From such a small number of cases no conclusion regarding the frequency can be drawn. In our 4 cases only 1 showed the metastases to be poorly vascularized. Whether or not our findings reflect the real frequency of hypervascularized metastases cannot be judged, because poorly vascularized lesions may be easily overlooked, especially if they are small.

SECONDARY GROWTH FROM ADJACENT ORGANS

The angiographic demonstration of invasion of the spleen is of importance for the surgeon when planning an operation. Baltaxe *et al.*[2] in 1972 reported invasion from the retroperitoneal space, angiographically appearing as a pseudocyst, but without sharp demarcation between the lesion and the surrounding parenchyma. Rösch[23] in 1966 reported a case of a retroperitoneal solid anaplastic carcinoma with splenic invasion. In the arterial phase there was an avascular region with displaced arteries, some of them being amputated. In the capillary phase irregularly demarcated avascular foci were seen. In our material 6 cases displayed encasement of arteries and in 3 of them tumor staining was evident. In 1 case arteriovenous shunting was also demonstrable. Parasitic blood supply to the primary tumor was demonstrable only in one of the richly vascularized renal carcinomas, a tumor type where such supply is common.

DIFFERENTIAL DIAGNOSIS

The findings at angiography of the spleen after trauma and in Hodgkin's disease have been reported from the Department of Diagnostic Radiology in Lund.[13,16,17] Therefore, we will not deal further with these differential diagnostic possibilities, but only make the comment that discrete intrasplenic hematomas possibly could be falsely diagnosed. The clinical history, however, should prevent that.

The normal spleen may sometimes present a puzzling appearance. However, in these cases there are no changes in the ar-

teries or veins. Primitive spleen (Fig. 8) should not give any diagnostic difficulty. Accessory spleens have been described by Kaude[14] in 1973. He states that the accessory spleen at angiography is well defined and homogeneously loaded with contrast medium during the capillary phase, and that the accessory splenic artery and vein often are demonstrable. The accessory spleen does not cause any vessel infiltration or occlusion, and neoplastic vasculature is absent. The fundus of the stomach (Fig. 9) should not be misinterpreted as a splenic tumor. A pancreatic cyst (Fig. 10) produces a smooth, sharply demarcated impression on the spleen and the appearance does not suggest a splenic cyst. Pseudocysts of the pancreas impressing the spleen have been reported by Kupic *et al.*[15] in 1967 and Delorme *et al.*[5] in 1971.

Osler's disease is rare (Fig. 11), and the large aneurysms in different organs should make the diagnosis of splenic malignancy unlikely.[3,12] Infarction (Fig. 12) is suggested by the atheromatous arterial changes, which, however, sometimes might be misinterpreted as encasement. The arterial collaterals and the parenchymatous changes in the capillary phase confirm the diagnosis.

Richly vascularized benign tumors, such as hamartomas, have been described by Wexler and Abrams[26] in 1964 and Rösch[23] in 1966. They reported numerous large tortuous vessels, and in 1 of the cases there was arteriovenous shunting. Diffuse enlargement of the spleen is seen in leukemia.[15,24] This condition as well as lymphoma and reticulosis is rarely a diagnostic problem when consideration is given to history, clinical findings, and roentgenologic evidence of involvement of other organs.

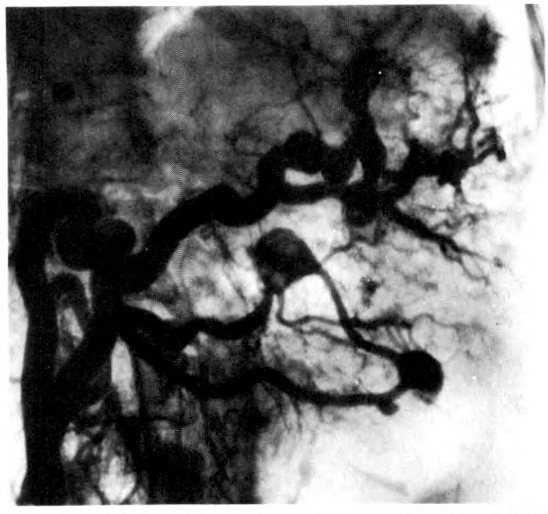

Fig. 11. Osler's disease in a 49 year old man. Wide left hepatic artery and small splenic artery with aneurysms.

Fig. 12. Infarctions of the spleen in woman, aged 69. Celiac angiography: Severe arteriosclerotic changes in main splenic artery and in hilum where there is an aneurysm. Collateral circulation from left gastric artery. Contrast defects in the periphery of the spleen. Histology: Severe arteriosclerosis and old infarctions of the spleen.

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REFERENCES

1. Abrams, H. Incidence of splenic metastasis of carcinoma. *Calif. Med.*, 1952, *76*, 281–282.
2. Baltaxe, H., Watson, R., and Levin, D. Angiographic appearance of splenic masses. *Angiology*, 1972, *23*, 316–327.
3. Borman, J. B., and Schiller, M. Osler's disease with multiple large vessel aneurysms. *Angiology*, 1969, *20*, 113–118.
4. Bron, K. M., and Hoffman, W. J. Preoperative diagnosis of splenic cysts. *A.M.A. Arch. Surg.*, 1971, *102*, 459–461.
5. Delorme, G., Tavernier, J., Grelet, P., Diard, F., Bellet, M., Fagola, M., and

LAFITTE, J. L'arteriographie des tumeurs spleniques. *Ann. radiol.*, 1971, *14*, 37–53.
6. DENNEEN, E. Hemorrhagic cyst of spleen. *Ann. Surg.*, 1942, *116*, 103–108.
7. EBAN, R. Splenic cyst with pre-operative diagnosis. *Brit. J. Radiol.*, 1959, *32*, 821–823.
8. EDSMAN, G. Malignant tumour of spleen diagnosed by lienal arteriography. *Acta radiol.*, 1954, *42*, 471–464.
9. ELLIS, H. Splenic cyst diagnosed by splenography. *Brit. J. Radiol.*, 1958, *31*, 331–332.
10. GOULD H. R., CLEMETT, A. R., and ROSSI, P. Radiologic diagnosis of splenic metastasis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 744–760.
11. HALPERN, M., TURNER, A. F., and CITRON, B. P. Angiodysplasias of abdominal viscera associated with hereditary hemorrhagic telangiectasia. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *102*, 783–789.
12. HERBUT, P. A., and GABRIEL, F. R. Secondary cancer of spleen. *Arch. Path.*, 1942, *33*, 917–921.
13. JONSSON, K., and LUNDERQUIST, A. Angiography of liver and spleen in Hodgkin's disease. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1974, *121*, 789–792.
14. KAUDE, J. Accessory spleens as demonstrated by celiac angiography. *Radiology*, 1973, *13*, 53–56.
15. KUPIC, E. A., MARSHALL, W. H., and ABRAMS, H. L. Splenic arterial patterns: angiographic analysis and review. *Invest. Radiol.*, 1967, *2*, 70–98.
16. LANDBERG, T., MÖLLER, T., BENGMARK, S., BÖRJESSON, B., OLSSON, A., CAVALLIN-STAHL, E., AHLSTRÖM, C. B., AKERMAN, M., JONSSON, K., LUNDERQUIST, A., and NAVERSTEN, Y. Staging laparotomy with splenectomy in Hodgkin's disease. *Acta chir. scandinav.*, 1974, *140*, 205–213.
17. LEPASOON, J., and OLIN, T. Angiographic diagnosis of splenic lesions following blunt abdominal trauma. *Acta radiol.* (Diag.), 1971, *11*, 257–273.
18. MILTON, G. W. Occurrence of secondary malignant disease in spleen. *M. J. Australia*, 1952, *2*, 736–740.
19. MORISSETTE, J., VIAMONTE, M., VIAMONTE, M., JR., and ROLFS, H. Primary spindle-cell sarcoma of spleen with angiographic demonstration. *Radiology*, 1973, *106*, 540–550.
20. POTTER, S., and WHOLEY, M. H. Splenic cysts: confirmation by selective visceral angiography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 418–420.
21. REUTER, S. R., and REDMAN, H. C. Gastrointestinal Angiography. W. B. Saunders Company, Philadelphia, 1972.
22. RIZK, G., TAYYARAH, K., and GHANDUR-MNAYMNEH, L. Angiographic changes in hydatid cysts of liver and spleen. *Radiology*, 1971, *99*, 303–309.
23. RÖSCH, J. Tumours of spleen: value of selective arteriography. *Clin. Radiol.*, 1966, *17*, 183–190.
24. SHANSER, J. D., MOSS, A. A., CLARK, R. E., and PALUBINSKAS, A. J. Angiographic evaluation of cystic lesions of spleen. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *119*, 166–174.
25. WARREN, S., and DAVIS, A. H. Studies on tumor metastases: metastases of carcinoma of spleen. *Am. J. Cancer*, 1934, *21*, 517–533.
26. WEXLER, L., and ABRAMS, H. L. Hamartoma of spleen: angiographic observations. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *92*, 1150–1155.
27. WICHT, C., FICHARDT, T., and RAUCH, R. Pneumoretroperitoneum en splenovenografie in die radiologiese Diagnose van Miltkiste. *South African M. J.*, 1953, *27*, 496–502.

VOL. 125, NO. 1

THE "STARRY NIGHT" SPLENIC ANGIOGRAM*

CONTRAST MATERIAL OPACIFICATION OF THE MALPIGHIAN BODY MARGINAL SINUS CIRCULATION IN SPLEEN TRAUMA

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ABSTRACT:

In traumatized human spleens, focal 0.3–1 cm. areas of contrast material staining appear to represent the malpighian marginal sinus circulation. This circulation, when seen, is static or very slow moving. Extravasation of blood and contrast material also may be present in the marginal sinus network. The identification of diffuse or localized small areas of contrast material in the splenic angiogram of the traumatized patient suggests splenic contusion, intrasplenic hematoma, or both. The splenic angiographic appearance described may be compared to the globular appearance of stars, as depicted by van Gogh in his painting "The Starry Night."

ANGIOGRAPHY of the traumatized spleen often reveals radiolucent defects compatible with intrasplenic or subcapsular hematomas. The splenic angiogram may also exhibit localized or diffuse, small, rounded shadows, which have been attributed to sinusoidal stasis or traumatic arteriovenous aneurysms.[2] The capillary phase of the study may be likened to van Gogh's representation of stars in "The Starry Night" (Fig. 1).*

In order to document the anatomical cause and study the significance of the shadows seen, the results of dog and human specimen studies are described. Four cases are presented which illustrate the splenic angiographic defects to be analyzed.

REPORT OF CASES

CASE 1. C.A. This 15 year old girl was struck by the bow of a motor boat while water skiing. Roentgenograms demonstrated fractures of the left 8th, 9th and 10th ribs. A falling hematocrit and the suggestion of splenic enlargement prompted splenic angiography. An area of avascularity and stretching of vessels was noted in the middle and upper aspect of the spleen (Fig. 2, *A* and *B*). Within this area were small, irregular collections of contrast material. The

FIG. 1. van Gogh's representation of stars in "The Starry Night" is reminiscent of stasis of contrast material in the malpighian body circulation of traumatized spleens.

* With permission of Museum of Modern Art, 11 West 53rd Street, New York, N. Y. 10019.

* Presented in part at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.

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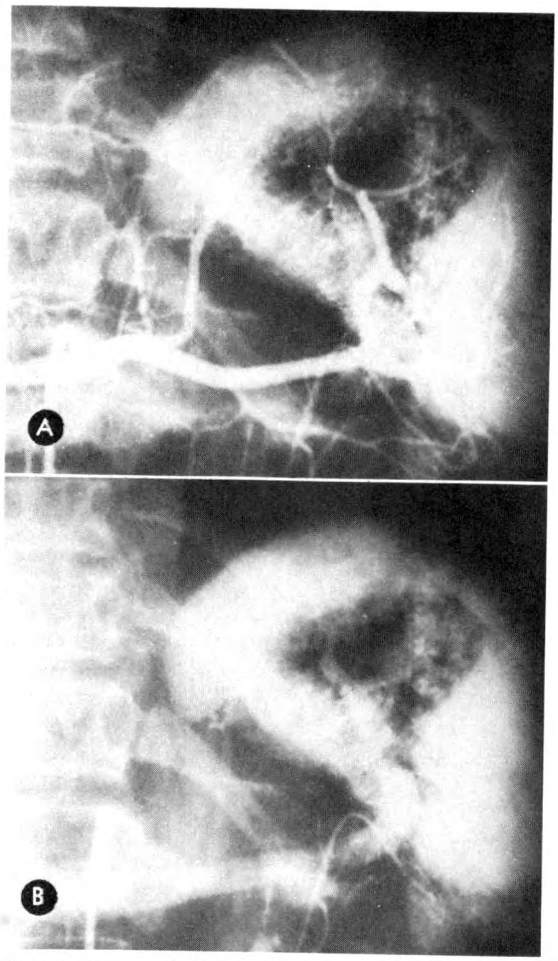

FIG. 2. Case I. (*A*) Arterial phase of angiogram in traumatized spleen exhibits 3 mm.-5 mm. mottled collections of contrast material in the upper pole seen best through the gastric air shadow. Stretched vessels and avascular area were due to intrasplenic hematoma. (*B*) The venous phase shows persistence of small, rounded shadows of opaque material in the injured upper pole. The lower pole of the spleen displays normal homogeneous staining.

surgical specimen revealed an upper pole hematoma 3 cm. in diameter, as well as smaller adjacent hemorrhages. The histologic sections showed smaller hemorrhages in the spleen, some of which were in the red pulp while others appeared to surround the malpighian corpuscles. The lower pole of the spleen showed no abnormality.

CASE II. V.M. This 19 year old female sustained a fractured acetabulum during an auto accident. During the first hospital day, the patient's symptoms included left shoulder and left upper quadrant pain. A celiac arteriogram exhibited diffuse focal mottling in the spleen, which persisted in the venous phase of the study (Fig. 3, *A* and *B*), but there was no definite avascular area. The moderate splenic enlargement and stasis of contrast material suggested splenic contusion. The patient's vital signs remained stable, and her symptoms cleared. Splenectomy was not performed. The patient recovered satisfactorily, having a normal pregnancy and delivery in the year after her accident.

CASE III. J.M. This 27 year old farmer fell across a bucket, injuring his left lower ribs and abdomen. The spleen was enlarged, but the patient was a member of a family with known congenital familial jaundice. Splenic angiography confirmed marked enlargement of the spleen. There was splaying of arteries in the midportion of the spleen as well as the upper pole (Fig. 4). Radiolucent defects in these areas suggested the presence of hematoma. Diffuse mottled blushing was evident also, and there was questionable extravasation of contrast material in the central portion of the spleen. At surgery, a large bleeding laceration was found in the midportion of the spleen, associated with a hematoma in the upper pole. The spleen weighed 672 grams and was markedly congested. Histologic examination showed areas of diffuse hemorrhage effacing the normal splenic architecture. Smaller hemorrhages which surrounded the malpighian bodies (Fig. 8*D*) were also found. The cause of the diffuse splenic congestion, aside from possible trauma, could not be determined. No spherocytes to suggest a coincident hemolytic anemia were identified in blood smears.

CASE IV. D.J. This 17 year old boy sustained severe trauma to the left upper quadrant and left shoulder during a fall in a wrestling match. Arteriography showed localized collections of contrast material as well as a radiolucent defect in the midportion of the spleen, compatible with a hematoma (Fig. 5). The spleen also appeared enlarged, probably due to generalized blood stasis. At surgery, the spleen was found to be lacerated centrally, and an intrasplenic hemorrhage approximately 1 cm. in width extended through the entire thickness of the spleen at the level of the laceration. Histologic sections confirmed the major hemorrhage as

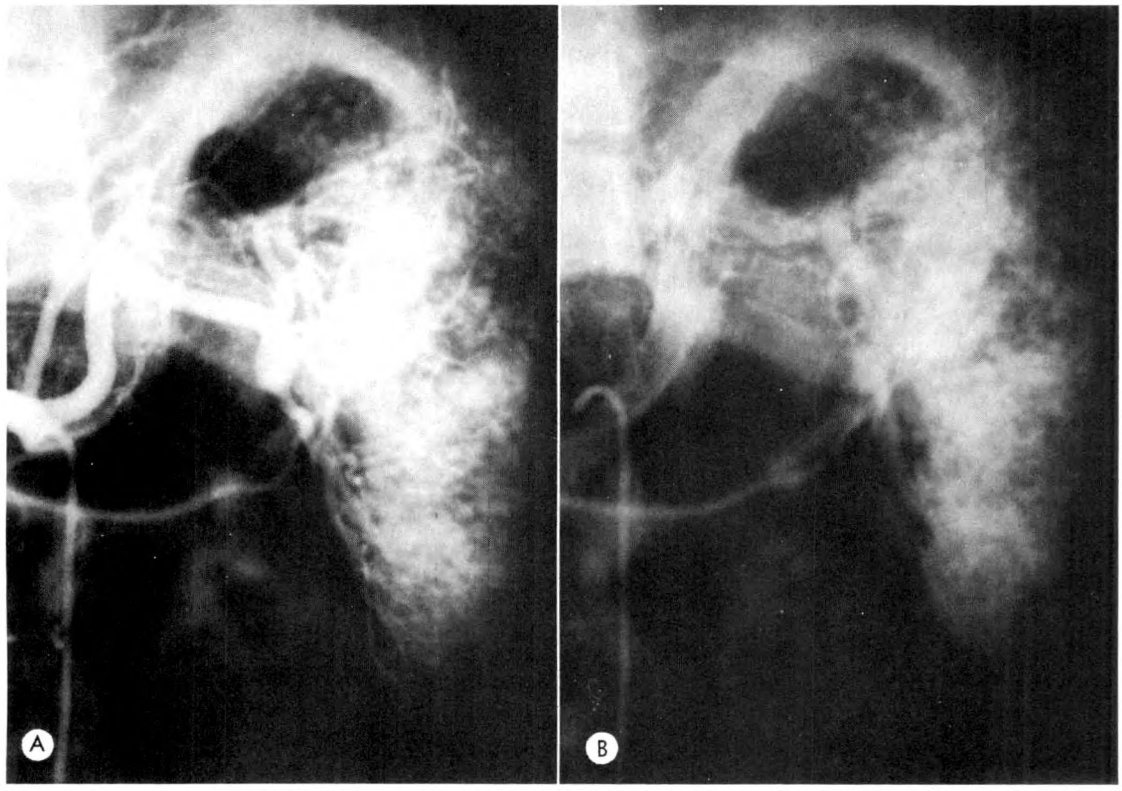

Fig. 3. Case 11. (*A*) Arterial phase of splenogram done for left upper quadrant trauma shows diffuse focal areas of contrast material staining. (*B*) Persistence of nodular staining suggested stasis of contrast material and blood in the malpighian body circulation secondary to spleen contusion. Surgery was not performed and patient made an uneventful recovery.

well as scattered smaller hemorrhages which were primarily in the red pulp.

VASCULAR ANATOMY OF THE SPLEEN

In both the dog and the human, arterial ramification occurs throughout the trabecular framework of the spleen. As the arteries leave the trabeculae, they are invested by strands of lymphoid tissue. The arteries also become intimately associated with the lymphoid or malpighian bodies. This relationship was recognized over a hundred years ago, as illustrated in a drawing from the first edition of *Gray's Anatomy*[5] (Fig. 6).

Of particular interest, and not well shown until recently by Ayers,[1] is the rich network of vessels that invest the malpighian body. This network, as delineated in dog spleen injections (Fig. 7, *A–E*) and human studies (Fig. 8, *A–D*), has been termed the marginal sinus and seems to create a junctional circulation between the arteries within the malpighian bodies (white pulp) and the sinusoidal channels in the red pulp.[6,12]

The network forms, with closely coiled vessels, a spheroidal vascular shell, the diameters of which may vary in the human spleen from 0.3 cm. to 1 cm. The arteries going through the malpighian bodies send vessels to the marginal sinus circulation. Some vessels in the marginal sinus appear to communicate directly with the red pulp sinusoids. The majority of vessels in the marginal sinus, however, make contact only with a surrounding syncytium of reticular cells through which red and white cells pass or are filtered. Janout and Weiss[7] have suggested that lymphatic vessels are intertwined with the arterioles of the malpighian bodies and marginal circulation, and this

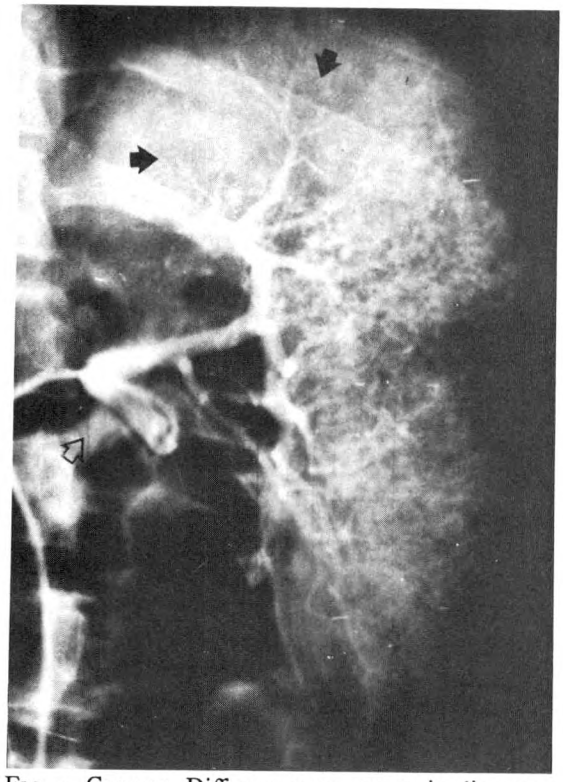

FIG. 4. Case III. Diffuse 3 mm.–5 mm. in diameter opacities are seen in the lower two-thirds of a spleen subjected to blunt trauma. An avascular area due to hematoma is present in the upper pole (black arrows). Early filling of the splenic vein is seen (open arrow), suggesting that some of the splenic contrast material is in sinusoids as well as the malpighian body circulation. The enlarged spleen was thought to be due to vascular stasis secondary to contusion.

has been shown histologically in lower species.

DISCUSSION

Spleen specimen injections, both in the dog and the human, suggest that small, rounded opacities seen in splenic angiograms of the cases reported represent contrast material in the marginal sinus circulation. As indicated in the histological sections from Cases I and III (Fig. 8*D*), disruption of the marginal sinus vessels secondary to trauma may permit local extravasation of contrast material. Most often, however, the mottling probably represents stasis of contrast material and blood in the marginal sinuses due to adjacent hematoma formation, spleen contusion, or hypotension. The possibility that contrast material pools or leaks out of fragmented sinusoids also exists, as suggested by Brindle.[2]

A survey of over 50 celiac axis arteriograms done in patients with no history of trauma did not demonstrate the mottled opacification described. In the normal human spleen, contrast material appears to flow rapidly into the sinusoidal circulation, producing homogeneous opacification of the organ. This can also be seen in Case I, wherein homogeneous staining of the normal lower pole of the spleen occurred, while the traumatized upper pole exhibited focal collections of contrast material.

Brindle[2] has shown convincing evidence

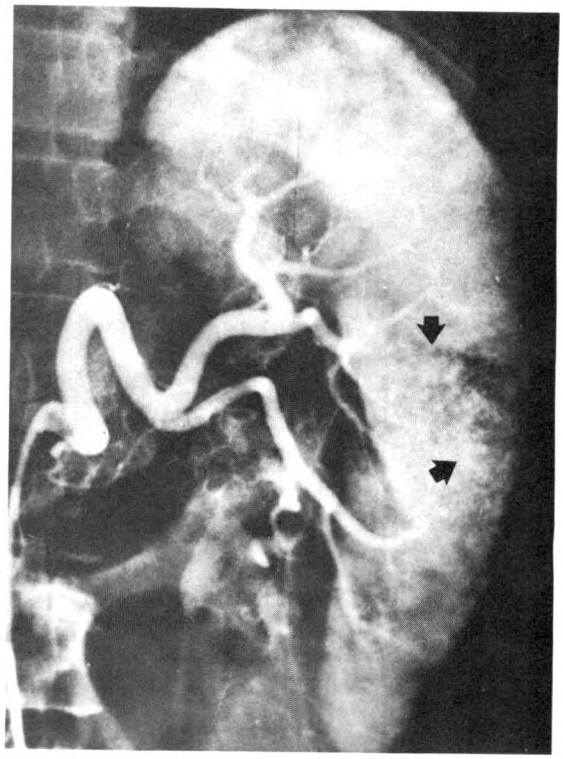

FIG. 5. Case IV. The arrows denote localized collections of contrast material in the central aspect of the spleen. A radiolucent defect in this area coincided with a splenic laceration and hematoma. The upper pole of the spleen presents broad areas of mottling, which are probably a variant in the normal splenic angiogram due to prominent trabeculae in the organ.

Fig. 325.—The Malpighian Corpuscles, and their Relation with the Splenic Artery and its Branches.

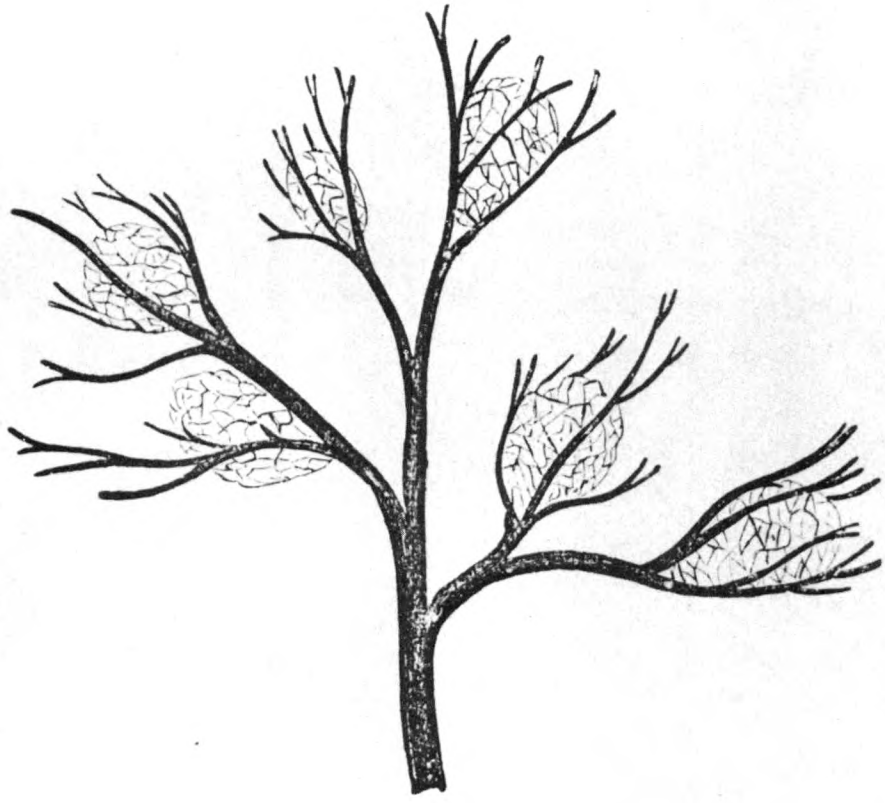

Fig. 6. An artist's representation of the malpighian bodies from the first edition of *Gray's Anatomy* (1858) emphasizes the vascular networks in the bodies and their relationship to the splenic arterial branches.

that arteriovenous aneurysms occur in the traumatized spleen. These have relatively sharp margins, however, resembling congenital aneurysm formation[9] or the aneurysms associated with periarteritis nodosa,[3] which can occur in the spleen. The mottled shadows of apparent stasis of contrast material in the malpighian circulation have less distinct margins than those seen in aneurysms.

Although the histological studies of our cases did not show definite traumatic arteriovenous fistulae within the malpighian bodies, it is reasonable to assume that they may occur. Abnormal shunting through this circulation could be responsible for the early venous filling seen in Case III in the absence of arteriographic evidence of traumatic aneurysm or fistula formation.

It is of interest that primary or secondary malignant involvement of the spleen will also produce a mottled appearance in the splenic angiogram. Rösch[10] has shown several examples of focal blushing of metastases in the spleen. A "cerebriform" staining of the spleen infiltrated by Hodgkin's disease has been described by Markovits *et al.*[8] A series of 33 cases of Hodgkin's disease[4] studied with splenic angiography exhibited a mottled appearance in 10 patients with histologically proved tumor in the spleen. In 12 patients in the same series, mottling was present and no tumor was found. The mottled areas in this case series appeared to be of greater caliber than those seen in the traumatized spleens we have observed. The speculation, however, that marginal sinus circulation has become the

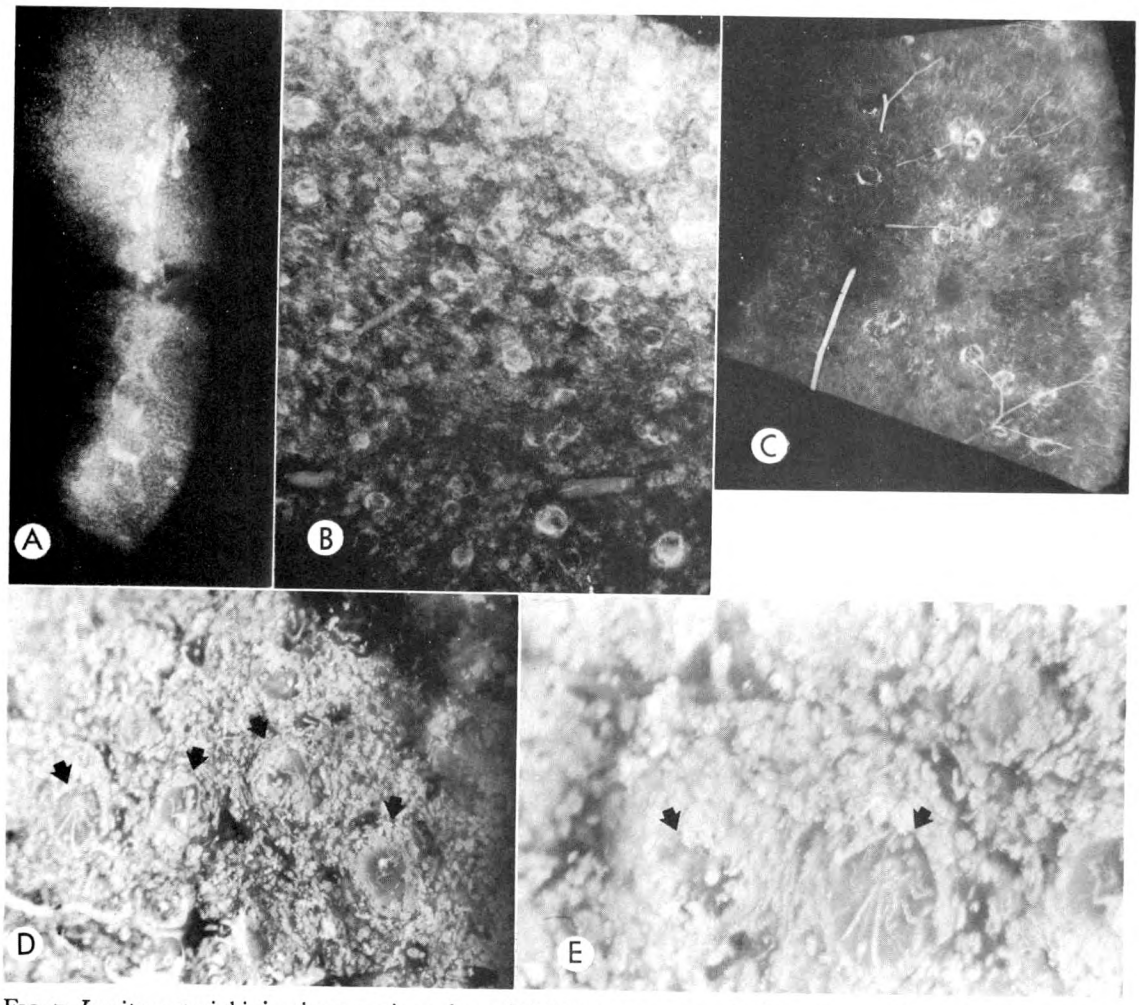

FIG. 7. *In vitro* arterial injections, canine spleen. (*A*) Roentgenogram of spleen shows diffuse ringlike shadows 3–4 mm. in diameter. (*B*) Microradiograph (tissue specimen 3 mm. thick) shows spheroidal nature of the vascular networks associated with malpighian bodies, confirmed histologically. (*C*) A thinner section (1 mm.) brings out relationship of small arteries with vascular networks of malpighian bodies. (*D*) Corrosion cast* of another dog spleen has been photographed with a dissecting microscope at 15× magnification. Arrows show angioarchitecture of 4 malpighian bodies. Intervening cast material is in splenic sinusoids. (*E*) A 25× magnification of two malpighian marginal sinus vascular networks. The spheroidal shell-like distribution of vessels in the malpighian body (left arrow) is seen. The arrow on the right denotes a malpighian body which has been cut through its center. Spoke-like vessels from a central artery appear to radiate to the marginal circulation.

* Prepared with Microfil, Canton Biomedical Products, Inc., P. O. Box 2017, Boulder, Col. 80302.

core blood supply for tumor deposits in the spleen is intriguing. Specimen injection and roentgenography might well shed further light on this question. It is suspected that in normal splenic tissue, where broad areas of mottling are present, the trabeculae are prominent and have produced compartmentalization of the sinusoids (Fig. 4).

The teleological aspects of the splenic marginal sinus circulation are of interest. The angioarchitecture of these structures is reminiscent of the renal glomerulus. Weiss[11] has suggested that both vascular units, *i.e.*, the marginal sinus and the glomerulus, serve as "plasma-skimming structures." Since plasma flows at a faster rate in the peripheral portion of a blood vessel lumen, it is more likely to be skimmed off in the

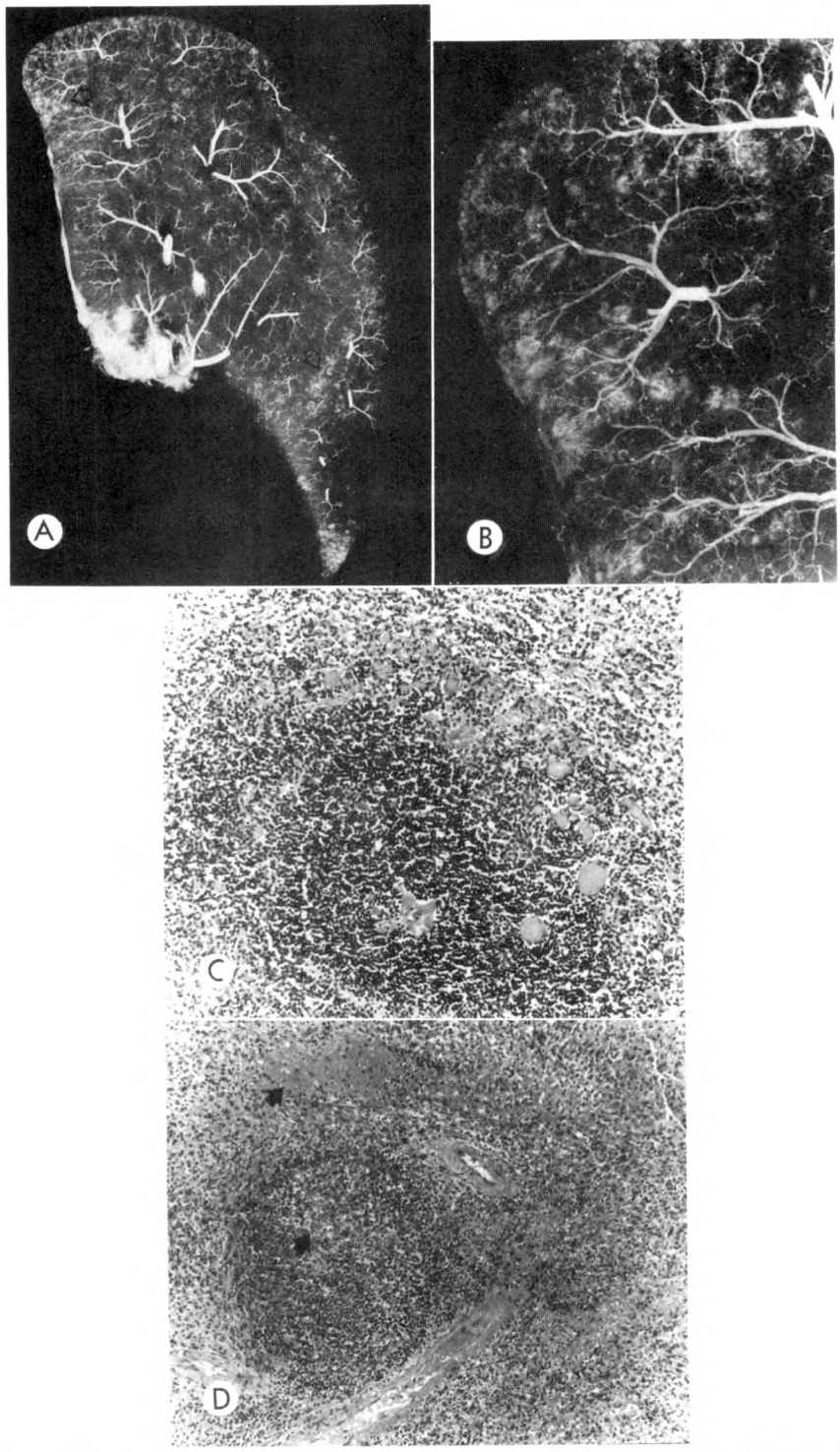

FIG. 8. (*A*) Microradiograph of a human *in vitro* spleen injection exhibits contrast material in marginal sinuses associated with malpighian corpuscles. Arrows denote corpuscles that are well outlined in upper and lower poles. The specimen is 1 cm. thick. (*B*) Close up (3× magnification) of specimen brings out mottled appearance of contrast material in marginal sinuses to good advantage and relationship of malpighian body—marginal sinus circulation to intrasplenic arteries. (*C*) *A* 10 μ histological section from same spleen (100× magnification) shows barium in a marginal sinus (homogeneous gray shadows) around dark staining cells which constitute the malpighian body. (*D*) *A* 10 μ section (100× magnification) from Case III shows extravasation of blood around and in a malpighian body (arrows). Marginal sinus disruption appears to be the cause for the hemorrhage.

multiples of right angle vessels in the marginal sinus and returned to splenic veins or the lymph circulation.

The intimal surface in the marginal vessels is also very irregular.[11] This surface, which is amplified in a network of vessels (the marginal sinus), may impose mechanical trauma on red cells, making the older cells more fragile and increasing the likelihood of their being destroyed by macrophages in the splenic sinusoids.

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REFERENCES

1. AYERS, A. B. Microvasculature of spleen. *Clin. Radiol.*, in press.
2. BRINDLE, M. J. Arteriography and minor splenic injury. *Clin. Radiol.*, 1972, *23*, 174–180.
3. BRON, K. M., STRATT, C. A., and SHAPIRO, A. P. Diagnostic value of angiographic observations in polyarteritis nodosa. *A.M.A. Arch. Int. Med.*, 1965, *116*, 450–454.
4. CASTELLINO, R. A., SILVERMAN, J. F., GLATSTEIN, E., BLANK, N., and WEXLER, L. Splenic arteriography in Hodgkin's disease: roentgenologic-pathologic study of 33 consecutive untreated patients. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *114*, 574–582.
5. Gray's Anatomy. First edition, 1858, p. 623.
6. GREEP, R. O., and WEISS, L. Histology. McGraw-Hill Book Company, Inc., New York, 1973.
7. JANOUT, V., and WEISS, L. Deep splenic lymphatics in marmot: electron microscopic study. *Anatom. Rec.*, 1972, *172*, 197–219.
8. MARKOVITS, P., GASQUET, C., and PARMENTIER, C. La rate Hodgkinienne: étude par artériographie coeliaque sélective (à propos d'une observation). *J. de radiol., d'électrol. et de méd. nucléaire*, 1968, *49*, 297–299.
9. MURRAY, J. M., THAL, A. P., and GREENSPAN, R. H. Splenic arteriovenous fistulas as cause of portal hypertension. *Am. J. Med.*, 1960, *29*, 849–856.
10. RÖSCH, J. Tumors of spleen: value of selective arteriography. *Clin. Radiol.*, 1966, *17*, 183–190.
11. WEISS, L. Structure of fine splenic arterial vessels in relation to hemoconcentration and red cell destruction. *Am. J. Anat.*, 1962, *111*, 131–179.
12. WEISS, L. Structure of normal spleen. *Seminars Hematol.*, 1965, *2*, 205–228.

VOL. 125, No. 1

EXPERIMENTAL USE OF PROSTAGLANDIN E_1 IN NONOCCLUSIVE MESENTERIC ISCHEMIA*

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ABSTRACT:

Nonocclusive mesenteric ischemia was produced in dogs by intraarterial infusion of digoxin and by hemorrhage of ⅓ of total blood volume. Both methods produced a substantial drop in the superior mesenteric arterial blood flow rate. In each case, a superior mesenteric arterial infusion of 0.1 μg./kg. per minute of prostaglandin E_1 allowed the blood flow rate to increase to above the control level without significantly altering heart rate or systemic arterial pressure. The improved arterial blood flow rates were accompanied by a reversal of the diffuse mesenteric vasoconstriction that was seen in the ischemic state. These findings were demonstrated by superior mesenteric arteriography performed prior to and following the production of mesenteric ischemia and again following the infusion of prostaglandin E_1.

MESENTERIC infarction is a significant cause of death in elderly patients. Acute mesenteric ischemia from all causes has been reported with increasing frequency during recent years.[5,20,21,32] A variety of factors other than occlusion of the mesenteric vasculature contributes to intestinal ischemia.[4–6,17,21] Over 500 cases of ischemic necrosis of the bowel in the absence of arterial or venous obstruction have been reported since 1943.[13] The relative frequency of this form of ischemic necrosis has now risen to over 50 per cent of all reported cases.[4,21,34]

The reported cases of nonocclusive mesenteric ischemia have most often occurred against a background of cardio-circulatory compromise such as following acute myocardial infarction, severe congestive failure, cardiac arrhythmias, dehydration and hemorrhagic shock.[7,13,17,21,22] It is known that digitalis preparations are potent vasoconstrictors in the mesenteric circulation,[9,14,25] and clinical and experimental studies have indicated that digitalis preparations are implicated in worsening if not precipitating nonocclusive mesenteric ischemia.[13,15,18,21,22] A theoretical chain of events has been proposed to explain the pathophysiology of nonocclusive mesenteric ischemia.[16] It is felt that once the ischemic process begins, a vicious cycle of events progresses predictably to complete bowel death unless the mesenteric vasospasm is reversed.[5]

Since no occlusion exists in the major vessels, this form of the disease is not readily amenable to surgical correction. At present, the mortality rate for nonocclusive mesenteric ischemia approaches 100 per cent.[21] There has been no effective mode of therapy in the past, but some recent experimental and clinical reports have indicated that the local intraarterial infusion of vasodilators may be effective in reversing mesenteric vasospasm and improving the prognosis.[1,5–7,27,31] The vasodilators which have been used for this purpose include phenoxybenzamine, tolazoline and papaverine.

All observers agree that early diagnosis is essential if improved survival rates are to be achieved. It is therefore important to

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.
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Supported in part by the George Alfred Cook Memorial Fund.
Supported in part by the Upjohn Company, Kalamazoo, Michigan.

perform early angiography on any patient suspected of suffering from acute mesenteric ischemia. Angiography can determine if emergency surgery is necessary to alleviate a major vascular occlusion and it is the only currently available method for establishing the diagnosis of nonocclusive mesenteric ischemia.[1,4–6,8,32,33] Once the clinical and angiographic diagnosis of nonocclusive mesenteric ischemia has been made, it is possible to begin an immediate therapeutic infusion of a vasodilating substance directly into the involved mesenteric arterial bed through the angiographic catheter.

Since, in the clinical situation, the patients most often present as elderly, acutely ill individuals in a setting of shock or cardiac dysfunction, the ideal vasodilating agent would be one which is potent and rapid acting but which does not further compromise systemic hemodynamics. Clinical and experimental studies have shown that prostaglandin E_1, with its rapid removal from the circulation by the liver and lungs, is such an agent.[10,11,23,26,29,30]

In the present study an attempt was made to produce an accurate experimental model of nonocclusive mesenteric ischemia. Hemorrhagic shock and digoxin were used alternately to produce mesenteric vasoconstriction. Prostaglandin E_1 was then physiologically and angiographically evaluated as to its ability to reverse the mesenteric vasospasm without significantly altering systemic hemodynamics.

MATERIAL AND METHOD

Ten mongrel dogs ranging in weight from 18 to 25 kg. were anesthetized with intravenous sodium pentobarbital. Under fluoroscopic control, a No. 7 Fr. catheter was introduced through the right femoral artery, and its curved tip was placed in the origin of the superior mesenteric artery proximal to its first branch. An open-ended No. 8 Fr. catheter was tied in the left femoral artery and connected to a pressure transducer. A multichannel recorder was used to continuously record systemic arterial pressure and heart rate (Fig. 1). When angiography was not in progress, an infusion pump was used to constantly deliver normal saline or normal saline plus a drug through the angiographic catheter at a rate of 1.25 cc. per minute. The animal was allowed to breathe room air and stabilize for a minimum of 15 minutes prior to initiating each experiment. In addition, at least 15 minutes stabilization was allowed between each experimental period. Data were collected for each of the 7 experimental periods:

1. *Control:* following the infusion of 1.25 cc. of normal saline per minute into the superior mesenteric artery for a minimum of 15 minutes.
2. *Digoxin:* following infusion of 15 μg./min. digoxin in 1.25 cc. saline per minute into the superior mesenteric artery for a minimum of 15 minutes.
3. *Digoxin and Prostaglandin E_1:* following the superior mesenteric arterial infusion of 0.1 μg./kg. per min. of PGE_1 with 15 μg. digoxin in 1.25 cc. saline per minute for 5 minutes (digoxin alone was infused for an additional 15 minute stabilization period prior to beginning test period 3).
4. *Post Digoxin Control:* following the cessation of the digoxin+PGE_1 infusion and subsequent intraarterial infusion of 1.25 cc. normal saline per minute for a minimum of 15 minutes.
5. *Post Hemorrhage:* a minimum of 15 minutes following the completion of a hemorrhage of 2.5 per cent of body weight (approximately $\frac{1}{3}$ of the circulating blood volume), while continuing the infusion of saline into the superior mesenteric artery at 1.25 cc. per minute.
6. *Post Hemorrhage+Prostaglandin E_1:* following the superior mesenteric arterial infusion of 0.1 μg./kg. per min. of PGE_1 in 1.25 cc. of saline per minute for 5 minutes beginning no sooner than 30 minutes after completion of the above hemorrhage (period 5).
7. *Post Hemorrhage Control:* following the intraarterial infusion of 1.25 cc. normal saline per minute for a mini-

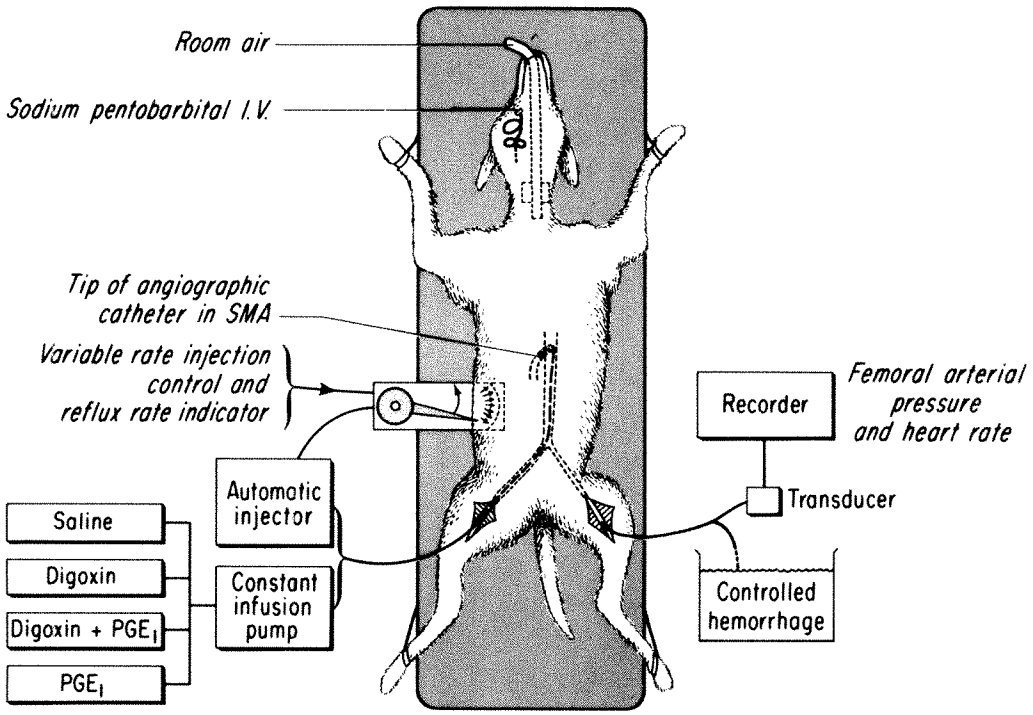

FIG. 1. Experimental preparation.

mum of 15 minutes subsequent to the cessation of the above PGE_1 infusion (period 6).

Superior mesenteric angiography was carried out at the end of each of the above experimental periods with the exception of periods number 4 and 7. For all arteriographic studies, the sequential filming was carried out at 4/second for 3 seconds, 0 for 3 seconds and then 1 for 12 seconds. The approximate superior mesenteric arterial blood flow rate was determined by a modified angiographic reflux method.[2,3,19] An injector was used which automatically and continuously increased the rate of injection of contrast medium during the entire injection. A synchronous rate indicator was projected on the angiographic films so that the rate of injection at the time of each exposure during the arterial phase could be determined. When necessary, the indicated diastolic and systolic reflux flow rates were averaged (Fig. 2). Each angiographic examination in a given animal used identical positioning, roentgenographic factors, film sequencing and total volume of contrast material injected. In addition to determining the approximate superior mesenteric arterial flow rates by reflux, the 5 angiographic studies were also compared for evidence of mesenteric vasospasm, *i.e.*, slowing of flow with decreased rate of filling of peripheral branches, narrowing and abnormal tapering of major intestinal branches, spasm and impaired filling of intestinal arcades, and decreased bowel wall staining.[6] Just prior to each angiographic examination and at the end of test periods 4 and 7, multiple determinations of systemic arterial pressure and heart rate were recorded.

RESULTS AND DISCUSSION

A sign test[28] was applied to the reflux arterial blood flow rate data and it was found that the superior mesenteric arterial blood flow rate (SMAQ) decreased significantly from control levels following both intraarterial digoxin infusion and hemorrhage ($p < .01$). The average decrease in SMAQ following intraarterial digoxin was

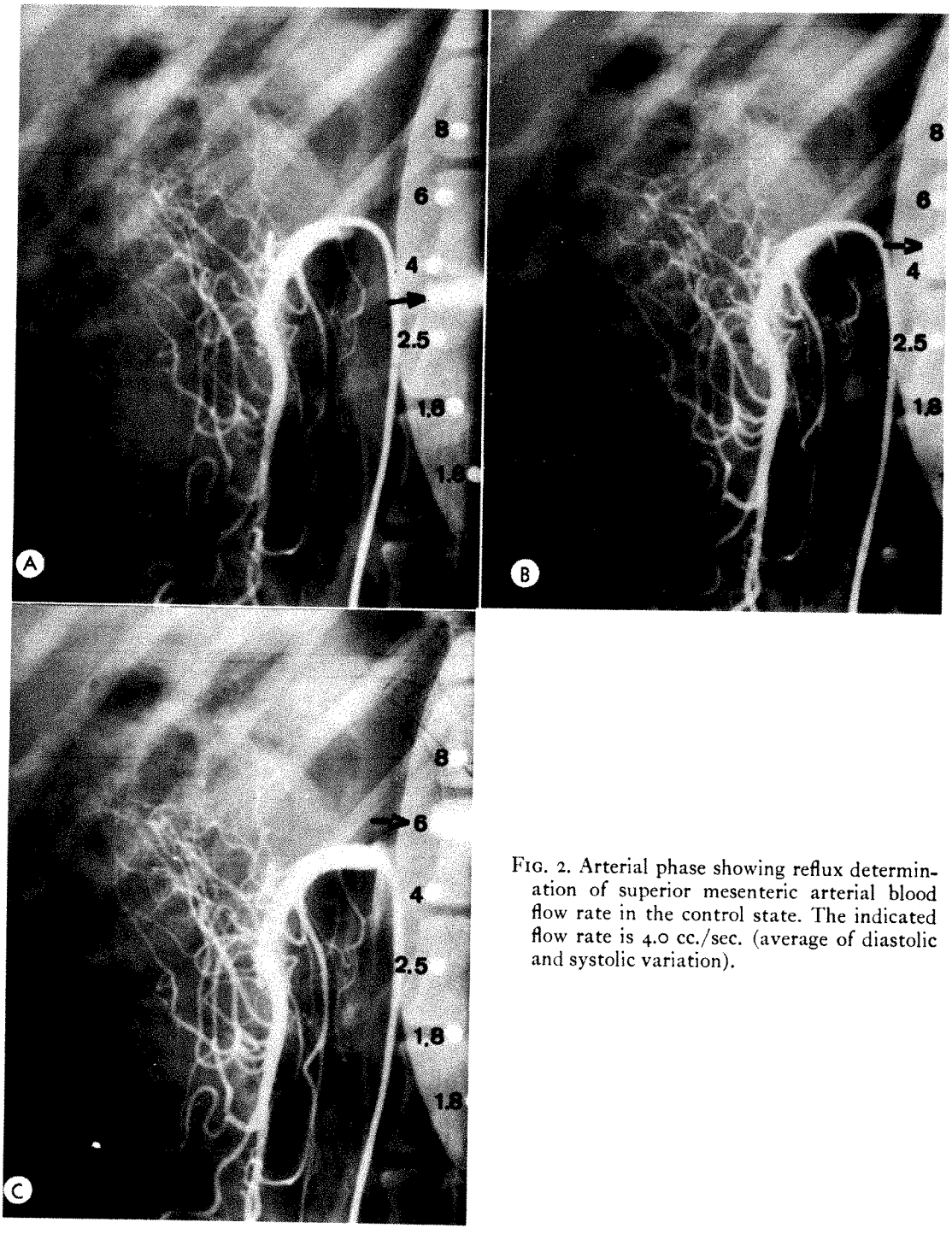

FIG. 2. Arterial phase showing reflux determination of superior mesenteric arterial blood flow rate in the control state. The indicated flow rate is 4.0 cc./sec. (average of diastolic and systolic variation).

26 per cent and the average decrease in SMAQ following hemorrhage was 45 per cent (Fig. 3). Similarly, the prostaglandin E_1 infusions significantly increased SMAQ from the levels seen following the intraarterial digoxin and hemorrhage ($p < 0.1$). Prostaglandin E_1 produced an average increase in SMAQ of 205 per cent over the rates seen following intraarterial digoxin; prostaglandin E_1 also produced an average

increase in SMAQ of 228 per cent over the rates seen following hemorrhage (Fig. 3).

The reduction in superior mesenteric arterial blood flow rates produced by digoxin or hemorrhage was accompanied by narrowing of major arterial branches and decreased filling of peripheral branches. These angiographic changes were reversed by the prostaglandin E_1 infusions. In addition, the superior mesenteric arterial blood flow rate also returned to equal or above the control rates (Fig. 4; and 5). In the late arterial and early venous phases of the angiograms it was found that the peripheral arterial arcades were not well filled and that the bowel wall stain was lost when the superior mesenteric arterial blood flow rates fell due to experimentally induced mesenteric vasospasm. When the vasospasm was reversed by prostaglandin E_1 allowing the mesenteric arterial blood flow to return to normal, bowel wall staining reappeared and filling of peripheral arterial branches improved (Fig. 6).

The ranked means for systemic arterial blood pressure and heart rate data were compared for each treatment period at the 0.05 significance level using Scheffe Contrasts.[24] The prostaglandin E_1 infusion caused no statistically significant rise in heart rate when given following hemorrhage. While the prostaglandin E_1 did produce a statistically significant drop in systemic arterial blood pressure following both hemorrhage and digoxin infusions, the degree of change was small and did not persist after the prostaglandin E_1 was discontinued (Table 1). A significant increase in the heart rate was observed in association with the prostaglandin E_1 infusion following intraarterial digoxin, but again the effect did not persist after the prostaglandin E_1 infusion was discontinued (Table 1).

The data from a single representative dog are illustrated in Figure 7. The angiograms illustrated in Figures 2, 4 and 5 were also from this animal.

Although no data are presented for the superior mesenteric arterial flow rate for test period 4 (post digoxin+PGE_1 control)

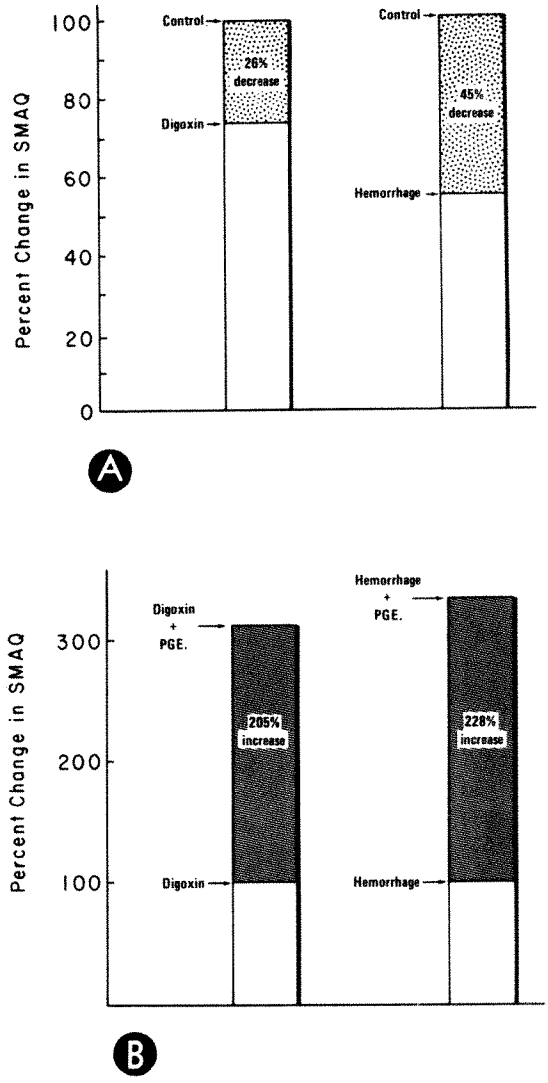

Fig. 3. (*A*) Average decrease from the control rate of superior mesenteric arterial blood flow following digoxin infusion or hemorrhage. (*B*) Average increase in the superior mesenteric arterial blood flow rate when PGE_1 was given following digoxin infusion or hemorrhage.

and test period 7 (post hemorrhage+PGE_1 control) it can be assumed that the mesenteric arterial flow rates returned to their pre-digitalis and pre-prostaglandin E_1 levels. This assumption is supported in part by the fact that the heart rate and systemic blood pressure returned to their original levels. The assumption was further confirmed in our laboratory by repeating the same series of experiments on two more dogs while

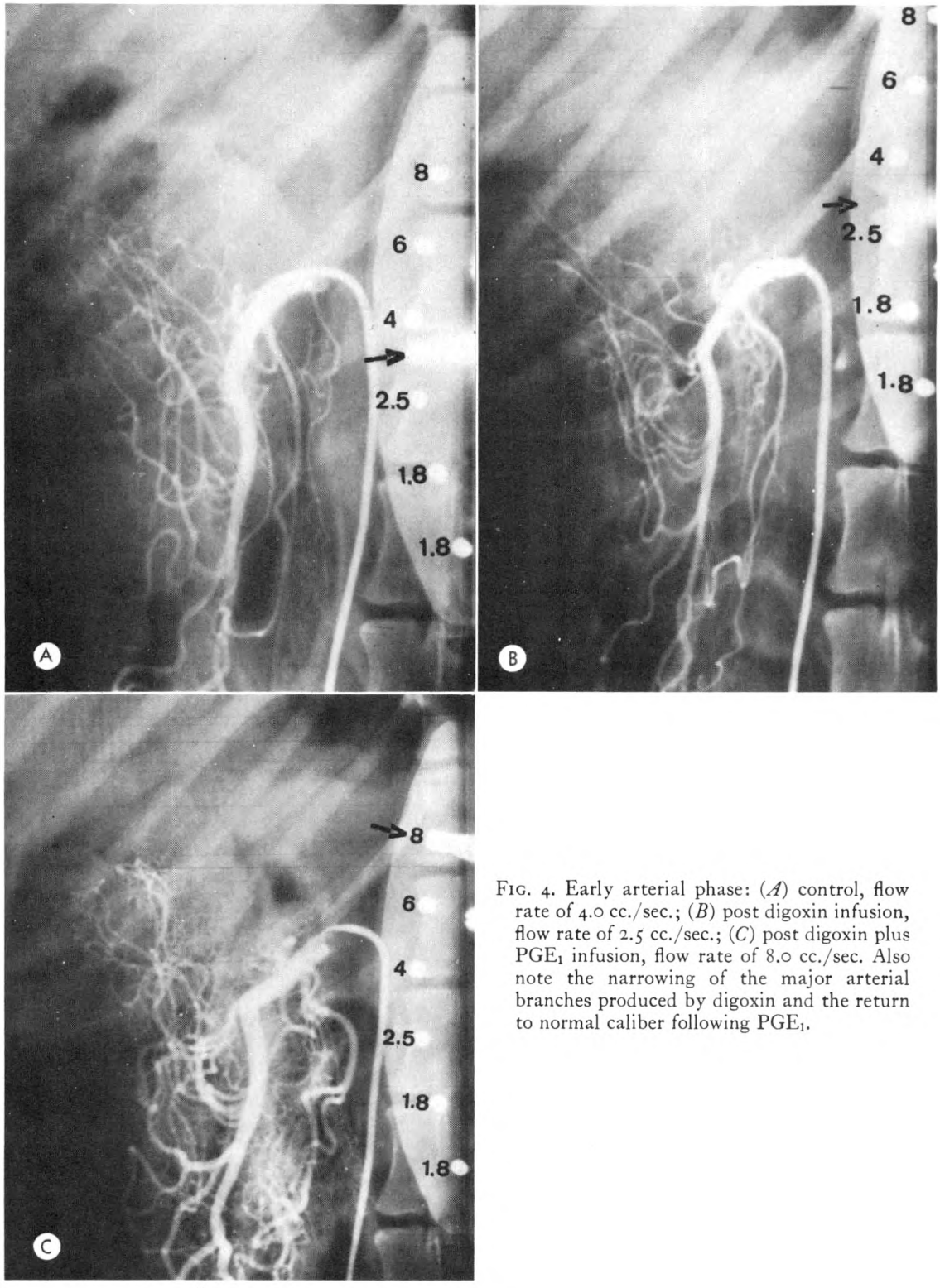

FIG. 4. Early arterial phase: (*A*) control, flow rate of 4.0 cc./sec.; (*B*) post digoxin infusion, flow rate of 2.5 cc./sec.; (*C*) post digoxin plus PGE_1 infusion, flow rate of 8.0 cc./sec. Also note the narrowing of the major arterial branches produced by digoxin and the return to normal caliber following PGE_1.

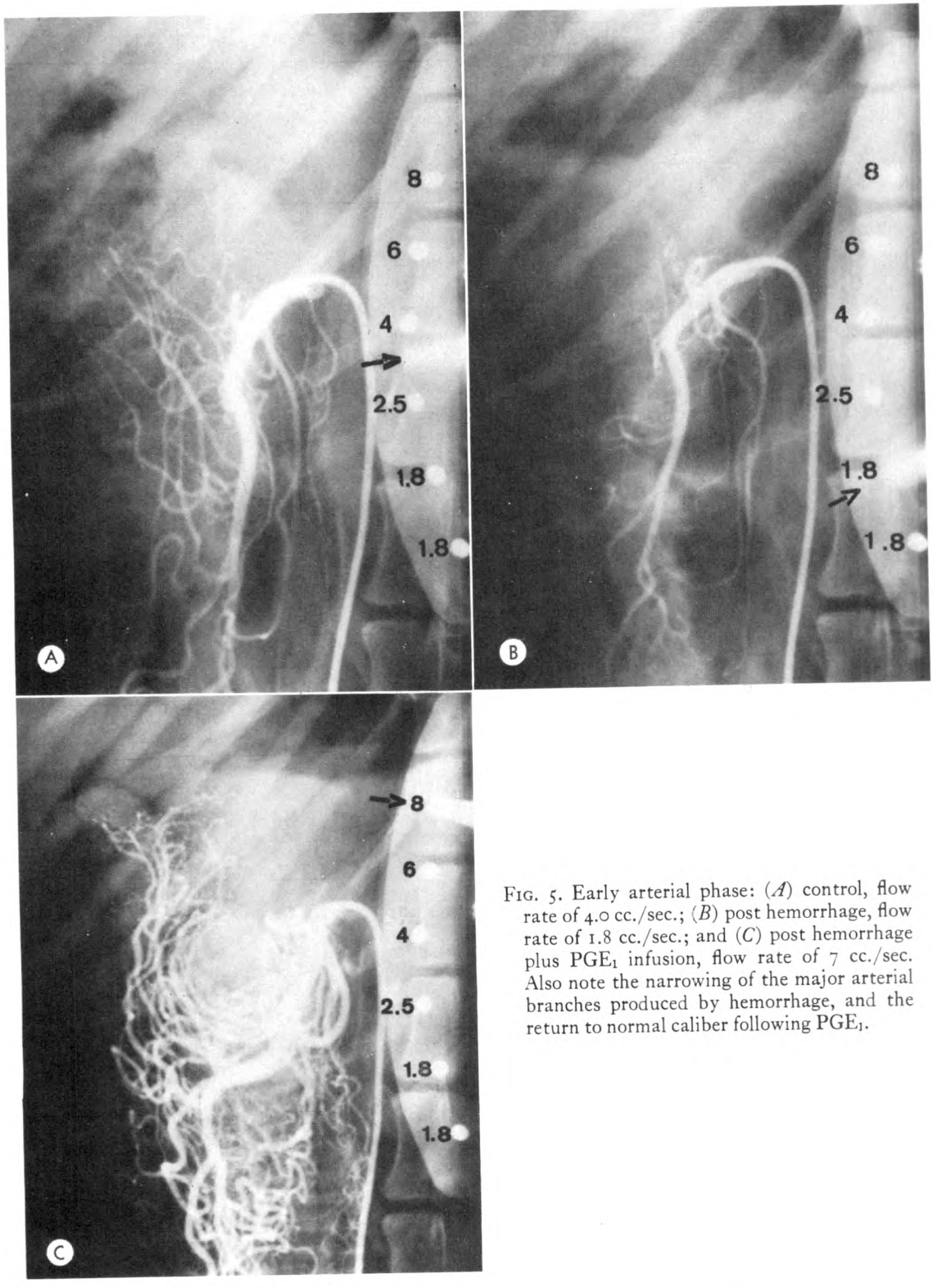

FIG. 5. Early arterial phase: (*A*) control, flow rate of 4.0 cc./sec.; (*B*) post hemorrhage, flow rate of 1.8 cc./sec.; and (*C*) post hemorrhage plus PGE_1 infusion, flow rate of 7 cc./sec. Also note the narrowing of the major arterial branches produced by hemorrhage, and the return to normal caliber following PGE_1.

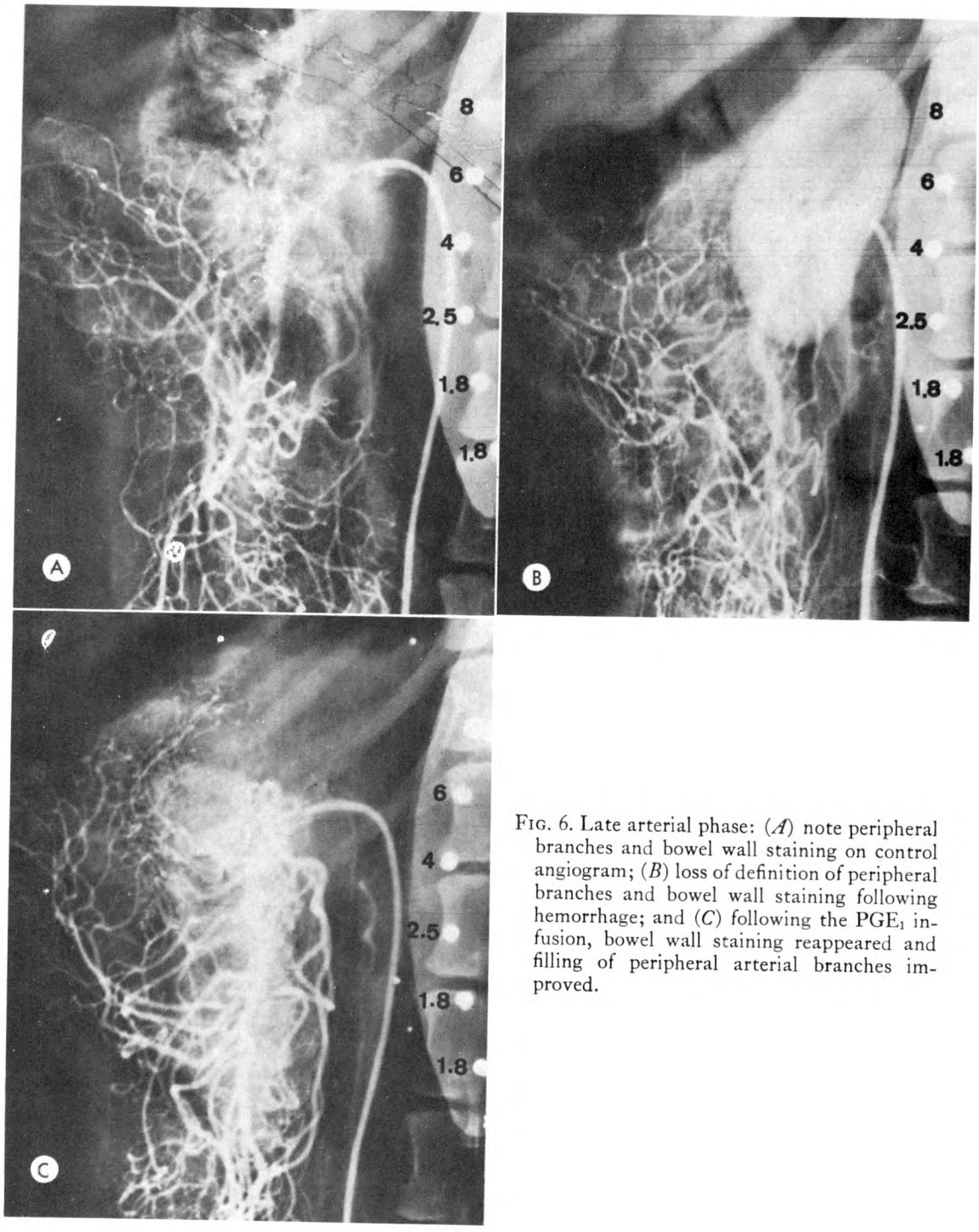

FIG. 6. Late arterial phase: (*A*) note peripheral branches and bowel wall staining on control angiogram; (*B*) loss of definition of peripheral branches and bowel wall staining following hemorrhage; and (*C*) following the PGE_1 infusion, bowel wall staining reappeared and filling of peripheral arterial branches improved.

TABLE I

MEAN VALUES FOR SYSTEMIC ARTERIAL BLOOD PRESSURE AND HEART RATE RANKED IN ASCENDING ORDER

Mean Systemic Arterial Pressure ($\overline{SAP}$)

(Ranked means (S—Method) on treatments at the 0.05 significance level)

Hemorrhage + PGE_1	15 minutes Post Hemorrhage	Post Hemorrhage Control 15 minutes after PGE_1 stopped	Post Digoxin + PGE_1	Initial Control	15 minutes Post Cessation of Digoxin & PGE_1 (prior to Hemorrhage)	Digoxin Infusion
109.9	118.0	118.0	134.8	138.8	140.7	144.5

not statistically different

not statistically different

A

Mean Heart Rate

(Ranked means (S—Method) on treatment at the 0.05 significance level)

Initial Control	15 minutes Post Cessation of PGE_1 & Digoxin (prior to Hemorrhage)	Digoxin Infusion	Digoxin + PGE_1	15 minutes Post Hemorrhage	Post Hemorrhage Control 15 minutes post PGE_1 stopped	Hemorrhage + PGE_1
148	150	156	180	184	184	187

not statistically different

not statistically different

not statistically different

B

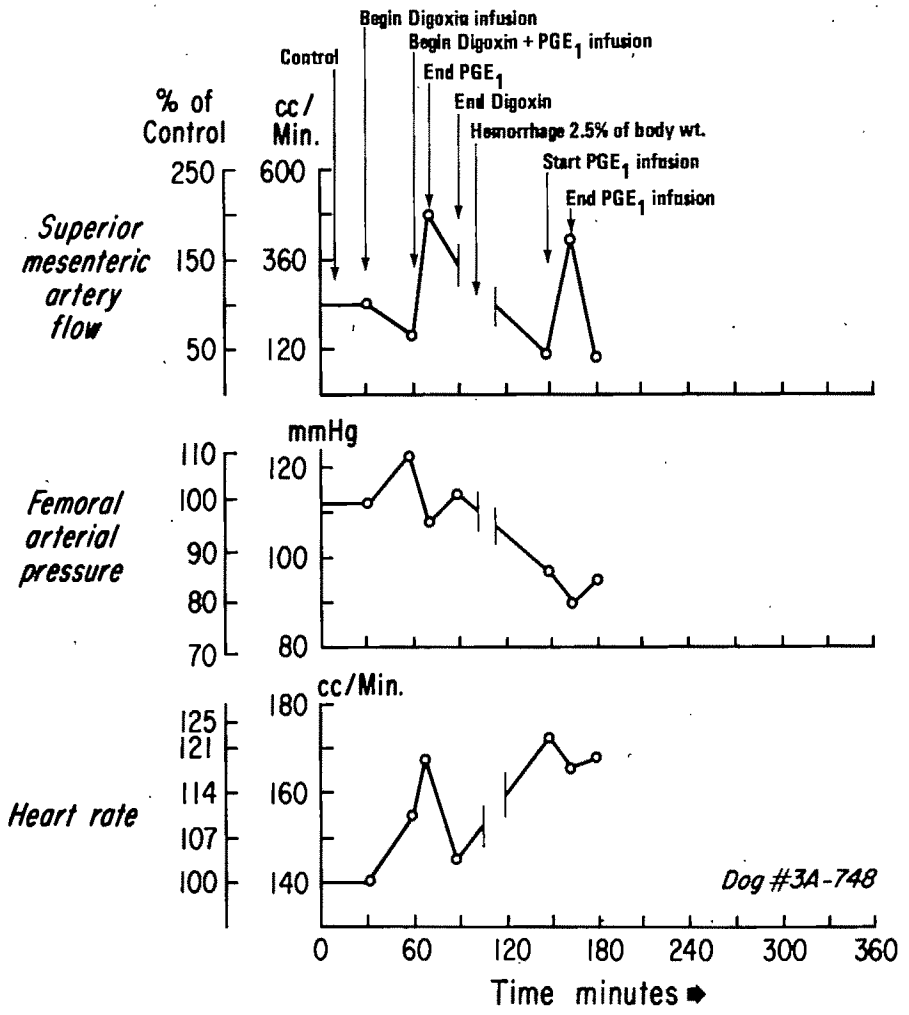

FIG. 7. Effect of digoxin, digoxin plus PGE_1, hemorrhage, and hemorrhage plus PGE_1 on superior mesenteric arterial blood flow, systemic arterial pressure, and heart rate. (Dog No. 3A-748.)

continuously monitoring the superior mesenteric arterial blood flow rate with an electromagnetic flow probe. Other investigators have also reported that the effects of prostaglandin E_1 in this dose range are transient.[26,29]

Potentially, prostaglandin E_1 may be superior to some of the vasodilators currently being considered for use in treating nonocclusive mesenteric ischemia, since the desired local effects can be obtained without greatly altering systemic hemodynamics. A comparative experimental study showed that the intramesenteric arterial administration of prostaglandin E_1 produced relatively fewer systemic hemodynamic changes than were observed with other vasodilators.[26] A recent clinical study confirmed that good local vasodilatation could be obtained in the peripheral vascular circulation with very low intraarterial doses of prostaglandin E_1 without producing adverse side effects.[10] The lack of a noticeable systemic hemodynamic effect from the intramesenteric use of prostaglandin E_1 is explained in part by the rapid removal of the drug from the circulation by the liver and lungs,[28,30] and in part by the low doses required to produce the desired local effect.

SUMMARY

This preliminary study suggests that due

to its potent vasodilator effect and rapid disappearance from the blood stream coupled with its known anti-platelet-aggregating properties, the intraarterial infusion of prostaglandin E_1 merits consideration as a possible mode of therapy in nonocclusive mesenteric ischemia.

Five minute infusions of 0.1 μg./kg. per min. prostaglandin E_1 satisfactorily reversed mesenteric arterial vasospasm which had been experimentally induced by digoxin or hemorrhage. The reversal of vasospasm was accompanied by a return to normal in the angiographic appearance of the superior mesenteric vascular bed and a resumption of a normal superior mesenteric arterial blood flow rate.

These effects were accomplished with only minor alterations in systemic hemodynamics.

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We wish to express our appreciation to Miss Irene Polansky (A.R.R.T.) and Mrs. Raquel Collins (B.S.) for their technical assistance. We are also indebted to Dr. Stephen George of the Biomathematics Department and Mr. Eugene Szwarc of the Medical Communications Department for their assistance.

REFERENCES

1. AAKHUS, T., and BRABAND, G. Angiography in acute superior mesenteric arterial insufficiency. *Acta radiol.* (Diag.), 1967, *6*, 1–12.
2. ANDERSON, J. H., GIANTURCO, C., and WALLACE, S. Automated technique for angiographic "spillover" determination of blood flow. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1975, *124*, 451–457.
3. ATHANASOULIS, C. A., WALTMAN, A. C., VAN URK, H., and BAUM, S. Blood flow measurement with spillover technique during intraarterial drug infusion. *Radiology*, 1973, *109*, 717–719.
4. BAUM, S. Small vessel angiography of gut. In: Symposium On Small Vessel Angiography. Edited by S. K. Hilal. C. V. Mosby Company, St. Louis, 1973, pp. 454–478.
5. BOLEY, S. J., SPRAYREGEN, S., VEITH, F. J., and SIEGELMAN, S. S. Aggressive roentgenologic and surgical approach to acute mesenteric ischemia. *Ann. Surg.*, 1973, *5*, 355–378.
6. BOLEY, S. J., and SIEGELMAN, S. S. Experimental and clinical nonocclusive mesenteric ischemia: pathophysiology, diagnosis, and management. In: Symposium on Small Vessel Angiography. Edited by S. K. Hilal. C. V. Mosby Company, St. Louis, 1973, pp. 438–453.
7. BRITT, L. G., and CHEEK, R. C. Nonocclusive mesenteric vascular disease: clinical and experimental observations. *Ann. Surg.*, 1969, 704–711.
8. BYNUM, T. E., and JACOBSON, E. D. Blood flow and gastrointestinal diseases. *Digestion*, 1971, *4*, 109–116.
9. BYNUM, T. E., HANLEY, H. G., and COLE, J. S. Effect of digitalis glycosides on splanchnic blood flow in man. *Clin. Res.*, 1973, *21*, 509.
10. CARLSON, L. A., ERICSSON, M., and ERIKSSON, U. Prostaglandin E_1 in peripheral arteriographies. *Invest. Radiol.*, 1973, 583–587.
11. DAVIS, L. J., ANDERSON, J., WALLACE, S., GIANTURCO, C., and JACOBSON, E. D. Use of prostaglandin E_1 to enhance angiographic visualization of splanchnic circulation. *Radiology*, 1975, *114*, 281–286.
12. FOGARTY, T. J., and FLETCHER, W. S. Genesis of nonocclusive mesenteric ischemia. *Am. J. Surg.*, 1966, *111*, 130–137.
13. GIANTURCO, C., SHIMIZU, T., STEFFERDA, F. R., and TAYLOR, R. P. Measurement of blood flow by angiography with increasing rate of injection. *Invest. Radiol.*, 1970, *5*, 361–363.
14. HARRISON, L. A., BLASCHKE, J., PHILLIPS, R. S., PRICE, W. E., DE V. COTTEN, M., and JACOBSON, E. D. Effects of ouabaine on splanchnic circulation. *J. Pharmacol. Exper. Therap.*, 1969, *169*, 321–327.
15. JACOBSON, E. D. Should digitalis be used in shock? *Medical Counterpoint*, 1969, 69–70.
16. JACOBSON, E. D. Mesenteric circulatory regulation in normal and ischemic states. In: Symposium on Small Vessel Angiography. Edited by S. K. Hilal. C. V. Mosby Company, St. Louis, 1973, pp. 434–437.
17. JORDAN, P. H., BOULAFENDIS, D., and GUINN, G. A. Factors other than major vascular occlusion that contribute to intestinal infarction. *Ann. Surg.*, 1970, *171*, 189–194.
18. MUGGIA, F. M. Hemorrhagic necrosis of intestine: its occurrence with digitalis intoxication. *Am. J. M. Sc.*, 1967, *253*, 263–271.
19. OLIN, T., and REDMAN, H. Spillover flowmeter: preliminary report. *Acta radiol.* (Diag.), 1964, *4*, 217–222.

20. OTTINGER, L., and AUSTEN, G. W. Study of 136 patients with mesenteric infarction. *Surg., Gynec. & Obst.*, 1967, *124*, 251–261.
21. PIERCE, G., and BROCKENBROUGH, E. Spectrum of mesenteric infarction. *Am. J. Surg.*, 1970, *119*, 233–239.
22. PRICE, W. E., ROHRER, G. V., and JACOBSON, E. D. Mesenteric vascular diseases. *Gastroenterology*, 1969, *57*, 599–604.
23. SAMUELSSON, B., GRANSTRÖM, E., GREEN, K., and HAMBERG, M. Metabolism of prostaglandins. *Ann. New York Acad. Sc.*, 1971, *180*, 138–161.
24. SCHEFFE, H. The Analysis of Variance. John Wiley & Sons, Inc., New York, 1959, pp. 69 and pp. 116–119.
25. SHANBOUR, L., JACOBSON, E. D., BROBMANN, G. F., and HINSHAW, L. B. Effects of ouabain on splanchnic hemodynamics in rhesus monkey. *Am. Heart J.*, 1971, *81*, 511–515.
26. SHEHADEH, Z., PRICE, W. E., and JACOBSON, E. D. Effects of vasoactive agents on intestinal blood flow and motility in dog. *Am. J. Physiol.*, 1969, *216*, 386–392.
27. SIEGELMAN, S. S., SPRAYREGEN, S., and BOLEY, S. J. Angiographic diagnosis of mesenteric arterial vasoconstriction. *Radiology*, 1974, *112*, 533–534.
28. STEEL, R. G. D., and TORVIE, J. H. Principles and Procedures of Statistics. McGraw-Hill Book Company, Philadelphia, 1960, pp. 401–402.
29. ULANO, H. B., TREAT, E., SHANBOUR, L. L., and JACOBSON, E. D. Selective dilation of constricted superior mesenteric artery. *Gastroenterology*, 1962, *1*, 39–47.
30. VANE, J. R. Release and fate of vasoactive hormones in circulation. *Brit. J. Pharmacol.*, 1969, *35*, 209–242.
31. WILLIAMS, L. F., JR., ANASTASIA, L. F., HASIOTIS, C. A., BOSNIAK M. A., and BYRNE, J. J. Experimental nonocclusive mesenteric ischemia. *Am. Surg.*, 1968, *115*, 82–88.
32. WILLIAMS, L. F., JR. Vascular insufficiency of intestines. *Gastroenterology*, 1971, *61*, 5, 757–777.
33. WILLIAMS, L. F., JR., and KIM, J. P. Nonocclusive mesenteric ischemia. In: Vascular Disease of the Intestine. Edited by S. J. Boley. Appleton-Century-Crofts, New York, 1971, pp. 519–530.
34. WITTENBERG, J., ATHANASOULIS, C. A., SHAPIRO, J. H., and WILLIAMS, L. F., JR. Radiological approach to patient with acute extensive bowel ischemia. *Radiology*, 1973, *106*, 13–24.

VOL. 125, NO. 1

SUPERIOR MESENTERIC ANGIOGRAPHY AND BLOOD FLOW FOLLOWING INTRA-ARTERIAL INJECTION OF PROSTAGLANDIN $F_{2\alpha}$*

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ABSTRACT:

The effect of intra-arterially administered prostaglandin $F_{2\alpha}$($PGF_{2\alpha}$) on venous demonstration in angiography of the superior mesenteric artery was compared to that of bradykinin in 10 patients, and the flow increase in this vessel following $PGF_{2\alpha}$ was measured by a dye dilution technique in another 8 patients.

The angiographic demonstration of veins was similarly improved by the 2 drugs, the effect of 60 μg. of $PGF_{2\alpha}$ being roughly equivalent to that of 10 μg. of bradykinin. The loss of arterial detail was less marked after $PGF_{2\alpha}$ which, however, had a longer duration of action. The flow studies indicated that 80 μg. of $PGF_{2\alpha}$ increased the superior mesenteric blood flow 103 ± 16 per cent. There was no influence on blood pressure or pulse rate. The patients experienced a sensation of warmth in the abdomen for a few seconds following the injection of the drugs, but no other side effect was noted.

It is concluded that $PGF_{2\alpha}$ causes a rapid increase of the superior mesenteric blood flow in man making it suitable for pharmacoangiography of this vascular bed.

THE prostaglandins form a group of biologically active fatty acids that have been found in many tissues in man and animals. They are thought to be involved in several regulatory mechanisms but their eventual physiological role is not known.

Exogenously administered prostaglandins principally affect glandular tissue and smooth muscle cells, among them vascular smooth muscle. As a rule, prostaglandins of the E and A series relax most vascular beds, reduce the total peripheral vascular resistance, and have been observed to produce a blood pressure fall despite an increased cardiac output.[1,4,7] Among the prostaglandins of the F series, prostaglandin $F_{2\alpha}$($PGF_{2\alpha}$) produces practically no change in blood pressure or heart rate in man in doses that produce marked effects on gastrointestinal or uterine smooth muscle.[12] However, even if the effect of $PGF_{2\alpha}$ on the total peripheral vascular resistance in man is negligible, it is possible that it can produce a redistribution of the peripheral blood flow, and after infusion of $PGF_{2\alpha}$ into the brachial artery in man, an increase of the forearm blood flow has been observed.[6] Therefore we included intra-arterially administered $PGF_{2\alpha}$ in a study of substances capable of producing an increase in the mesenteric blood flow of the type that is desirable in diagnostic arteriography.

MATERIAL

The effect of $PGF_{2\alpha}$ during angiography was investigated in 10 patients, and measurement of its effect on the superior mesenteric blood flow by a dye dilution technique was performed in another 8 patients.

Angiography. Angiographic examination of the superior mesenteric artery was performed in 4 males and 6 females, aged 31–67 years. Their eventual diagnoses were liver cirrhosis with portal hypertension (3), pancreatic pseudocyst (2), hypertension (2),

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carcinoma of the choledochal duct (1), carcinoma of the pancreas (1), and unclassified hematuria (1). The patients came for examination because of suspicion of liver metastasis, a suspicion that was not verified by angiography, liver scintigraphy, or subsequent operation.

Flow Studies. Determination of the superior mesenteric blood flow was performed in 6 women and 2 men, aged 65–78 years. Two of the patients had been operated upon 1 and 3 years previously with abdominoperineal amputation of the rectum because of rectal carcinoma. Three of the patients had rectal carcinoma and the remaining 3 patients carcinoma of the descending colon. None of these patients proved to have any tumor changes within the range of the superior mesenteric artery.

METHOD

Angiography. Catheterization of the superior mesenteric artery was made *via* the femoral artery using the Seldinger technique. A catheter with 2 side-holes and OD/ID 2.2/1.4 mm. was used. The contrast medium was injected with a pressure regulated syringe. Films were exposed with 1 film per second for the first 2 seconds, 2 films per second for 5, and another 4 films during 8 seconds.

The time between the start of injection and appearance of contrast medium in veins draining the jejunum and the ileocecal area was evaluated as well as the degree of maximal density of the venous filling. Moreover, the degree of reflux of contrast medium into the aorta at the time of the injection was estimated.

The drug to be tested was diluted in 10 ml. of saline. Bradykinin was always injected as a bolus followed immediately by the contrast injection. The $PGF_{2\alpha}$ was administered as a bolus in 5 patients, and in another 5 patients was slowly infused during 2 to 4 minutes; after a pause of 60 seconds this was followed by contrast injection.

Flow Studies. One to 2 days before the investigation the umbilical vein was opened with a probe under local or general anesthesia. A soft PVC catheter was introduced into the left branch of the portal vein and was kept patent by constant infusion of heparinized saline. After an overnight fast transumbilical portography was performed and was followed by measurements of the blood flow with the patient supine. During fluoroscopy the soft PVC catheter was replaced by a radiopaque catheter with 4 side-holes within 1.5 cm. from the curved tip. This catheter was placed in the superior mesenteric vein distal to the opening of the splenic and inferior mesenteric veins and proximally to the gastrocolic trunk and the first jejunal branch. The superior mesenteric artery was catheterized from the femoral artery with the same type of catheter with 2 side-holes.

The superior mesenteric blood flow was determined with a dye dilution technique. Indocyanine green (Cardiogreen; Hynson, Westcott & Dunning, Inc., U.S.A.) was given as a bolus injection into the superior mesenteric artery. During the sampling period blood from the superior mesenteric vein was continuously drawn through a spectrophotometer and the concentration curve was monitored on a potentiometer recorder. The catheterization and measuring techniques have been described in detail elsewhere.[16]

The superior mesenteric blood flow was calculated according to the Stewart-Hamilton formula as described by Lingårdh *et al.*[13] All investigations were started by determination of the superior mesenteric blood flow at rest based on 3 to 4 dye dilution curves. $PGF_{2\alpha}$ in a dose of 80 μg. dissolved in 10 ml. saline was injected into the superior mesenteric artery immediately after which the blood flow was repeatedly determined at different intervals. The appearance time and the mean transit time, in seconds, were determined with the method of Göthlin and Olin.[11] The vascular volume was estimated by multiplying the mean transit time in seconds by the blood flow in ml./seconds.

The portal venous pressure, in cm. H_2O, was recorded by an open manometer connected to the superior mesenteric vein

catheter. The midaxillary line with the patient in the supine position was taken as the baseline.

RESULTS

Angiography. A bolus of either 10 or 20 μg. of $PGF_{2\alpha}$, injected 60 seconds before contrast medium injection, improved angiographic demonstration of the veins markedly in those 2 patients in whom it was compared with angiography without drug. Veins of the jejunum as well as those of the cecum appeared earlier. Regurgitation to the aorta, which was present in 1 patient on the injection not preceded by drug, was decreased.

The effect of $PGF_{2\alpha}$ was similar to that of bradykinin. The time of appearance of contrast medium in jejunal as well as ileocecal veins was shortened and the density of the contrast medium in the superior mesenteric and portal veins was high. The effect of the dose of 28 μg. of $PGF_{2\alpha}$ corresponded well to that of 5 μg. of bradykinin and 60 μg. well to that of 10 μg. of bradykinin. (Fig. 1, *A* and *B*). Further increase of the dose of $PGF_{2\alpha}$ to 80 μg. was tried in 1 patient but did not further improve venous density. Among 5 patients in whom a dose of 60 μg. of $PGF_{2\alpha}$ was compared with 10 μg. of bradykinin, the venous appearance time was shorter after $PGF_{2\alpha}$ in 2 patients, and after bradykinin in 2; it was the same in 1. The density of the contrast medium in the veins was considered higher after $PGF_{2\alpha}$ in 3 patients, higher after bradykinin in 1, and similar in 1. The differences were, however, small. The flow changes were reflected correspondingly in diminished reflux of contrast medium to the aorta (Table 1).

The injection of $PGF_{2\alpha}$ in doses used by us did not cause any significant changes in the blood pressure or heart rate. At higher doses a few patients had a sensation of warmth in the lower abdomen. Some colicky pain of short duration was also ex-

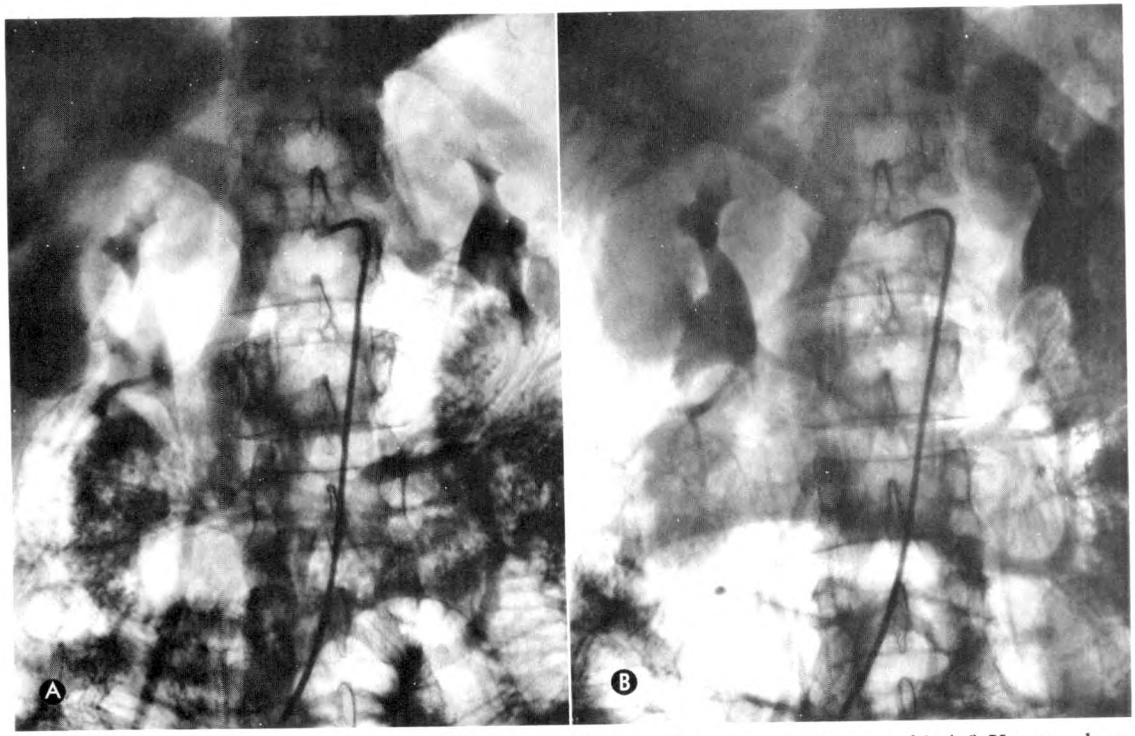

FIG. 1. Pharmacoangiography of the superior mesenteric artery in a woman 58 years old. (*A*) Venous phase after 10 μg. bradykinin and 40 ml. Isopaque Coronar. (*B*) Venous phase after 80 μg. $PGF_{2\alpha}$ and 40 ml. Isopaque Coronar. The density of the contrast medium in the veins after $PGF_{2\alpha}$ is similar to that after bradykinin.

TABLE I

RESULTS OF SUPERIOR MESENTERIC ANGIOGRAPHY AFTER PROSTAGLANDIN $F_{2\alpha}$ AND BRADYKININ IN 10 PATIENTS

Patient No.	Drug Dose		Volume of Contrast	Injection Rate (ml./sec.)	Appearance Time (sec.)		Contrast Density	Aortic Reflux	
	μg. $PGF_{2\alpha}$	μg. BK			Jejunal Veins	Ileocecal Veins			
1	—	—	30	11,5	8,4	14	+	−	
	10	—	40	14,2	4,4	4,4	++	−	
2	—	—	50	11,5	13,2	13,2	(+)	++	
	20	—	50	11,5	7,6	7,6	++	+	
3	—	5	60	20	7,6	6,4	++	−	
	28	—	60	21	9,6	6,4	++	+	
4	—	10	50	12	5,6	5,6	+	−	
	32	—	50	13	6,4	6,4	(+)	++	
5	—	10	60	11,1	4,0	—	++	−	$PGF_{2\alpha}$ infused during 4 min.
	60	—	60	13,0	7,6	—	+	+	
6	—	10	50	11,1	10,4	8,0	+	++	$PGF_{2\alpha}$ infused during 2 min.
	60	—	50	12,2	6,2	5,8	++	+	
7	—	10	50	12,7	6,5	6,5	(+)	++	$PGF_{2\alpha}$ infused during 2 min.
	60	—	50	12,6	6,0	6,0	++	(+)	
8	—	10	50	15,0	8,5	10,6	+	−	$PGF_{2\alpha}$ infused during 2 min.
	60	—	50	15,0	5,4	5,4	++	−	
9	—	10	50	12,5	5,4	—	++	−	$PGF_{2\alpha}$ infused during 2 min.
	60	—	50	13,0	6,6	—	++	−	
10	—	10	40	12,8	4,8	5,6	++	+	
	80	—	40	13,5	5,6	5,6	++	+	

perienced by some patients. These local sensations are familiar from the use of bradykinin.

Flow Studies. The superior mesenteric blood flow (SMBF) averaged 710 ml./min. (range 535–985 ml./min.) during the control period with the patients at rest after fasting. Injection of a bolus of 80 μg. $PGF_{2\alpha}$ into the superior mesenteric artery caused a rapid increase in the superior mesenteric blood flow (Fig. 2; and Table II). A mean maximal flow increase of 103 ± 16 per cent above the control value was reached 2 minutes after the injection, which deviated significantly from the normal blood flow ($p < 0.001$). During the following 10 minutes the flow gradually decreased to a value about 40 per cent above the resting flow, and even 40 minutes after the drug injection the flow was slightly increased.

The mean values of the appearance time, mean transit time, and vascular volume are given in Table III. The appearance time and the mean transit time decreased promptly with the increase in flow. At the time of maximal flow increase the initial values had decreased by 33 and 43 per cent, respectively, and these differed significantly from the control values ($p < 0.01$). The estimated vascular volume increased gradually from a mean resting value of 240 ± 42 ml. to 290 ± 60 ml. by the time of maximal

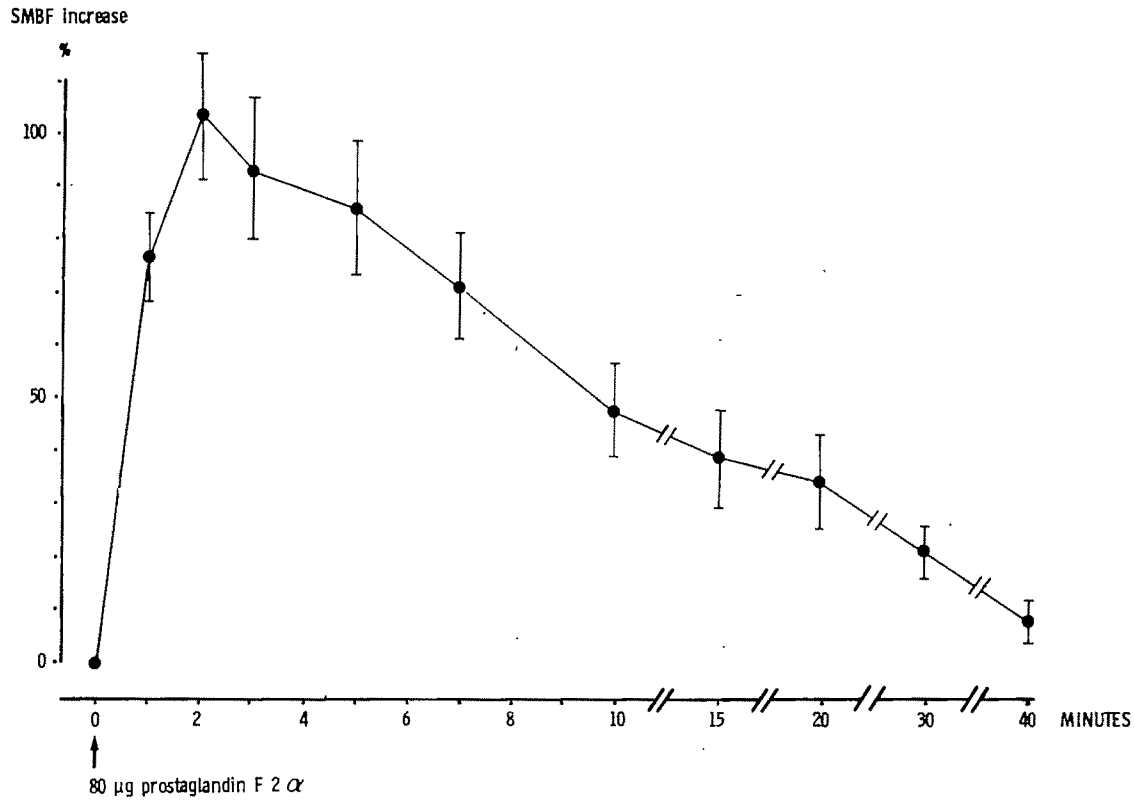

FIG. 2. Increase in superior mesenteric blood flow (SMBF) after injection of 80 µg. $PGF_{2\alpha}$ into the superior mesenteric artery. The curve represents mean values (± standard error in 8 patients). The arrow denotes the injection of $PGF_{2\alpha}$.

TABLE II

SUPERIOR MESENTERIC BLOOD FLOW (ML./MIN.) AFTER A BOLUS OF 80 µG. PROSTAGLANDIN $F_{2\alpha}$ INTO THE SUPERIOR MESENTERIC ARTERY IN 8 PATIENTS

Case No.	Control	Minutes after Prostaglandin Injection									
		1	2	3	5	7	10	15	20	30	40
1	750	1270	1450	1600	1420	1340	1330	1340	1130	—	—
2	985	1840	2200	—	2480	—	1330	1330	—	—	—
3	585	—	1150	1550	—	1110	—	690	670	—	—
4	750	1120	1090	—	1020	970	—	—	—	—	—
5	740	1070	1350	—	1350	1350	1100	1080	1010	870	840
6	710	1690	2000	—	1840	1650	—	—	1160	930	890
7	625	—	—	900	900	—	800	760	660	650	630
8	535	—	1080	800	800	—	840	730	740	720	—
n	8	5	7	4	7	5	5	6	6	4	3
mean	710	1398	1474	1213	1401	1284	1092	988	895	793	787
S.E. ±	48	155	171	211	225	116	109	123	95	65	80

TABLE III

	AT sec.	MTT sec.	VV ml.
Control value	6.3±0.4	19.7±1.4	240±40
Value at time of maximal flow	4.2±0.3	11.2±0.9	290±60
Per cent changes	−33±3	−43±3	+21±2

Appearance time (AT), mean transit time (MTT) and vascular volume (VV) of the superior mesenteric circulation following intra-arterial injection of prostaglandin $F_{2\alpha}$. Mean values in 8 patients with Standard Error Mean.

flow increase; the change, however, was not statistically significant.

The portal venous pressure increased slightly in 4 patients from a mean resting value of 8 cm. H_2O to 11 cm. H_2O ($p < 0.05$). The increase in pressure persisted for 20 to 30 minutes after which it returned to the resting level. The systemic blood pressure and pulse rate did not change significantly.

DISCUSSION

The importance of drugs for improvement of the demonstration of vessels such as venous collaterals in portal hypertension and tumor involvement of the veins in pancreatic carcinoma is well known. The more commonly used drugs for this purpose have been bradykinin[3] and tolazoline.[18] Glucagon[8] and the combination of phentolamine and isoproterenol[5] have also been tried. We have mainly been using bradykinin but this drug is no longer available. We therefore have been trying to find substitutes and included prostaglandin $F_{2\alpha}$ in this screening for reasons mentioned earlier.

Arteriographically it was found that after the injection of 60 μg. of $PGF_{2\alpha}$ the mesenteric blood flow increased as indicated by a shorter contrast appearance time and decreased regurgitation into the aorta. The venous demonstration was judged to be as good as, or better than, that produced by 10 μg. of bradykinin. It is to be noted that there was always an improved contrast density in the veins after $PGF_{2\alpha}$ as well as after bradykinin. Individual variations in the density of the contrast medium were, however, considerable and difficult to predict. It is therefore probable that many factors influence the result of drug injection on venous demonstration at arteriography, *e.g.*, the action of bradykinin on vascular permeability.[10] This increase of the vascular permeability also causes loss of arterial detail. After $PGF_{2\alpha}$ there was, despite the flow increase, much less loss of arterial detail compared to that caused by bradykinin, and the density of contrast medium in the veins was markedly increased. The mechanism behind this cannot be explained, but in rat experiments, $PGF_{2\alpha}$ has been observed to inhibit the increase in vascular permeability caused by histamine, serotonin, and bradykinin.[21] The capability of $PGF_{2\alpha}$ to constrict veins[19] may also be of importance. Noticeable constriction of the large veins at angiography was, however, not observed.

An estimation of the increase of the superior mesenteric blood flow after vasodilator drugs may be made from angiograms by evaluating the density of the venous filling and the time of appearance of contrast medium in the veins.[2,3] This estimation must be very crude, however, since exposures are made only at intervals rather than continuously, and quantitative conclusions suffer from this uncertainty. Several other factors such as patient size and exposure data also markedly influence the accuracy of the evaluations. We therefore tried to obtain more accurate data about the superior mesenteric blood flow by using a recently introduced dye dilution technique.[16] This method of flow determination has proved easy to handle and may be used during the angiographic procedure. The results obtained have been reproducible with a coefficient of variation of only 7 per cent[16] when measuring superior mesenteric blood flow under resting conditions. The method can only be used in patients without collateral circulation and with normal liver function.

The results of the flow studies indicated about 100 per cent increase of the superior mesenteric blood flow after the intra-arterial

administration of 80 μg. of $PGF_{2\alpha}$. A similar flow increase may be obtained by 5 μg. of bradykinin intra-arterially, but by the use of 20 μg. of bradykinin a flow increase amounting to 200 per cent may be achieved.[16] Whether such a great flow increase may be achieved by using $PGF_{2\alpha}$ in still increasing doses has not been investigated by us since the changes in blood flow obtained by doses up to 80 μg. proved adequate for arteriography.

The duration of the bradykinin effect is comparatively short, basal flow again being reached after about 15 minutes.[17] The duration of the $PGF_{2\alpha}$ effect was about 45 minutes which makes this drug a little less easy to handle, but since the loss of arterial detail is small this drawback may be of little importance. The long duration of the effect of $PGF_{2\alpha}$ contrasts to the very short half life of this substance in the circulation.[15] Apparently $PGF_{2\alpha}$ when bound to its target structure in the vessel wall may cause an effect with a duration quite different from that of the prostaglandin itself in circulating blood.

The approximately doubled superior mesenteric blood flow following the intra-arterial administration of 80 μg. of $PGF_{2\alpha}$ indicated a relaxation of the resistance of the vessels as the blood pressure remained unchanged. The flow increase was confirmed by the studies where the effect was apparent even with doses down to 10 μg. This splanchnic vasodilatation represents another example of the species variability often found when vascular effects of $PGF_{2\alpha}$ are studied. For example, infusion of $PGF_{2\alpha}$ into the mesenteric artery of the dog usually increases vascular resistance.[20] Prostaglandins of the A and E series seem to be more consistently relaxing in this respect, and might have been of interest in the present investigation but were not readily available.

The increased superior mesenteric blood flow in our patients following $PGF_{2\alpha}$ was not combined with any significant blood pressure fall. This may be explained by a vasoconstriction in some other vascular bed resulting in an unchanged total peripheral vascular resistance. Another explanation may be an increased venous return and increased cardiac output due to constriction of capacitance vessels, an effect of $PGF_{2\alpha}$ known from studies in animals[9] as well as in man.[19]

In our experience $PGF_{2\alpha}$ fills the requirement for a useful vasodilator in arteriography. It has few side effects and does not affect systemic blood pressure or heart rate. However, it should not be used in patients with a history of bronchial asthma as it is known to contract airway smooth muscle cells.[14]

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REFERENCES

1. Bergström, S., Duner, H., v. Euler, U. S., Pernow, B., and Sjövall, J. Observation of effects of infusion of prostaglandin E in man. *Acta physiol. scandinav.*, 1959, *45*, 145–151.
2. Boijsen, E., and Härtel, M. Kontrastmittelpassagezeit im Versorgungsgebiet der Arteria mesenterica superior. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1973, *118*, 491–498.
3. Boijsen, E., and Redman, H. C. Effect of bradykinin on celiac and superior mesenteric angiography. *Invest. Radiol.*, 1966, *1*, 422–430.
4. Carr, A. A. Hemodynamic and renal effects of prostaglandin, PGA_1, in subjects with essential hypertension. *Am. J. Med. Sc.*, 1970, *259*, 21–26.
5. Cioffi, C. M., Ruzicka, Jr., F. F., Carillo, F. J., and Gould, H. R. Enhanced visualization of portal system using phentolamine and isoproterenol in combination. *Radiology*, 1973, *108*, 43–49.
6. Collier, J. G., Karim, S. M. M., Robinson, B., and Somers, K. Effects of prostaglandins A_1, A_2, B_1, E_2 and $F_{2\alpha}$ on forearm arterial bed of man. *Brit. J. Pharmacol.*, 1972, *46*, 551–552.
7. Covino, B. G., Lee, J. B., and McMorrow, J. V. Circulatory effects of prostaglandins. *Circulation*, 1968, *38*, 60.
8. Danford, R. O., and Davidson, A. J. Use of glucagon as vasodilator in visceral angiography. *Radiology*, 1969, *93*, 173–175.
9. DuCharme, D. W., Weeks, J. R., and Montgomery, R. G. Studies on mechanism of

hypertensive effect of prostaglandin F_2 alpha. *J. Pharmacol. & Exper. Therap.*, 1968, *160*, 1–10.

10. Fasth, S., and Hulten, L. Effect of bradykinin on intestinal motility and blood flow. *Acta chir. scandinav.*, 1973, *139*, 699–705.
11. Göthlin, J., and Olin, T. Dye dilution technique with nephroangiography for determination of renal blood flow and related parameters. *Acta radiol. (Diag.)*, 1973, *14*, 113–137.
12. Karim, S. M. M., and Filshie, G. M. Therapeutic abortion using prostaglandin $F_{2\alpha}$I. *Lancet*, 1970, *1*, 157–159.
13. Lingårdh, G., Muth, T., and Olin, T. Renal blood flow in dogs studied by means of dye-dilution technique. *Scandinav. J. Urol.*, 1969, *3*, 281–290.
14. Mathe, A. A., Hedqvist, P., Holmgren, A., and Svanborg, N. Bronchial hyperreactivity to prostaglandin $F_{2\alpha}$ and histamine in patients with asthma. *Brit. M. J.*, 1973, *1*, 193–196.
15. McGiff, J. C., Terragno, N. A., Strand, J. C., Lee, J. B., and Lonigro, A. J. Selective passage of prostaglandins across lung. *Nature*, 1969, *223*, 742–745.
16. Norryd, C., Dencker, H., Lunderquist, A., and Olin, T. Superior mesenteric blood flow in man studied with dye-dilution technique. *Acta chir. scandinav.*, 1975, *141*, 109–118.
17. Norryd, C., Dencker, H., Lunderquist, A., and Olin, T. Superior mesenteric blood flow in man following injection of bradykinin and vasopressin into superior mesenteric artery. *Acta chir. scandinav.*, 1975, *141*, 119–128.
18. Redman, H. C., Reuter, S. R., and Miller, W. J. Improvement of superior mesenteric and portal vein visualization with tolazoline. *Invest. Radiol.*, 1969, *4*, 24–27.
19. Robinson, B. F., Collier, J. G., Karim, S. M. M., *et al.* Effect of prostaglandins A_1, A_2, B_1, E_2 and $F_{2\alpha}$ on forearm arterial bed and superficial hand veins in man. *Clin. Sc.*, 1973, *44*, 367–376.
20. Shehadeh, Z., Price, W. E., and Jacobsson, E. D. Effects of vasoactive agents on intestinal blood flow and motility in dog. *Am. J. Physiol.*, 1969, *216*, 386–392.
21. Willoughby, D. A. Effects of prostaglandins $PGF_{2\alpha}$ and PGE_1 on vascular permeability. *J. Path. & Bact.*, 1968, *96*, 381–387.

VOL. 125, NO. 1

SELECTIVE ARTERIAL EMBOLIZATION FOR CONTROL OF MASSIVE UPPER GASTROINTESTINAL BLEEDING*

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ABSTRACT:

Massive upper gastrointestinal bleeding was controlled in 11 of 15 patients by the use of selectively injected arterial emboli. Embolization is most successful in the treatment of patients with demonstrated arterial bleeding sites at angiography. This group of patients generally has ulcers and it is this group in whom vasopressin infusion has the lowest success rate.

At the same time we were successful in controlling only 1 of 4 patients who were bleeding from diffuse hemorrhagic gastritis, those patients in whom vasopressin infusion is very successful. We, therefore, now embolize only patients in whom arterial bleeding sites are demonstrated at angiography or in whom vasopressin infusion has failed to control the bleeding from hemorrhagic gastritis. Our experience also indicates that short acting occlusive agents, such as autogenous blood clot mixed with aminocaproic acid, are as successful in controlling bleeding as the more permanent types of embolic material.

THE generally accepted method for angiographic control of massive gastrointestinal bleeding is infusion of vasoconstrictive drugs, generally vasopressin, into the bleeding artery. In various series that have been reported, arterial bleeding has been controlled in 60–80 per cent of patients.[2,3,12,17] The method, however, is not without problems. First, the catheter must be left in the bleeding artery for several hours to days with the potential problem of thrombus formation. Second, fluid retention has been observed following long infusions because of the anti-diuretic hormone present in the commercially available vasopressin. Finally, vasopressin infusions have been less successful in controlling bleeding from peptic ulcers than in other forms of massive gastrointestinal bleeding.[12]

We have therefore evaluated an alternative method of controlling massive gastrointestinal bleeding: selective embolization of the bleeding artery.

MATERIAL AND METHOD

Between February, 1973, and July, 1974, we have treated 15 patients for massive upper gastrointestinal bleeding by embolization of the bleeding artery (Table 1). To date, only gastric and duodenal bleeders have been treated with this method. Of the 15 patients, 14 were not candidates for operation because of coexisting heart, lung, liver or renal disease; all had required transfusions of several units of blood.

After demonstration of the bleeding site by selective angiography in each patient, the catheter was advanced into the orifice of the left gastric or gastroduodenal artery, depending on whether the bleeding was gastric or duodenal. Autogenous blood clot mixed with epsilon aminocaproic acid

* Presented to the Seventy-Fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.

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The opinions or assertions contained herein are the private views of the authors and are not to be construed as official nor as reflecting the views of the Department of the United States Air Force or the Department of Defense.

TABLE I

SUMMARY OF CASES

Success	Age	Sex	Diagnosis	Angiographic Findings	Treatment	Result	Follow-up
1.	62	M	Left gastric aneurysm (angiography)	Left gastric aneurysm	Fat globules	Control	No rebleed up to 11 months Died of pneumonia
2.	48	M	Gastric ulcer (upper GI)	Active bleeding in fundus	0.5 cc. Amicar clot	Immediate control	No rebleed up to 14 months
3.	56	M	Gastric ulcer (endoscopy)	Active bleeding in fundus	0.5 cc. Amicar clot	Immediate control	No rebleed up to 14 months
4.	47	M	Hemorrhagic gastritis (endoscopy)	Gastritis with active bleeding in fundus	0.5 cc. Amicar clot	Immediate control	No rebleed for 2 weeks Subsequent variceal bleed and portacaval shunt
5.	52	M	Gastric ulcer (endoscopy)	Active lesser curvature bleeding	1.5 cc. Amicar clot	Immediate control	No rebleed up to 9 months
6.	62	F	No diagnosis established-too much clot (endoscopy)	Active bleeding in fundus	1.5 cc. Amicar clot	Control	No rebleed up to 6 months
7.	62	M	Hemorrhagic gastritis with a superficial fundal ulcer (endoscopy)	Active bleeding in body	5 pc. Gelfoam	Immediate control	No rebleed up to 6 months
8.	41	M	Duodenal ulcer (history and angiography)	Active bleeding in duodenal bulb	1.5 cc. Amicar clot	Immediate control	No rebleed up to 2 months
9.	22	M	Gastric ulcer (endoscopy)	Active lesser curvature bleeding	1.5 cc. Amicar clot	Immediate control	No rebleed up to 18 months
10.	64	M	Gastric ulcer (angiography)	Active bleeding in fundus	6 pc. Gelfoam	Immediate control	Bled 2 weeks later from gastritis; expired 1 month later from hepatic failure
11.	47	M	Pseudoaneurysm of gastroduodenal artery (angiography)	Actively bleeding gastroduodenal aneurysm	0.5 cc. Oxycel	Immediate control	No rebleed up to 3 months
Failure							
12.	47	M	Hemorrhagic gastritis (endoscopy)	Gastritis	2.5 cc. Oxycel	Continuous slow bleed	Vagotomy and pyloroplasty at 5 days; expired 1 month later
13.	84	F	Gastric ulcer (autopsy)	Active lesser curvature bleeding	0.5 cc. Amicar clot	Continuous bleeding	Expired at 20 hours
14.	35	F	Hemorrhagic gastritis (operation)	Gastritis	1.5 cc. Amicar clot	Continuous slow bleed	Vagotomy with pyloroplasty at 24 hours; expired at 1 month
15.	24	M	Hemorrhagic gastritis (endoscopy)	Gastritis	1.0 cc. Oxycel	Continuous slow bleed	Vagotomy and antrectomy at 24 hours; gastrectomy at 48 hours; expired at 1 month

(Amicar, Lederle) was then injected in 9 patients, Gelfoam (Upjohn, Inc.) in 2, Oxycel (Upjohn, Inc.) in 3 and fat emboli in 1 (Table I).

The Amicar-mixed autogenous blood clot was prepared by drawing 10 cc. of the patient's blood into a glass syringe containing 0.5 to 1 cc. Amicar. This was done immediately after insertion of the catheter into the patient's abdominal aorta and prior to the first flush with heparinized saline. The time required for demonstration of the bleeding, usually about 30 minutes, allowed the blood to form a firm clot. The clot was removed from the back end of the syringe, cut into 0.5 cc. pieces, and then forced through the selective catheter with a tuberculin syringe. Gelfoam emboli were prepared by cutting a strip of Gelfoam into cubes 2 to 4 mm. on a side. These cubes were injected along with saline. Oxycel emboli were prepared by mixing Oxycel fibers with blood and then cutting small pieces of 0.2 to 0.3 cc. and injecting them with saline through the catheter. Fat emboli were obtained from the adipose tissue adjacent to the femoral puncture site.

Following the injection of the emboli, a repeat angiogram was obtained to observe the completeness of the occlusion. If bleed-

ing persisted, additional emboli were injected in 0.5 cc. increments until the bleeding was successfully controlled. The catheter was then generally left in place from 1 to 6 additional hours in case bleeding recurred and further embolization was required.

RESULTS

Successful control of the bleeding was achieved in 11 of the 15 patients (Table 1). Success could be evaluated easily since all patients had nasogastric tubes in place and changes in the rate of bleeding could be judged by changes in the color of the saline rinses. In 9 patients, in whom control was immediate, the nasogastric return changed from bright red to pink to almost clear over a 5 to 15 minute period. In 2 additional patients, who were also considered to be controlled, the bleeding slowed immediately but the nasogastric return did not become clear for 1 to 3 hours. Follow-up of the 11 successfully treated patients revealed that 9 were still alive and well 2 to 18 months following embolization. One patient died of pneumonia unrelated to his original bleeding problem 11 months after embolization. Further bleeding had not occurred in this interval. The other patient had no further bleeding for 2 weeks, at which time a repeat massive hemorrhage was shown by endoscopy to be caused by hemorrhagic gastritis. The bleeding was controlled with an 18 hour infusion of epinephrine but he died a month later of hepatic failure.

The 4 patients who were not controlled by embolization continued to bleed. One, who had a large gastric ulcer eroding into a major branch of the left gastric artery, exsanguinated in 20 hours. The other 3, who had diffuse hemorrhagic gastritis, died of complications secondary to gastric surgery 1 month following embolization.

Follow-up angiograms were performed 12 to 36 hours following embolization in 5 of the patients receiving Amicar clot, 2 receiving Gelfoam, 1 receiving Oxycel, and the patient receiving fat emboli. In all patients receiving Amicar-mixed blood clot, most of the previously occluded branches were again patent but no active bleeding was seen (Fig. 1). In the patients receiving Gelfoam and Oxycel, however, the entire left gastric artery was thrombosed at the second examination, indicating that clot had propagated proximally from the site of the occlusion (Fig. 2). The patient receiving fat emboli fell between the two extremes, with a few branches open but most remaining occluded.

In 8 of the 15 patients the gastric mucosa was observed by endoscopy, operation, or autopsy 1 to 5 days following embolization. In 7 there was no mucosal necrosis, although patients frequently had changes of gastritis which probably antedated the embolization. In 1 patient, who received 2.5 cc. of Oxycel in an attempt to control bleeding from diffuse hemorrhagic gastritis, operation 5 days later revealed areas of sloughed gastric mucosa, but no bleeding was seen.

DISCUSSION

Several materials may be used for embolic occlusion. These can be grouped as short acting, of intermediate duration, and permanent. In the first group, the most commonly used is autogenous blood clot, which has the advantage of being an endogenous material. Also, it fragments into many small emboli as it passes through the catheter. Fragmentation of emboli is an important advantage because the many small embolic fragments shower the branches of the bleeding artery, causing peripheral occlusions. The more peripheral the occlusion, the less likely that collateral blood supply will develop around the occlusion. Also, the small emboli hopefully are flow directed toward the bleeding artery, in which the lack of peripheral resistance results in an increased blood flow. Most autogenous blood clot emboli are absorbed within a few hours.[13] For this reason, we have mixed the blood clot with epsilon aminocaproic acid (Amicar) to prolong its duration of occlusion. Amicar reacts with plasmin to form a fibrin which has an increased

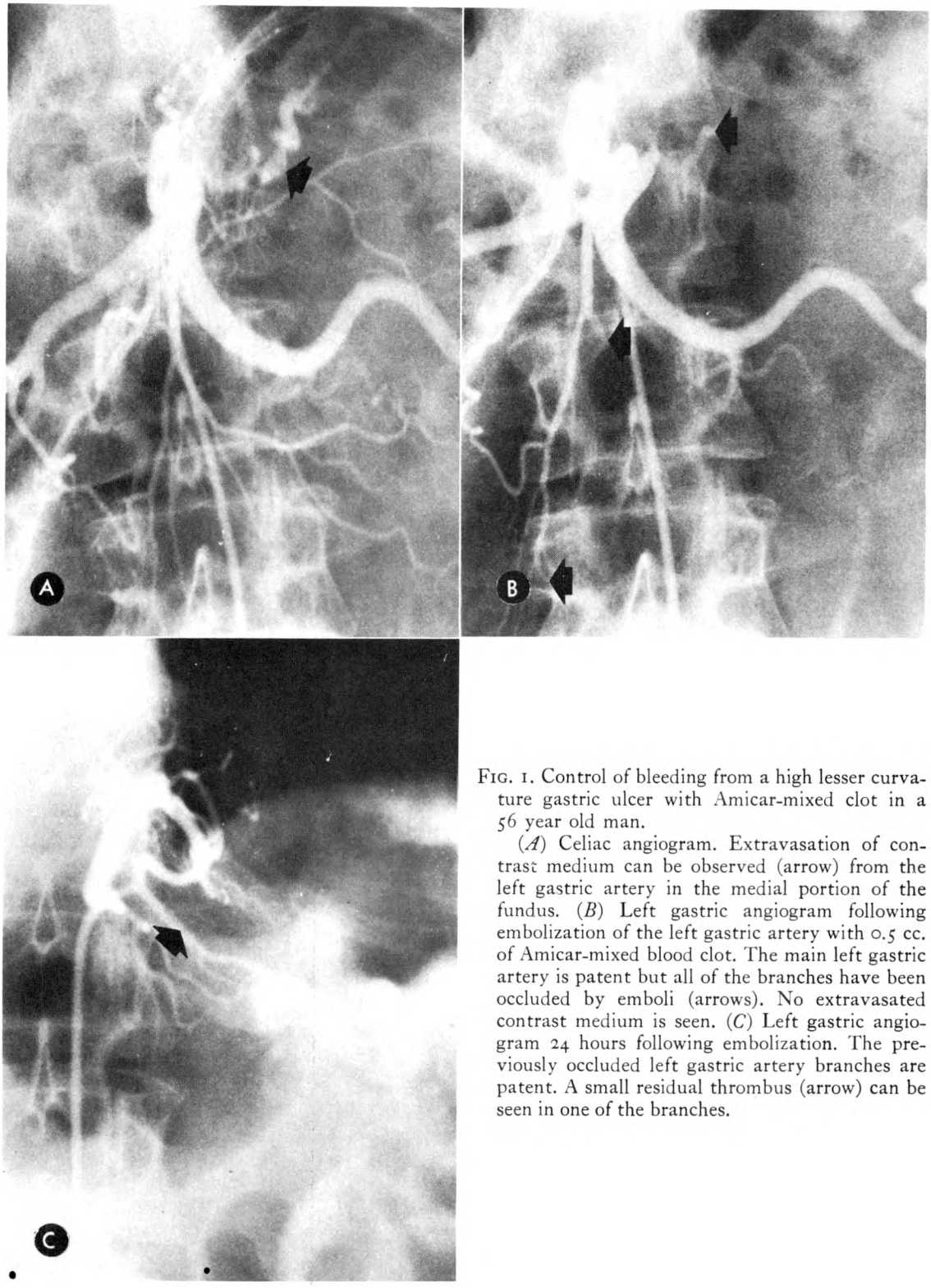

FIG. 1. Control of bleeding from a high lesser curvature gastric ulcer with Amicar-mixed clot in a 56 year old man.

(*A*) Celiac angiogram. Extravasation of contrast medium can be observed (arrow) from the left gastric artery in the medial portion of the fundus. (*B*) Left gastric angiogram following embolization of the left gastric artery with 0.5 cc. of Amicar-mixed blood clot. The main left gastric artery is patent but all of the branches have been occluded by emboli (arrows). No extravasated contrast medium is seen. (*C*) Left gastric angiogram 24 hours following embolization. The previously occluded left gastric artery branches are patent. A small residual thrombus (arrow) can be seen in one of the branches.

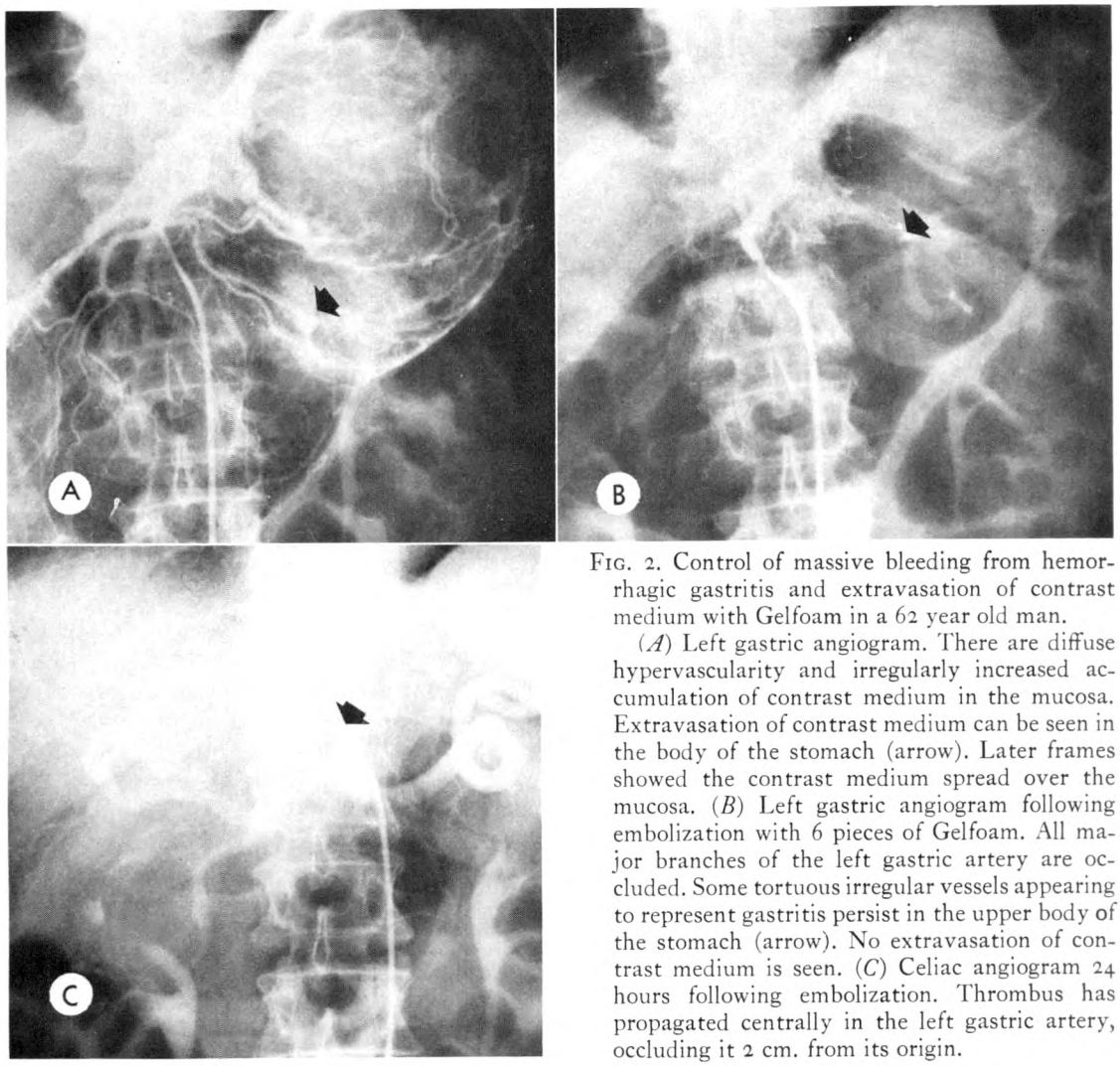

Fig. 2. Control of massive bleeding from hemorrhagic gastritis and extravasation of contrast medium with Gelfoam in a 62 year old man.

(*A*) Left gastric angiogram. There are diffuse hypervascularity and irregularly increased accumulation of contrast medium in the mucosa. Extravasation of contrast medium can be seen in the body of the stomach (arrow). Later frames showed the contrast medium spread over the mucosa. (*B*) Left gastric angiogram following embolization with 6 pieces of Gelfoam. All major branches of the left gastric artery are occluded. Some tortuous irregular vessels appearing to represent gastritis persist in the upper body of the stomach (arrow). No extravasation of contrast medium is seen. (*C*) Celiac angiogram 24 hours following embolization. Thrombus has propagated centrally in the left gastric artery, occluding it 2 cm. from its origin.

resistance to fibrinolysis.[7] In animal experiments, most Amicar-mixed clot emboli lyses within 24 hours, but some have lasted up to 2 weeks.[5]

An interesting observation accompanying the use of Amicar-mixed clot is that the bleeding artery appears to remain occluded, while most of the surrounding, normal arteries become patent by 24 hours.[10] The only explanation we can find for this persistence is the work of Warren *et al.*,[16] who have found that the amount of plasmin activator in the intima of damaged vessels is decreased. However, the amount of plasmin activator in arterial walls is small compared to circulating activator, and the importance of this observation is not certain. It is apparent, however, that clot does lyse from normal vessels while the bleeding arterial branch appears to remain occluded. A more controlled type of temporary occlusion might be produced by gelatin microspheres of known size and duration of occlusion.[1] Such emboli have the advantages of autogenous blood clot but have a more controllable duration of occlusion.

Occlusion lasting from weeks to permanently can be obtained by using absorbable, non-autogenous materials, such as Oxycel[4] or Gelfoam. Although Gelfoam is not absorbed for up to 2 weeks, animal experiments have shown that it is pushed peripherally and compressed by arterial pulsation.[5] Therefore, an occluded artery

may appear to open and branches fill with contrast medium over a period up to 24 hours. However, in the patients in this series occlusions with Oxycel or Gelfoam appear to have become permanent, with propagation of thrombus from the point of occlusion back to the orifice of the left gastric artery.

Finally, permanent occlusions can be achieved with the use of isobutyl-2-cyanoacrylate,[14] lead shot,[11] Ivalon,[15] or plastic microspheres.

Although massive arterial bleeding can be controlled with any of these embolic materials, our experience indicates that adequate control can be achieved with Amicar-mixed autogenous blood clot, and that the more permanent types of occlusive materials are not necessary unless the patient's blood cannot be made to clot. In some patients, particularly those who have received several transfusions, or who have a coagulopathy, the blood will not clot because of the depletion of platelets or clotting factors. Occasionally such blood can be made to clot by the addition of a few drops of thrombin. However, the resulting clot is not particularly firm or easy to cut, and an artificial embolic material may be preferred in this type of patient.

Our experience also indicates that embolic control of upper gastrointestinal bleeding is more successful when the bleeding is localized, such as generally occurs with ulcers. Of the 11 patients in whom a bleeding site was demonstrated on the angiograms, we controlled the bleeding in 10 (91 per cent). Four patients were embolized without angiographic demonstration of active extravasation. One, with a left gastric artery aneurysm, was treated successfully. The other 3 had hemorrhagic gastritis shown by endoscopy; the treatment failed in all 3 and they died following gastric surgery. Probably the same rich anastomotic circulation which prevents gastric necrosis also hinders the control of diffusely bleeding lesions such as gastritis or multiple ulcerations. We therefore recommend that patients with hemorrhagic gastritis be treated by vasopressin infusion. According to Athanasoulis *et al.*,[2] bleeding from diffuse gastritis can be controlled in 84 per cent of patients. Embolization should be reserved for those patients in whom a localized bleeding site is seen at angiography or in whom infusion fails to control the bleeding.

Of the 8 patients in whom observation of the gastric mucosa was possible following embolization, slough of mucosa occurred in only 1 patient and he was not bleeding from the denuded areas. The large quantity of 2.5 cc. of Oxycel mixed clot was used for the embolization, and repeated injections of emboli were made until the left gastric artery was occluded. We have not used as much embolic material in the other patients. However, even with small amounts of material, a segment of the bleeding organ becomes at least temporarily ischemic, and infarction is a possibility that must be considered with each embolization. Prochaska *et al.*[9] have reported gastric necrosis in a patient in whom the left gastric artery was embolized with autogenous clot. However, this patient also had 54 hours of vasopressin infusion in addition to the embolization and it is not clear that the ischemia would have occurred with the clot alone.

Another potential complication is inadvertent embolization of the spleen or pancreas. Although splenic infarction may be painful, serious sequelae do not appear to result from embolization of the spleen. Maddison[6] has infarcted a spleen in a patient with hypersplenism, and Tadavarthy *et al.*[15] have occluded a splenic artery during the embolization of a bleeding false aneurysm. Also, animal experiments have shown that the spleen can be embolized without adverse effects.[5] The effects of pancreatic embolization are less certain. It is known that occlusion of small pancreatic arteries with microspheres 8–20 microns in diameter consistently results in severe pancreatitis experimentally.[8] None of the patients in this series had symptoms to suggest that pancreatitis had occurred. In both of the patients with gastroduodenal embolization, pancreatic branches were occluded by em-

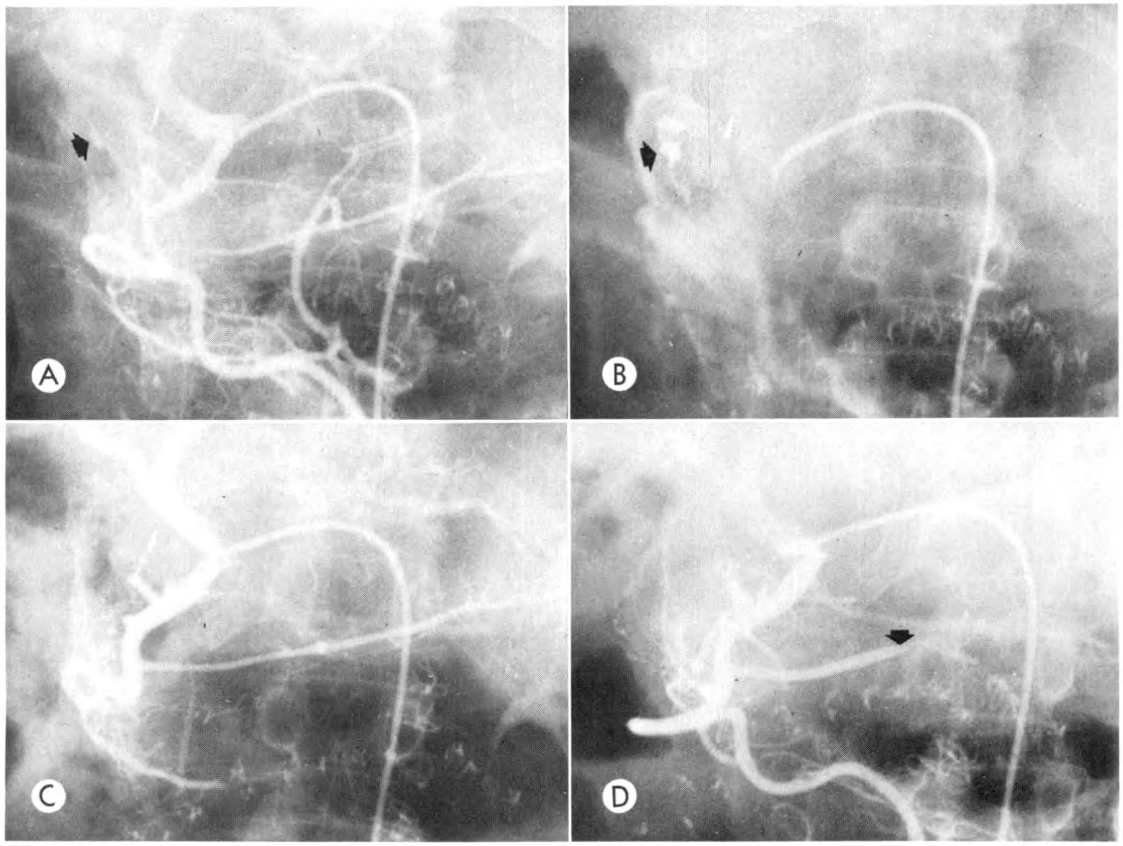

Fig. 3. Control of massive bleeding from a duodenal ulcer with Amicar-mixed clot in a 41 year old man with hepatic coma.

(*A*) Gastroduodenal angiogram. Arterial phase. Contrast medium extravasates from a proximal branch of the posterior pancreaticoduodenal arcade (arrow). (*B*) Capillary phase. The extravasated contrast medium layers in the duodenal bulb (arrow). (*C*) Gastroduodenal angiogram following embolization of the gastroduodenal artery with 1.5 cc. of Amicar-mixed autogenous blood clot. Both posterior and anterior pancreaticoduodenal arcades are occluded. No extravasation is seen in the region of the duodenal bulb. (*D*) Gastroduodenal angiogram 24 hours after embolization. The pancreaticoduodenal arcades and gastroepiploic artery are again patent, although some residual thrombus remains. No extravasation of contrast medium is seen. The apparent occlusion of the transverse pancreatic artery (arrow) is caused by reversal of blood flow in this vessel; the contrast medium washed out toward the gastroduodenal artery.

boli (Fig. 3). However, the emboli we use are large relative to microspheres, and the pancreatic occlusions probably occur proximally. This should allow the formation of an extensive collateral circulation to develop distally. The pancreas is probably the organ in the body with the best possibilities for collateral circulation, receiving its blood supply from several different arteries. Although pancreatitis must be considered a potential complication of the method, we have had no indication that it occurs.

It should be stressed that most of these 15 patients were terminal bleeders. Although they were bleeding massively, the surgeons refused to operate because of coexisting heart, lung, kidney, or liver disease. Our over-all success rate in these extremely difficult patients was 73 per cent (11 of 15 patients). If the 4 patients who had no localized extravasation at angiography are excluded, the success rate was 91 per cent (10 of 11 patients). Embolization appears to have 2 major advantages over infusion as a method for treating massive bleeding. First, it is extremely simple

and the results can be assessed immediately. Second, the patient does not receive large doses of vasoconstrictive drugs. The many hours of monitoring for side effects of the drugs and checking that the catheter tip does not come out of the bleeding artery, therefore, are not necessary. Although much further experience will be necessary before embolization can be considered as safe and effective as it is simple, our preliminary results are encouraging.

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ADDENDUM

Since the submission of this paper we have attempted to control massive upper gastrointestinal hemorrhage by left gastric artery embolization in seven additional patients. Two had gastric ulcers; two, duodenal ulcers; two Mallory-Weiss lacerations; and one gastritis. The bleeding was controlled immediately in the first six and no further bleeding has occurred up to six months of follow-up. The bleeding in the patient with gastritis was controlled for 24 hours, when further bleeding necessitated a gastrotomy with ligation of several bleeding sites. A repeat bleed one month later was successfully controlled by vasopressin infusion.

Thus, we have controlled massive upper gastrointestinal hemorrhage by selective embolization of the bleeding artery in 17 of 22 patients. In patients with bleeding from ulcers or Mallory-Weiss lacerations, 16 of 17 (94 per cent) were controlled.

REFERENCES

1. ALMEN, T., and NYLANDER, G. Personal communication.
2. ATHANASOULIS, C. A., BAUM, S., WALTMAN, A. C., RING, E. J., IMBEMBO, A., and VANDER SALM, T. J. Control of acute gastric mucosal hemorrhage: intra-arterial infusion of posterior pituitary extract. *New England J. Med.*, 1974, *290*, 597–603.
3. BAUM, S., and NUSBAUM, M. Control of gastrointestinal hemorrhage by selective mesenteric arterial infusion of vasopressin. *Radiology*, 1971, *98*, 497–505.
4. BOOKSTEIN, J. J., CHLOSTA, E., WALTERS, J., and FOLEY, D. Trans-catheter hemostasis of gastrointestinal bleeding using modified autogenous clot. *Radiology*, 1974, *113*, 277–285.
5. CHUANG, V. P., and REUTER, S. R. Experimental diminution of splenic function by selective embolization of splenic artery. *Surg., Gynec. & Obst.*, 1975, *140*, 715–720.
6. MADDISON, F. E. Embolic therapy of hypersplenism. *Invest. Radiol.*, 1973, *8*, 280.
7. MAXWELL, R. E., and ALLEN, D. Interactions of E-aminocaproic acid with thrombin clotting and fibrinolytic systems. *Nature*, 1966, *209*, 211–213.
8. PFEFFER, R. B., LAZARINI-ROBERTSON, A., JR., SAFADI, D., MIXTER, G., JR., SECOY, C. F., and HINTON, J. W. Gradations of pancreatitis, edematous through hemorrhagic, experimentally produced by controlled injection of microspheres into blood vessels in dogs. *Surgery*, 1962, *51*, 764–769.
9. PROCHASKA, J. M., FLYE, M. W., and JOHNSRUDE, I. S. Left gastric artery embolization for control of gastric bleeding: complication. *Radiology*, 1973, *107*, 521–522.
10. REUTER, S. R., and CHUANG, V. P. Control of abdominal bleeding with autogenous embolized material. *Radiologe*, 1974, *14*, 86–91.
11. RIZK, G. K., ATALLAH, N. K., and BRIDI, G. I. Renal arteriovenous fistula treated by catheter embolization. *Brit. J. Radiol.*, 1973, *46*, 222–224.
12. RÖSCH, J., DOTTER, C. T., and ANTONOVIC, R. Selective vasoconstrictor infusion in management of arterio-capillary gastrointestinal hemorrhage. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *116*, 279–288.
13. RÖSCH, J., DOTTER, C. T., and BROWN, M. J. Selective arterial embolization: new method for control of acute gastrointestinal bleeding. *Radiology*, 1972, *102*, 303–306.
14. RÖSCH, J., GOLDMAN, N. L., and DOTTER, C. T. Percutaneous occlusion of gastric coronary vein. Twenty-second Annual Meeting, Association of University Radiologists, New York, May 8–11, 1974.
15. TADAVARTHY, S. M., KNIGHT, L., OVITT, T. W., SNYDER, C., and AMPLATZ, K. Therapeutic transcatheter arterial embolization. *Radiology*, 1974, *112*, 13–16.
16. WARREN, B. A., *et al.* Fibrinolytic activity of vascular endothelium. *Brit. M. Bull.*, 1964, *20*, 213–214.
17. WHITE, R. E., GIRARGIANA, F. A., JR., and BELL, W. Bleeding duodenal ulcer control: selective arterial embolization with autogenous blood clot. *J.A.M.A.*, 1974, *229*, 546–548.

VOL. 125, NO. 1

LIMITED FORM OF WEGENER'S GRANULOMATOSIS IN ULCERATIVE COLITIS*

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ABSTRACT:

The limited form of Wegener's granulomatosis is described as a complication in 2 cases of chronic ulcerative colitis. The coexistence of ulcerative colitis and the limited form of Wegener's granulomatosis raises the question of a common etiologic basis, possibly an immunologic defect. Awareness of such an association may help shed further light on the etiology of both disorders.

PULMONARY lesions can rarely be an extracolonic complication of ulcerative colitis.[2,4] The limited form of Wegener's granulomatosis, as described in the 2 patients in this paper, has not previously been reported in association with chronic ulcerative colitis. This experience indicates that the limited form of Wegener's granulomatosis should be considered in the differential diagnosis of nodular lung lesions in ulcerative colitis. It is possible that there is a common immunologic basis for both entities.

REPORT OF CASES

CASE 1. This 30 year old Caucasian female presented with a 3 day history of right pleuritic chest pain without dyspnea, cough, or fever. She had an 8 year history of intermittent bloody diarrhea lasting 3–6 months and requiring, at times, 60 mg. of prednisone daily for control. The last course of steroids (5 mg. per day) was March through July, 1974. The patient had been symptom-free for 2–3 months prior to admission. She claimed to be allergic to sulfonamides but was unable to recall the specific reaction. Physical examination revealed the patient to be afebrile and the chest clear to auscultation. The findings otherwise were unremarkable.

Chest roentgenograms in August 1974 (Fig. 1, *A–C*) showed a small amount of right pleural fluid, a 4 cm. nodular, pleural-based density in the right lower lobe, and a 1.5 cm. nodular, pleural-based density in the left lower lobe. A chest roentgenogram 1 year previously had been normal. The perfusion lung scan showed nonspecific defects in the area of the nodular densities. Skin test with intermediate strength PPD was negative. Thoracentesis yielded 50 cc. of pink fluid with specific gravity 1.021, red blood cell count of 30,250 and white blood cell count of 12,700 with a differential count of 38 neutrophils, 40 lymphocytes, 4 eosinophils and 18 mesothelial cells. Routine culture yielded heavy growth of *Staphylococcus aureus*. Other laboratory studies included hemoglobin 10.4 gm. per cent, hematocrit 32.6 per cent, white blood cell count 7,800 with a differential count of 61 neutrophils, 1 eosinophil, 27 lymphocytes, and 11 monocytes. The erythrocyte sedimentation rate was 74 mm. Serologic study was negative for Histoplasma, Aspergillus, Candida, Coccidioidomyces and *Mycoplasma pneumoniae*. Cold agglutinins, antinuclear antibodies and LE preparations were negative. Serum for rheumatoid factor was weakly positive. Stool was negative for occult blood. Urinalysis was normal. Barium enema examination (Fig. 2, *A* and *B*) showed typical changes of ulcerative colitis without evidence of malignancy. Proctoscopy showed a mucosal pattern compatible with ulcerative colitis in remission.

Needle biopsy of the right lower lobe lung lesion was interpreted as nondiagnostic. Open biopsy was done September 16, 1974, on the right lower lobe lesion. There were adhesions to the chest wall and on gross examination the mass was considered to be tumor. Cultures of

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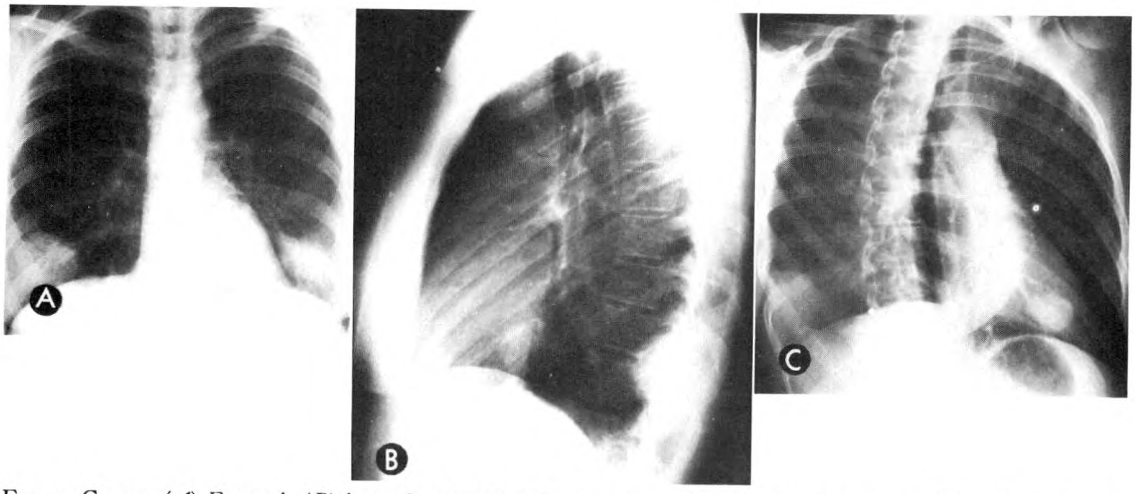

FIG. 1. Case 1. (*A*) Frontal, (*B*) lateral and (*C*) right anterior oblique roentgenograms of the chest show a pleural-based density in the right lower lobe and a smaller nodule in the left lower lobe in the area of the major fissure.

the specimen showed no growth.

Chest roentgenogram 3 months after the initial presentation showed complete resolution of the left lobe lesion without any treatment. The patient is asymptomatic at this time.

CASE II. This 45 year old Caucasian female has a 19 year history of ulcerative colitis. In 1955 during a pregnancy she developed bloody diarrhea accompanied by an erythematous, nodular rash, hand stiffness, fever, left lung infiltrate and myocarditis. No hematuria or renal involvement was present. A diagnosis of polyarteritis nodosa (without tissue documentation) was made and she responded to ACTH therapy with only occasional exacerbation of

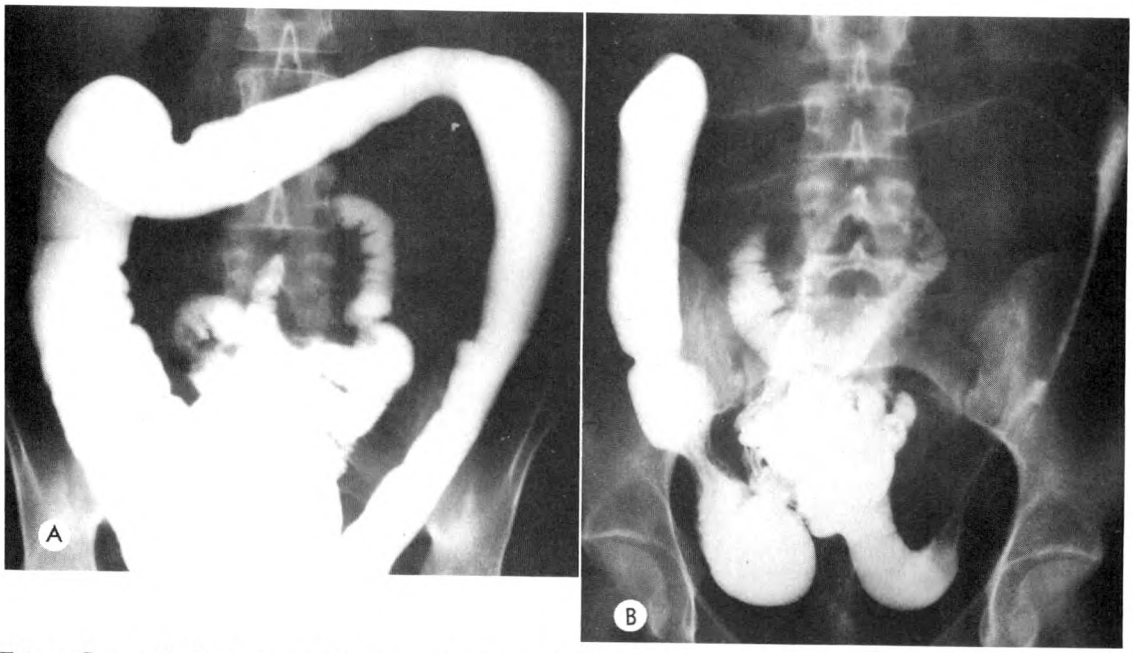

FIG. 2. Case 1. Barium-enema (*A*) pre-evacuation and (*B*) post-evacuation anteroposterior roentgenograms show loss of haustration and mucosal edema throughout the colon. The terminal ileum is normal.

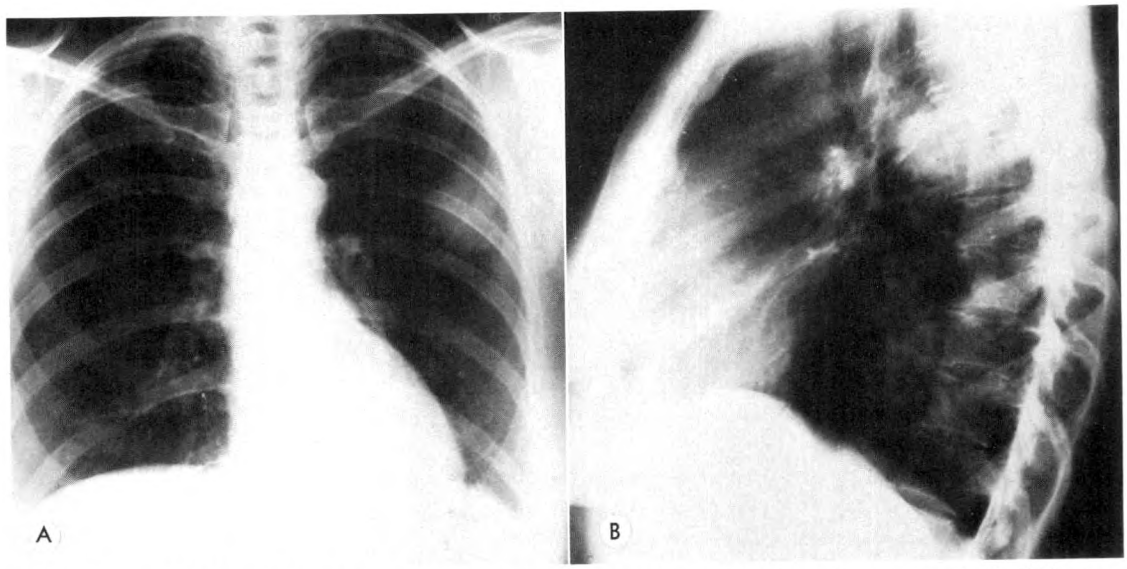

Fig. 3. Case 11. (*A*) Anteroposterior and (*B*) lateral roentgenograms of the chest show a mass in the left upper lobe and a questionable second lesion projecting over the spine on the lateral roentgenogram.

bloody diarrhea. In 1959 a barium enema examination showed left-sided colitis. Her last severe episode was 3 years ago and responded to steroids and Lomotil. The patient claimed to be allergic to sulfonamides and penicillin, although she could not recall a specific reaction. She had had bronchial asthma at age 14 which lasted approximately 1 year. In November, 1973, 2 months after a trip to California, she had an exacerbation of colitis followed by the abrupt onset of left pleuritic chest pain not associated with cough or hemoptysis. This was accompanied by occasional increase in temperature to 102°F. The chest roentgenograms (Fig. 3, *A* and *B*) revealed a 3 cm. pleural-based density in the left upper lobe, and a possible second lesion in the left lower lobe projecting over the spine on the lateral chest roentgenogram. A previous chest roentgenogram in April 1973 was normal. Because of the febrile episode she was treated with a course of antibiotics without change in the lesion. She was then admitted for further work-up.

On admission the patient was afebrile and her chest was clear to auscultation and percussion. The remainder of the physical examination was likewise unremarkable. Laboratory studies included hemoglobin 11.8 gm. per cent and hematocrit 33.6 per cent. The white blood cell count was 9,200 with a differential of 19 rod neutrophils, 67 neutrophils, 3 eosinophils, 10 lymphocytes and 1 monocyte. Anisocytosis, poikilocytosis, and hypochromia were noted on the blood smear. Urinalysis was normal. Stools were moderately positive for occult blood. Other laboratory studies included erythrocyte sedimentation rate 63 mm., serum total protein 6.3 gm. per cent, with albumin 3.1 per cent. Antinuclear antibody, latex fixation, rheumatoid factor and serologic study for coccidioidomycosis were all negative. Barium enema examination (Fig. 4, *A* and *B*) revealed active ulcerative colitis involving the entire colon. Intravenous pyelography and upper gastrointestinal tract examination were negative. Protein electrophoresis was normal.

Because of the possibility of an infectious process, the patient was again treated with antibiotics and discharged under observation. The lesion remained unchanged and she was admitted to the surgical service. The patient underwent bronchoscopy with biopsy and mediastinoscopy with biopsy. Bronchial washings were negative for malignancy. Carinal and mediastinal lymph node biopsies were negative for malignancy and granuloma. Left thoracotomy was performed and a mass 5 cm. in diameter was found in the major fissure involving both upper and lower lobes. Multiple pleural adhesions to the chest wall and diaphragm were seen.

The patient is well without any new lesions 14 months after the initial presentation. She was treated with steroids.

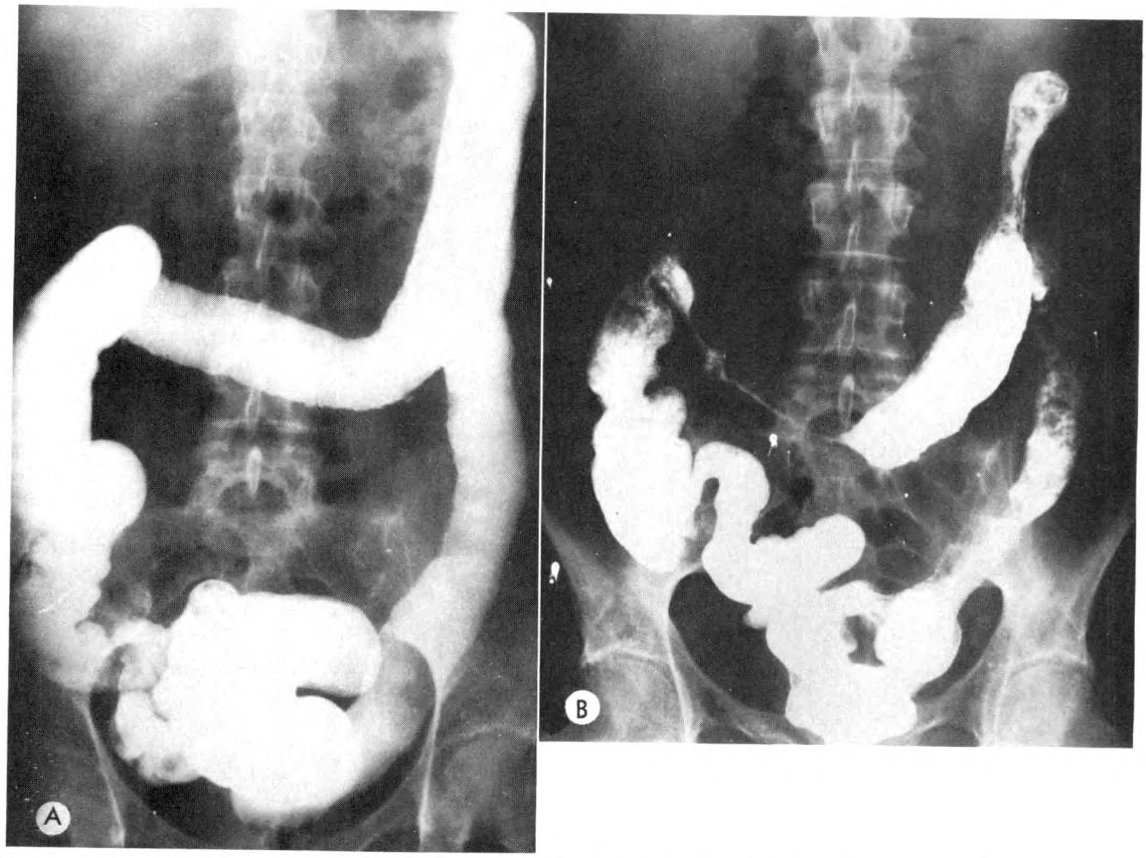

FIG. 4. Case II. Barium enema (*A*) pre-evacuation and (*B*) post-evacuation roentgenograms show extensive ulceration, mucosal edema, and loss of haustration throughout the colon. The terminal ileum shows backwash ileitis.

PATHOLOGICAL FINDINGS

The microscopic changes in these 2 cases were remarkably similar, and will therefore be described together. A chronic pleuritis and organizing pneumonitis (Fig. 5) of nonspecific type were noted in each case. Against this background there were areas of parenchymal necrosis, either sizable or smaller, with a multifocal distribution and having serpiginous borders. At the edge of the necrotic zones were elongated histiocytes, aligned in a palisading pattern (Fig. 6). The necrotic zones contained proteinaceous coagulum and some nuclear debris; occasionally some viable medium-sized arteries with intimal fibrosis and marked luminal narrowing were encompassed by the infarct-like areas of necrosis. Smaller arteries and veins frequently demonstrated a mural infiltration by round cells, focal areas of fibrinoid necrosis, and sectoral loss of elastic lamellae (Fig. 7; and 8). Many such vessels also displayed intimal fibrosis and a variable degree of luminal narrowing. Other vessels revealed peri-adventitial fibrosis (Fig. 9). Some involved vessels were accompanied by non-caseating granulomata composed of epithelioid cells and Langhans' type giant cells (Fig. 5; and 8). Similar granulomata were found unassociated with vessels or necrotic foci (Fig. 6; and 9). In Case II a single granuloma was noted in the pleura, immediately adjacent to the lung parenchyma, but neither vasculitis nor necrosis was seen in the pleura. Case II also contained fair numbers of eosinophils within the pneumonitic areas but none was found

occupying vessel walls or granulomata. Case 1, on the other hand, showed rare eosinophils in 2 vein walls. Special stains for acid-fast bacilli and fungi were negative in both cases. The pathologic diagnosis was a limited form of Wegener's granulomatosis.

DISCUSSION

The roentgenographic findings in both our cases are nonspecific. The presumptive roentgenographic diagnosis of the pulmonary lesions was neoplasm, possibly metastatic. The differential diagnosis included infectious processes, granulomata, infarcts and vasculitis. Because the roentgenographic findings remained stable for approximately 1 month in both patients, a lung biopsy was required for definitive diagnosis: primarily to exclude neoplasm. The other previously reported cases of lung lesions in ulcerative colitis are distinctly different from those in our patients. Wasserman and co-worker's[4] patient with chronic ulcerative colitis had asthma, persistent eosinophilia, hepatosplenomegaly, paresthesias, and convulsions. The chest roentgenogram was described as showing a moderate increase in the pulmonary vascular markings throughout both lung fields. Lung biopsy showed diffuse

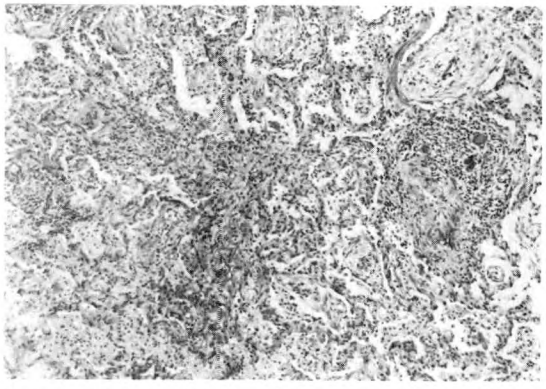

Fig. 5. Case 11. Nonspecific pneumonitis which is active in the lower portion and organizing in the upper part of the field. A venule with fibrinoid necrosis is seen toward the right. Below it there is some peri-adventitial fibrosis, while in its upper right wall a noncaseating granuloma is present. (HPS×100.)

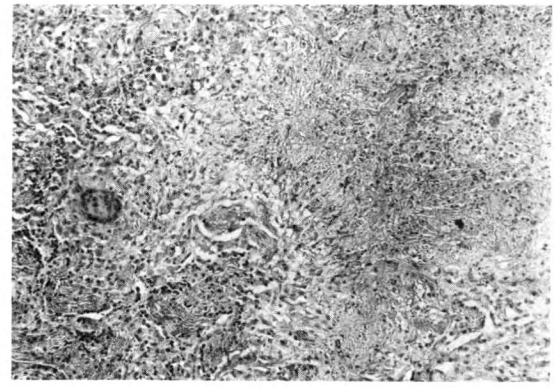

Fig. 6. Case 1. Necrosis of lung parenchyma at right side of field with serpiginous border which is marginated by palisading histiocytes (center). On the left a multinucleated giant cell is noted. (HPS×160.)

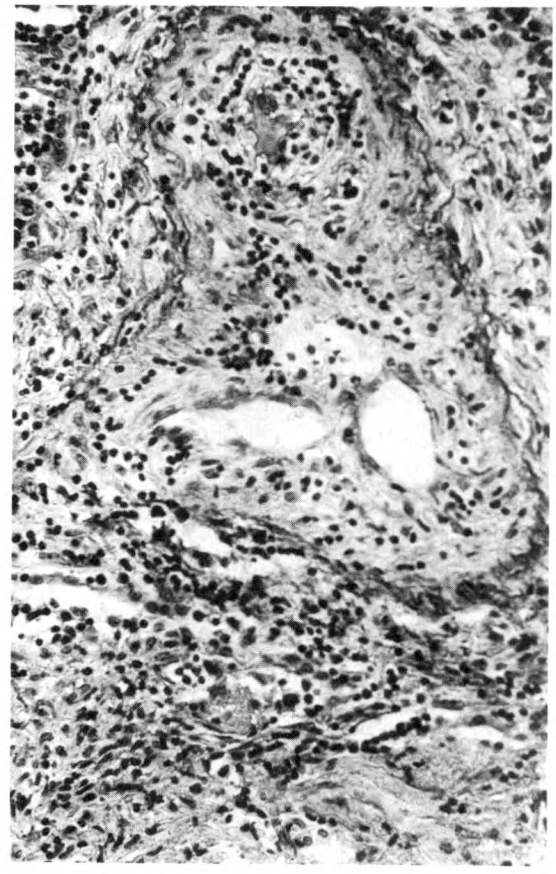

Fig. 7. Case 1. A small branch of pulmonary artery showing mural infiltration by round cells, fibrous intimal proliferation with marked narrowing of lumen. The elastic lamella (wavy black lines) is fragmented. (Verhoeff elastic tissue stain×256.)

FIG. 8. Case 1. Perivascular fibrosis is noted in the upper part of the field and a noncaseating granuloma in the lower portion. (HPS×40.)

eosinophilic infiltration of the muscular coats of some of the large arteries. In the recently reported case of Isenberg *et al.*[2] the chest roentgenogram showed diffuse bilateral pulmonary infiltrates. Microscopic examination of the biopsy specimen disclosed diffuse vasculitis involving small vessels with fibrinoid necrosis and dense masses of acute inflammatory cells, eosinophils, and areas of necrosis, but no granulomata.

Specific infections have been ruled out by cultures, histochemical studies, and the clinical course. The pathologic differential diagnosis includes infarction, Boeck's sarcoid, allergic and granulomatous vasculitis, and rheumatoid lung disease. Pulmonary infarctions are not associated with necrotizing vasculitis or granulomas. Boeck's sarcoid should not show such massive areas of necrosis or evidence of vasculitis. However, noncaseating granulomas of the sarcoid type can occur in approximately 20 per cent of the lesions in the limited form of Wegener's granulomatosis.[3] Allergic and granulomatous vasculitis reported by Churg and Strauss[1] should not be associated with large areas of parenchymal necrosis. The degree of eosinophilic participation in the lungs in our cases is much less than one would expect in that entity. Also peripheral eosinophilia is characteristic, while this is not so in the limited form of Wegener's granulomatosis and was not seen in our patients. Hypersensitivity angiitis lacks granulomata; the affected vessels are all at the same stage of the disease, unlike the cases under discussion. The roentgenographic picture is extensive and often resembles Loeffler's pneumonia. Although both patients averred an allergy to sulfonamides none was administered to them for at least 2 years prior to the onset of the lung lesions. Rheumatoid lung disease often has a significant pleural component; the necrosis of rheumatoid nodules is not so extensive as seen in our cases. The degree of vasculitis in our cases is greater than would be expected in rheumatoid disease. Neither of our patients had evidence of rheumatoid

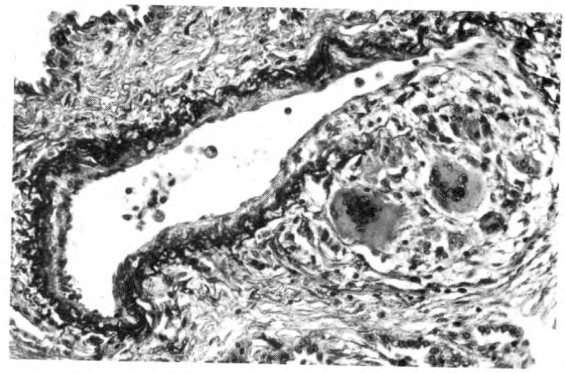

FIG. 9. Case 1. Small artery showing sectoral loss of elastic membrane (right center) corresponding to the site of a noncaseating granuloma. (Verhoeff ×400.)

arthritis clinically, although Case 1 did have a mildly positive test for rheumatoid factor.

In Liebow's series[3] of the limited form of Wegener's granulomatosis the most consistent feature was an abnormal chest roentgenogram. The lesions were fairly discrete, multiple, and bilateral in two-thirds of his cases. Pulmonary symptoms and signs consisted of cough, dyspnea, pleurisy or pleural effusion. Systemic signs of fever, weight loss, and malaise were frequent. Both our cases fulfill the criteria necessary for making a diagnosis of the limited form of Wegener's granulomatosis, *viz.*, a combination of infarct-like areas of necrosis, vasculitis involving small arteries and veins seen at varying stages of development and the presence of granulomata, at least some of which are separate from vessels. The absence of renal involvement would put it in the category of the "limited form" rather than the classical, full-blown picture of Wegener's granulomatosis. In addition, the bacteriologic studies were negative. Both the preoperative diagnosis and the impression on examination of the gross specimen (Fig. 10) in Case 1 was that of neoplasm, a common error in Wegener's granulomatosis in the lungs.

The major considerations in the differential diagnosis are lesions generally thought to be based on derangements of the immune system, *viz.*, rheumatoid lung disease, allergic and granulomatous vasculitis, and hypersensitivity angiitis. Our 2 cases suggest for the first time a possible association between ulcerative colitis and a limited form of Wegener's granulomatosis. Both conditions may be immunologic derangements, both may respond to immunosuppressive therapy, and both may be associated with pyoderma gangrenosum, erythema nodosum, or other skin lesions.

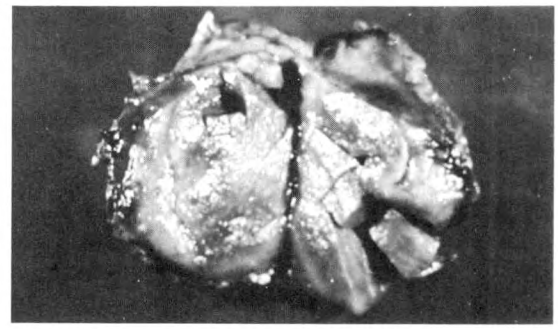

Fig. 10. Case 1. Gross photograph of resected lesion from lower lobe of right lung. Note resemblance to a neoplasm. The mass measures 4 cm. in vertical diameter.

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We wish to thank Drs. B. Aftalion and J. I. Berkman, Pathologists, Long Island Jewish Hospital, for lending pathologic material, Dr. Kent Ellis for critical review of this manuscript, Dr. Thane Asch for his advice and encouragement, and Mrs. J. Spivack and Mrs. Helen Sauer for their technical assistance.

REFERENCES

1. Churg, J., and Strauss, L. Allergic granulomatosis, allergic angiitis and periarteritis nodosa. *Am. J. Path.*, 1951, *27*, 277–301.
2. Isenberg, J. T., Goldstein, H., Korn, A. R., Ozeran, R. S., and Rosen, V. Pulmonary vasculitis: uncommon complication of ulcerative colitis. *New England J. Med.*, 1968, *279*, 1376–1377.
3. Liebow, A. A. Pulmonary angiitis and granulomatosis. *Am. Rev. Resp. Dis.*, 1973, *108*, 1–18.
4. Wasserman, F., Krosnick, A., and Tumen, H. Necrotizing angiitis associated with chronic ulcerative colitis. *Am. J. Med.*, 1954, *17*, 736–743.

PSEUDO-RETROPERITONEAL GAS IN RUPTURE OF ANEURYSM OF ABDOMINAL AORTA*

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ABSTRACT:

Four cases of ruptured abdominal aortic aneurysm producing retroperitoneal radiolucent shadows are presented.

Gross and microscopic examination showed this to be due to dissection of blood through the retroperitoneal fat.

This finding has been helpful in confirming the presence of retropertoneal hemorrhage.

EARLY diagnosis of rupture of the abdominal aorta is of vital importance. By outlining the calcified aneurysm and the mass obliterating the psoas and renal shadows, Grayson and Kennedy[4] were able to make the diagnosis of a rupture of abdominal aortic aneurysm in 3 cases. Crane[3] concluded that plain films of the abdomen were of extreme importance in verifying the diagnosis of leaking abdominal aortic aneurysms. Janover[5] diagnosed 18 of 20 cases of ruptured abdominal aortic aneurysm by the plain roentgen-ray findings of a mass with hazy indistinct margins and extension of the mass beyond the calcified aortic rim. The triad of pain, pulsating abdominal mass, and hypotension was emphasized by Tsai *et al.*[6] They also found that the abdominal roentgenogram often gives confirmatory evidence of the rupture, and in 6 of 30 cases a paravertebral calcification alerted them to a palpable mass not previously felt. Since the diagnosis is imperative for prompt surgical treatment, it is the purpose of this article to bring attention to an additional finding of retroperitoneal radiolucent shadows of blood dissecting the fatty tissues encountered in 4 cases of rupture of abdominal aortic aneurysms.

REFORT OF CASES

CASE 1. A 67 year old obese Caucasian male was admitted to Providence Medical Center for evaluation of right lower quadrant pain and a positive blood culture for *Clostridium perfringens*. A roentgenogram of the abdomen revealed residual barium in the colon from a previous examination, a deformed cecum, and a large collection of radiolucent bubbles in the left retroperitoneal space (Fig. 1). This was interpreted as a carcinoma of the cecum and retroperitoneal gas. An infusion pyelogram showed adequate renal function and a laterally displaced left kidney. A water soluble contrast study of the upper gastrointestinal tract revealed no ulcer or perforation. After transfer

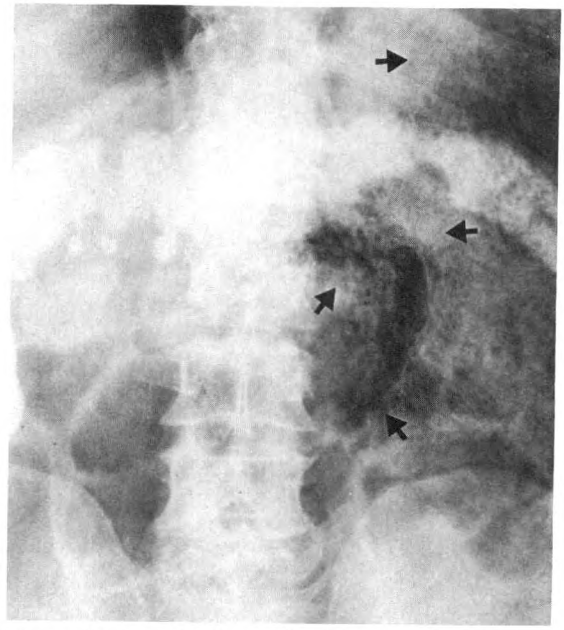

FIG. 1. Case 1. Abdominal roentgenogram demonstrates many radiolucent bubbles in left retroperitoneal area and a larger pseudo-gas shadow in the psoas muscle region.

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to the intensive care ward, the patient went into extreme shock and expired. The admission blood culture was negative, but the urine culture revealed *E. coli*, 100,000 bacteria per cc. and *Streptococcus faecalis* with over 100,000 organisms per cc. At autopsy a 7×4 cm. abdominal aortic aneurysm was found which had ruptured with severe hemorrhagic infiltration into the left retroperitoneal space. There was also a carcinoma of the cecum, but no perforated viscus or retroperitoneal gas was found.

Comment. This case illustrates the difficulty in diagnosing a ruptured aneurysm in an obese individual. The confusing radiolucent shadows led one to believe there was either a visceral perforation or an infection from a gas forming organism. Aortic calcifications were not seen, but a mass was suspected. The radiolucent shadows were discovered to be blood dissecting into the retroperitoneal fat and separation of fat into globules simulating gas bubbles in the plain roentgenogram.

Case II. A 56 year old markedly obese male was admitted to Providence Medical Center for abdominal distention and discomfort of 4 days' duration. No palpable abdominal mass was found, but tenderness was present in the left flank. The initial abdominal roentgenograms revealed a distended colon, but no mass or calcification was seen. Eighteen hours later an intravenous pyelogram demonstrated a laterally displaced left kidney and ureter with radiolucent areas throughout (Fig. 2). Because a rupture of an aneurysm of the abdominal aorta was suspected, an aortogram was performed

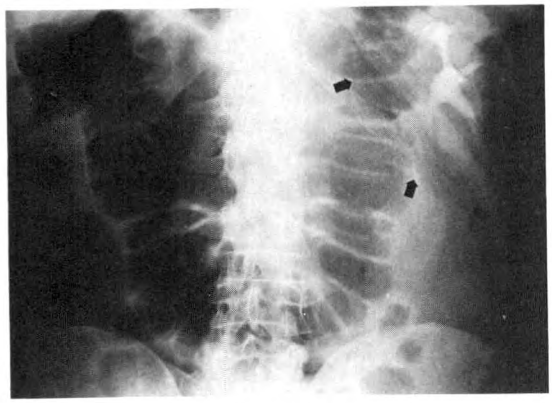

Fig. 2. Case II. The intravenous pyelogram shows many faint radiolucent streaks lateral to the left psoas shadow. The left kidney is also displaced laterally.

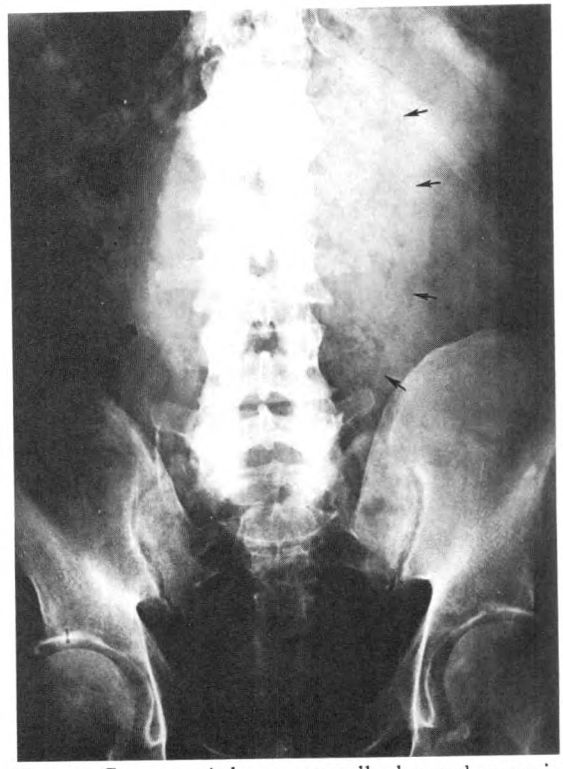

Fig. 3. Case III. A large centrally located mass is present. A calcified aortic rim is seen to the right of the spine, and many radiolucent bubbles are seen to the left of the spine.

confirming the presence of an abdominal aortic aneurysm with a soft tissue mass extending beyond the dilated lumen. Surgery was performed, and the ruptured aneurysm was replaced with a woven Dacron bifurcation graft. The patient recovered and was discharged in 3 weeks.

Comment. This patient had a slowly leaking aneurysm. Although he had clinical evidence of a rupture of an aneurysm, his roentgenograms were not conclusive for 18 hours after admission. The degree of hemorrhage was great, but it was not readily appreciated because of the patient's obesity. The aortogram was not required for the diagnosis; however, it did aid the surgeon in evaluating the extent of the aneurysm.

Case III. A 71 year old white male was admitted to Woodland Park Hospital, Portland, Oregon, with abdominal and left flank pain following a fall at home. The patient was in severe shock. Abdominal roentgenograms showed a calcified rim in a large aortic aneurysm with a mass obliterating the left psoas shadow. In

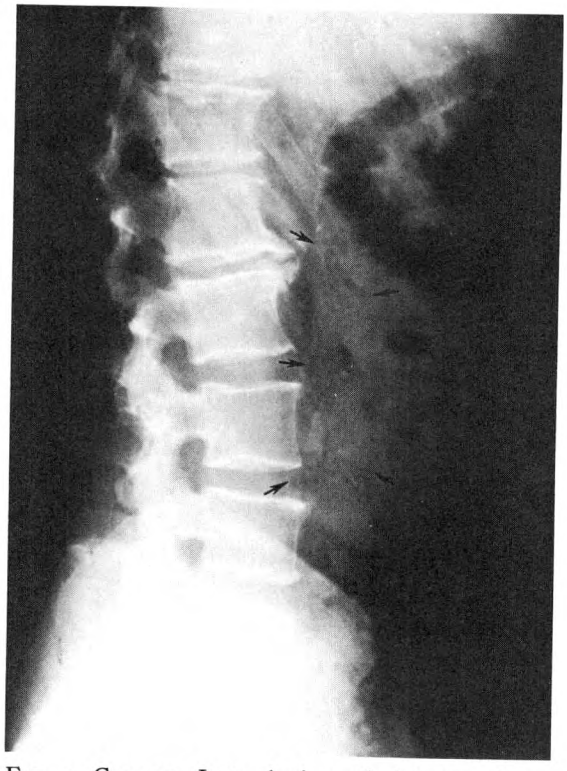

FIG. 4. Case III. Lateral view of abdomen shows radiolucent shadows extending anterior to the lumbar vertebrae along the course of the abdominal aorta and distally into the pelvis.

addition, radiolucent shadows were present throughout the hematomatous mass (Fig. 3; and 4). The diagnosis of ruptured aortic abdominal aneurysm was made, and he was operated on immediately. A Dacron woven bifurcation graft was placed from the abdominal aorta below the renal arteries to the iliac arteries. Following surgery the patient became oliguric and was transferred to Providence Medical Center for hemodialysis. In spite of hemodialysis and supportive treatment, he expired from a cardio-respiratory arrest 6 days later. Autopsy revealed massive retroperitoneal hemorrhage.

Comment. The plain roentgenograms demonstrated the extensive hemorrhagic infiltration of the para-aortic structures. The bubbles of fat surrounding the blood were easily seen extending about the wall of the abdominal aorta and into the adjacent retroperitoneal space. The calcified aortic wall bulged to the right of the lumbar spine while the leak was to the left. The radiolucent pseudo-gas shadows were most prominent in the preoperative roentgenograms, but they persisted following surgery. An additional hemorrhage occurred to cause death.

CASE IV. A thin 57 year old male was admitted to Providence Medical Center because of vague abdominal pains of 1 day's duration. He had had multiple medical problems dating back to a bilateral carotid endarterectomy done 4 months previously. His abdomen was slightly distended and no mass was palpable. He had diffuse generalized arteriosclerosis. The abdominal pain became more severe, and a plain roentgenogram of the abdomen revealed a thin linear calcification in the wall of a small abdominal aneurysm with faint radiolucent shadows in the left psoas muscle area (Fig. 5). This was interpreted as leakage of the aneurysm. The patient expired while he was being transferred from the stretcher to bed. The autopsy revealed a ruptured abdominal aortic aneurysm with massive retroperitoneal hemorrhage, as well as severe generalized arterial atherosclerosis.

Comment. Although this man was very thin and had only a short history of abdominal pain, his plain abdominal roentgenograms showed faint radiolucent shadows in the left paraverte-

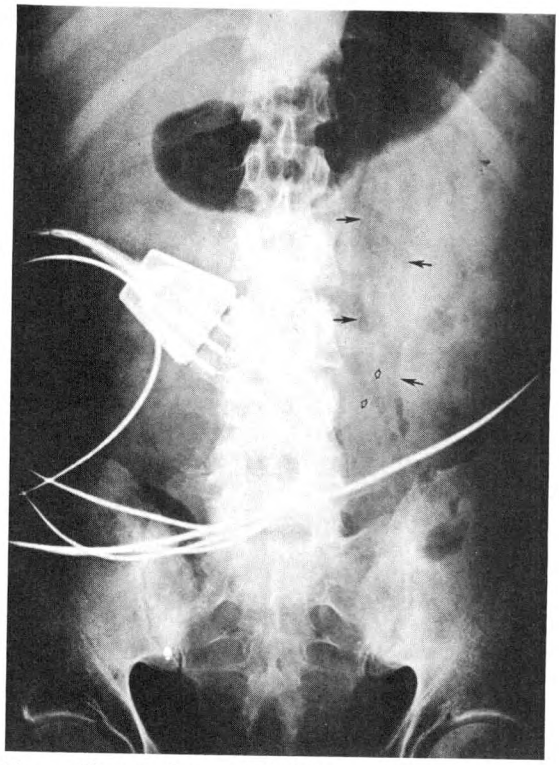

FIG. 5. Case IV. Large radiolucent streaks are seen in the left psoas area (black arrows). A faint calcified aortic rim (open arrows) runs through the radiolucent shadows.

bral area. The faint linear calcification to the left of the lumbar spine called attention to the aneurysm, and the pseudo-gas shadows in the retroperitoneal area indicated the leakage of the blood vessel.

DISCUSSION

The retroperitoneal space is covered by a thin layer of fat which is more prominent in obese patients and is most abundant about the kidneys. When retroperitoneal hemorrhage occurs from rupture of the abdominal aorta, the blood will often dissect along the fascial planes of the psoas muscles and then distally along the iliac vessels, sometimes extending into the groin, and may present as an inguinal hernia.[2] At operation or autopsy it is sometimes noted that the blood dissects in sheets through the retroperitoneal fat giving a variegated appearance to the hemorrhage. This is also seen microscopically (Fig. 6; and 7). Because of the greater density of the blood, the fat will cause a relatively radiolucent bubble on the roentgenogram. A similar finding is seen in cases of hemorrhagic pancreatitis with fat necrosis.[1]

Because it is difficult to distinguish the small air bubbles of a retroperitoneal abscess from small fat globules in the retroperitoneal space, the above cases were presented to alert physicians to the additional possibility that a ruptured abdominal aneurysm may produce radiolucent shadows in the retroperitoneal space. This finding may also be helpful in obese patients with abdominal pain when the abdomen is difficult to palpate. Having incorrectly diagnosed this finding as a gas forming infection in Case 1, the next 3 cases showing this finding were correctly interpreted.

Fig. 6. Case IV. Histologic section of the retroperitoneal hematomatous mass shows hemorrhagic infiltration through the retroperitoneal fat.

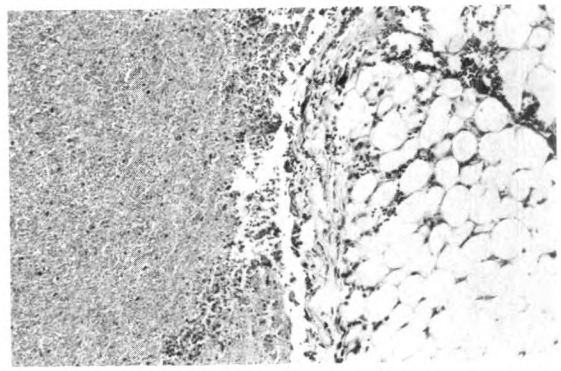

Fig. 7. Case IV. Magnified view of the same specimen showing the contrasting fat globules adjacent to the hemorrhage.

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The authors wish to express their gratitude to Dr. Ray F. Friedman of Portland, Oregon for Case III, Dr. Joel M. Shilling for pathologic interpretation, and Fred H. Lemcke for photographic assistance.

REFERENCES

1. Berenson, J. E., Spitz, H. B., and Felson, B. Abdominal fat necrosis sign. *Radiology*, 1971, *100*, 567–571.
2. Blum, L., and Kark, A. E. Ruptured aneurysm of abdominal aorta simulating incarcerated inguinal hernia. *Am. J. M. Sc.*, 1966, *252*, 97–107.
3. Crane, C. Arteriosclerotic aneurysm of abdominal aorta: some pathological and clinical correlations. *New England J. Med.*, 1955, *253*, 954–958.
4. Grayson, C. E., and Kennedy, B. R. Roentgen diagnosis of ruptured aneurysm of abdominal aorta. *Radiology*, 1950, *54*, 413–416.
5. Janover, M. L. Ruptured arteriosclerotic aneurysms of abdominal aorta: roentgenographic findings on plain films. *New England J. Med.*, 1961, *265*, 12–16.
6. Tsai, Y. W., Blodgett, J. B., Wilson, G. S., Lucas, R. J., and Tumacder, O. C. Ruptured abdominal aortic aneurysm: pathognomonic triad. *Vasc. Surg.*, 1973, *7*, 232–237.

JEJUNOILEAL BYPASS*

ROENTGENOGRAPHIC OBSERVATIONS

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ABSTRACT:

Jejunoileal bypass procedures may be performed with the bypassed small intestine draining, as usual, into the cecum, or with end-to-end anastomosis of the jejunum and terminal ileum, with the bypassed segment draining into the transverse colon through another anastomosis. Modifications of these methods have also been devised. After good response with weight loss initially, most patients fail to continue to lose weight into the third year postoperatively. Some of the features which cause failure are: prolonged transit time, intestinal dilatation with mucosal hypertrophy, and reflux into the bypassed segment, in the earlier type of operation.

SURGICAL treatment for morbid obesity has been attempted, but generally with poor or temporary results. Procedures such as gastroplasty, stimulation of hypothalamic feeding centers and abdominal panniculectomy,[1,5,8,13] have been completely abandoned in many centers, while others still occasionally use them. In the last decade, intestinal shunting procedures have become increasingly popular. They are being accepted in many institutions, including ours, as a relatively safe and efficient means of treatment. They are performed when the obesity remains life threatening, and only after all other medical forms of treatment have been tried without success.

The purpose of this presentation is twofold: (1) to describe the common types of bypass procedures and their roentgenologic appearance; and (2) to familiarize the radiologist and clinician with the important roentgenographic observations in the follow-up examinations that correlate and explain the success or failure of such procedures.

RATIONALE AND DESCRIPTION OF OPERATIVE PROCEDURES

Experimental animal investigations[14] and empirical[18] observations have shown that by reducing the absorptive surface of the small bowel (chronic intestinal diseases or surgical resections following trauma, infarctions, etc.) rapid weight loss follows, which sometimes leads to death. However,

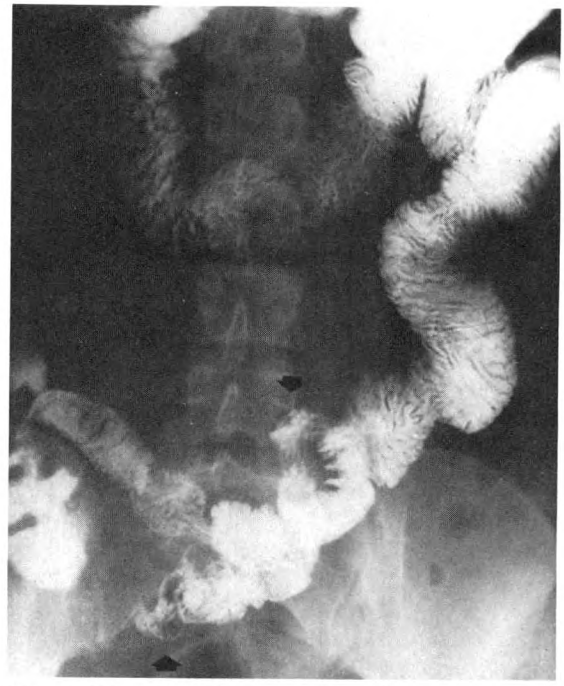

FIG. 1. Jejunoileal bypass, end-to-side anastomosis, Payne procedure. Small degree of reflux into the nonfunctioning ileum (arrows). Note dilatation of functioning bowel and prominent mucosal folds.

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to achieve a negative caloric balance almost the entire small bowel should be excluded, as the preservation of any significant segment, particularly of the jejunum, is tolerated well by the patients.

First attempts at shunting procedures date back to the early sixties when end-to-side jejunocolic anastomoses were tried.[5,7,19] While rapid weight reduction was accomplished, there was a great number of complications, including death, mainly due to irreversible liver injury.

Attempts at bypass procedures were again reported in the mid-sixties but most of them with discouraging or even catastrophic results.[3,12] In 1967, Morgan and Moore[9] reported an individual case treated by a new surgical technique. Using a similar technique in 1969, Payne and DeWind[11] reported their experiences with 80 obese patients. Their procedure is as follows: The jejunum is divided 14 inches from the ligament of Treitz and anastomosed end-to-side to the distal ileum 4 inches before the ileocecal valve (Fig. 1). The proximal end of the bypassed intestine is closed and left in the abdomen to drain freely into the terminal ileum. The functioning small bowel loop thus created is about 18 inches long. Encouraging results were reported with an average postoperative weight loss of 130 pounds. Most of the weight loss occurred in the first 2 years, significantly less in the third year with a tendency to reach a plateau afterwards. A promising response and sometimes excellent control of diabetes, exertional dyspnea, edema, and hypertension, and a significant decrease in the serum cholesterol levels were noted by this surgical technique. The Payne and DeWind procedure offered the means of a permanent reduction in body weight, and was well accepted by the patients.

Nevertheless the procedure was subse-

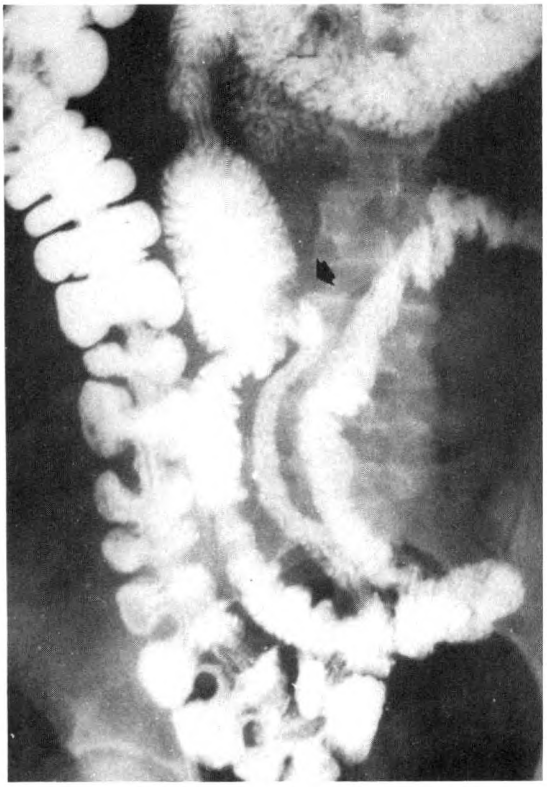

FIG. 2. Jejunoileal bypass, Payne procedure, 60 minute film. Large amount of reflux into the defunctionalized ileum visualizing long ileal segment. Arrow indicates the area of anastomosis. Note the dilated jejunum proximally.

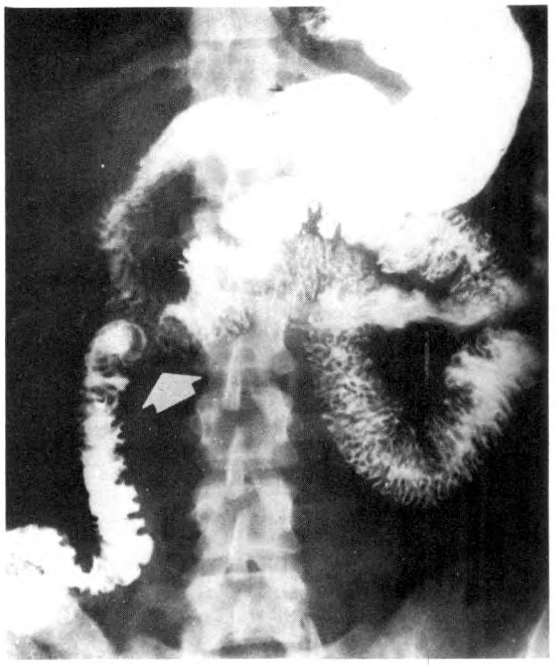

FIG. 3. Jejunoileal bypass, end-to-end anastomosis, Scott procedure. Note length and size of functioning bowel. There is marked hypertrophy of the ileal folds resembling a jejunal loop. Arrow indicates the area of anastomosis.

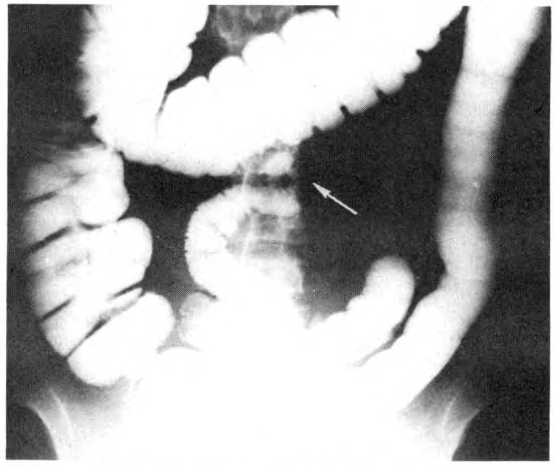

FIG. 4. Barium enema film, Scott procedure with ileo-transverse colon anastomosis. A segment of the defunctionalized ileum fills from transverse colon (arrow).

quently criticized. Animal experiments and clinical experience accumulated by other investigators using the Payne and DeWind procedure[14,15] showed that the results could be erratic. Many patients failed to lose adequate amounts of weight, or after a promising beginning, regained weight. Most of these patients when examined roentgenologically, exhibited reflux of intestinal contents into the bypassed ileum for distances of 3 to 5 feet (Fig. 2). Surgical revision of some of these anastomoses was performed leading to excellent results.

For these reasons Scott *et al.*[15] in 1971 described a new technique of intestinal shunting which created an end-to-end anastomosis (Fig. 3). The jejunum is divided 12 inches distal to the ligament of Treitz and the ileum is divided 12 inches proximal to the ileocecal valve. The proximal jejunum is anastomosed end-to-end to the distal ileum. The proximal end of the bypassed small bowel is closed and its distal end is anastomosed for draining purposes to the transverse or sigmoid colon (Fig. 4; and 5). Significant reflux of intestinal contents into the nonfunctional loop is therefore prevented. Good results were claimed, because of the following gradual and steady weight loss.

A number of other surgical techniques which represent slight modifications to the above described procedures have since been attempted. They are beyond the scope of this presentation.

DISCUSSION

Follow-up examinations in cases of intestinal shunting procedures have consistently shown[11,15] a tendency of rapid weight loss in the immediate postoperative period. This is followed by a period of a less dramatic response, and eventually in the third year by a failure to lose additional weight. The explanation of this phenomenon lies in the gradual anatomic changes

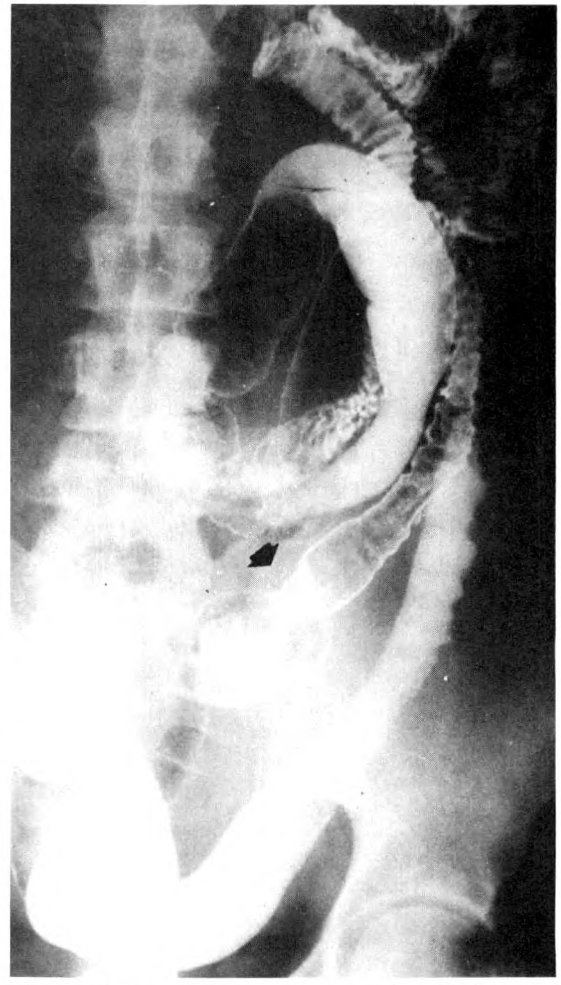

FIG. 5. Barium enema post-evacuation film in a patient who had the Scott procedure with an ileal sigmoid colon anastomosis. A long segment of ileum is visualized due to reflux from the sigmoid colon (arrow).

taking place in the functioning small bowel as a response to the new physiologic stimulus.

There are significant enlargement of the small bowel and hypertrophy of the mucosal folds which are demonstrated roentgenographically (Fig. 3). These findings, together with elongation of the functioning gut, have been substantiated by surgical measurements and histologic examinations.[4] The most impressive changes take place in the terminal ileum where because of massive mucosal hypertrophy so-called jejunization occurs (Fig. 6). One of our patients who underwent a second laparotomy for a segmental ileal resection clearly showed on histologic examination, hypertrophic villi and an increased number of goblet cells.

Secondary to this gradual increase of the intestinal mucosal surface and with the significant increase in the absorption of nutrients, the body weight stabilizes or occasionally starts climbing again. It is impossible to predict for a given individual the degree and rapidity of small bowel hypertrophy and, therefore, the exact length of functional gut required for an ideal weight. In cases where the patient's weight plateaus rapidly at high levels, resection of an additional segment of small bowel is indicated to obtain a satisfactory result.

Fig. 6. Jejunoileal bypass visualizing terminal ileum. Note hypertrophy of the mucosal folds with jejunization of ileum.

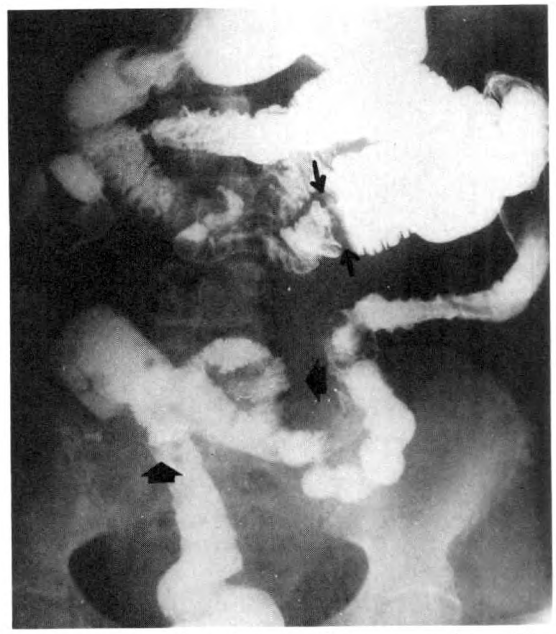

Fig. 7. Jejunoileal bypass, Scott procedure. The anastomotic site (small arrows) is narrowed and there is marked dilatation of the pre-anastomotic jejunal segment. The entire colon fills with barium and a small segment of ileum is visualized because of reflux through ileo-sigmoidostomy (large arrows).

Roentgenologic evaluation of the gastrointestinal tract before and after a bypass procedure is performed routinely. Follow-up examinations are required only when there is an unsatisfactory clinical course.

Several features are looked for and reported:

1. Transit time from pylorus to cecum is, in our experience, 5 to 10 minutes. We consider the mouth to colon transit time as well as the emptying time of small bowel meaningless, since they are conditioned by the rapidity of gastric emptying.

2. Approximate length of functioning small bowel, degree of dilatation, mucosal hypertrophy and type of anastomosis should be evaluated, particularly when comparison studies are available. Obviously, exact measurements are impossible and only general impressions are obtained. Occasionally, the anastomosis may be nar-

rowed (Fig. 7), causing markedly distended pre-anastomotic jejunal loops explaining the patient's postoperative complaints.

3. One must look for reflux into bypassed ileum. A small degree of reflux of 3 to 4 inches is usually seen in the Payne and DeWind end-to-side anastomosis (Fig. 1) and should be considered normal. Longer segments, particularly when they do not empty rapidly, are significant and explain the unsatisfactory results of such procedures (Fig. 2). In the Scott *et al.* procedure (end-to-end anastomosis), reflux was never a problem in our experience. Small segments of ileum were occasionally visualized in the antegrade study (Fig. 7) and longer segments were seen only with barium enema examinations (Fig. 4; and 5).

CONCLUSIONS

Although still controversial, intestinal bypass procedures are being performed as the only efficient means for controlling morbid obesity and its complications.

The rationale, short description of common types of shunting procedures, and their roentgenographic appearance are presented.

The most important roentgenographic observations seen in the follow-up examinations, which correlate with the success or failure of these procedures, are discussed.

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REFERENCES

1. BELIN, R. P., STONE, N. H., FISCHER, R. P., and Scott, W. R. Improved technique of paniculectomy. *Surgery*, 1966, *59*, 222–225.
2. BUCHWALD, H., and VARCO, R. L. Partial ileal bypass for hypercholesterolemia and atherosclerosis. *Surg., Gynec. & Obst.*, 1967, *124*, 1231–1238.
3. DEMUTH, W. E., JR., and ROTTENSTEIN, H. S. Death associated with hypocalcemia after small bowel short circuiting. *New England J. Med.*, 1964, *270*, 1239–1240.
4. DOWLING, R. H., and BOOTH, C. C. Functional compensation after small bowel resection in man: demonstration by direct measurement. *Lancet*, 1966, *2*, 146–147.
5. HIRSCH, J. Jejunoileal shunt for obesity: Editorial. *New England J. Med.*, 1974, *290*, 962–963.
6. HOLZBACH, R. T., WIELAND, R. G., LIEBER, C. S., DECARLI, L. M., KOEPKE, K. R., and GREEN, G. S. Hepatic lipid in morbid obesity: assessment at and subsequent to jejunoileal bypass. *New England J. Med.*, 1974, *290*, 296–299.
7. LEWIS, L. A., TURNBULL, R. B., and PAGE, I. H. Short circuiting of small intestine. *J.A.M.A.*, 1962, *182*, 77–79.
8. MEYEROWITZ, B. R., GRUBER, R. P., and LAUB, D. R. Massive abdominal panniculectomy. *J.A.M.A.*, 1973, *225*, 408–409.
9. MORGAN, A. P., and MOORE, F. D. Jejunoileostomy for extreme obesity. *Ann. Surg.*, 1967, *166*, 75–82.
10. MOXLEY, R. T., POZEFSKY, T., and LOCKWOOD, D. H. Protein nutrition and liver disease after jejunoileal bypass for morbid obesity. *New England J. Med.*, 1974, *290*, 921–926.
11. PAYNE, J. H., and DEWIND, L. T. Surgical treatment of obesity. *Am. J. Surg.*, 1969, *118*, 141–147.
12. PAYNE, J. H., DEWIND, L. T., and COMMONS, R. R. Metabolic observations in patients with jejunocolic shunts. *Am. J. Surg.*, 1963, *106*, 273–289.
13. PRINTEN, K. J., and MASON, E. E. Gastric surgery for relief of morbid obesity. *A.M.A. Arch. Surg.*, 1973, *106*, 428–431.
14. SALMON, P. A. Results of small intestine bypass operations for treatment of obesity. *Surg., Gynec. & Obst.*, 1971, *132*, 965–979.
15. SCOTT, H. W., JR., SANDSTEAD, H. H., BRILL, A. B., BURKO, H., and YOUNGER, R. K. Experience with new technique of intestinal bypass in treatment of morbid obesity. *Ann. Surg.*, 1971, *174*, 560–571.
16. SCOTT, H. W., JR., and LAW, D. H. Clinical appraisal of jejunoileal shunt in patients with morbid obesity. *Am. J. Surg.*, 1969, *117*, 246–253.
17. SCOTT, H. W., JR., LAW, D. H., SANSTEAD, H. H., LANIER, V. C., and YOUNGER, R. K. Jejuno-ileal shunt in surgical treatment of morbid obesity. *Ann. Surg.*, 1970, *171*, 770–780.
18. WINAWER, S. J., BROITMAN, S. A., WOLOCHOW, D. A., OSBORNE, M. P., and ZAMCHECK, N. Successful management of massive small bowel resection based on assessment of absorption defects and nutritional needs. *New England J. Med.*, 1966, *274*, 72–78.
19. WOOD, L. C., and CHREMOS, A. N. Treating obesity by short circuiting small intestine. *J.A.M.A.*, 1963, *186*, 63.

VOL. 125, NO. 1

COMPARATIVE DETECTION OF PANCREATIC BODY—TAIL ENLARGEMENT USING THE SUPINE TRANSLATERAL AND AXIAL PANCREATIC VIEWS: A PROSPECTIVE STATISTICAL STUDY*

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ABSTRACT:

A prospective statistical study of 429 patients was undertaken to determine if the axial pancreatic view would improve detection of pancreatic body-tail enlargement. This view is produced by directing a roentgen-ray beam along the axis of the pancreatic body-tail cylinder. On this view and on the supine translateral view, a pancreatic body-tail space can be identified as a subdivision of the retrogastric space. When the pancreatic body-tail enlarges, this space selectively enlarges. The percentage change of this space is particularly impressive on the axial pancreatic view, where 10 of 12 pancreatic body-tail neoplasms were detected with only a 1.8 per cent false positive rate.

THIS communication documents the improved accuracy of detecting enlargement of the body and tail of the pancreas using the axial pancreatic view. A prospective statistical study of 429 patients using both the supine translateral and the axial pancreatic views was carried out. In each view, the determination of enlargement was based exclusively on the quantitative analysis of the pancreatic body-tail space, a roentgenographically well demarcated subdivision of the retrogastric space. These results were then compared with results of an independent evaluation of the conventional upper gastrointestinal examination and determinations of the antrum-to-spine distance on the supine translateral view.

The qualitative signs most commonly cited as predictive of enlargement of the body and tail of the pancreas are local impressions and invasion of the stomach and ligament of Treitz, downward displacement of the ligament of Treitz, and anterior displacement of the stomach.[5] Of these, the most reliable sign is a localized extrinsic tumor impression on the stomach.[1,4]

A popular parameter for evaluating pancreatic enlargement is the antrum-to-spine distance.[2,4,6,7,8] This distance is heavily dependent on body habitus[9,11] and is most reliable in the left lateral erect position.[11] The best technique for measuring this parameter has been described by Poole,[6] who uses a very small amount of barium, fluoroscopically applied antral compression, and corrections for both body habitus and roentgenographic magnification. The major limitation of this method is that it is valid only for enlargement of the midline pancreatic neck and body, and only when the antrum is in the midline overlying the pancreas.[7]

Quantitative parameters of enlargement of the body and tail to the left of the midline have received little attention in the literature. Herbert and Margulis[4] described the "coronal plane posterior gastric body-to-spine distance." This measurement is derived from frontal and lateral

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.
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erect views and theoretically represents the true posterior gastric body-to-spine distance in the coronal plane. They noted that the average measurement in normals was twice that of abnormals, suggesting an unexpected medial component of gastric displacement. These distances, however, were insufficiently dissimilar to permit differentiation in the individual case.

The initial observation leading to the quantitative approach developed in this study was made by Hajdu.[3] He showed that the distal pancreatic body-tail was located at the base of a triangular space between the posterior gastric wall and ligament of Treitz on the supine translateral view. Seaman *et al.*[10] reasoned that masses in the pancreatic body-tail should therefore separate the gastric fundus from the ligament of Treitz.

In a cadaver roentgenographic demonstration, Sorabella *et al.*[12] showed that the long axis of the pancreatic body-tail cylinder was neither in the transverse nor coronal planes and, therefore, this organ cylinder may be completely obscured in some patients by the barium filled stomach in the supine translateral view. They devised a new projection, the axial pancreatic view, in which the central roentgen-ray beam coincided with the long axis of the pancreatic cylinder. In this view the pancreatic body-tail appeared in a barium-free space, the pancreatic body-tail space. In clinical trials, they noted that in this view the identification of the ligament of Treitz was facilitated and was occasionally the only view to show local gastric impressions or quantitative enlargement of the pancreatic body-tail space. Their initial appraisal was that the space was enlarged if a 3.5 cm. circle could be entirely contained within it.

MATERIAL AND METHOD

Case material for this study was drawn from patients referred for upper gastrointestinal examinations at Columbia-Presbyterian Medical Center. One group was composed of patients suspected of having pancreatic disease. A second group included randomly selected patients who were having barium meal examinations for reasons other than suspected pancreatic disease.

Supine translateral and axial pancreatic views in deep inspiration and deep expiration, together with a deep expiratory supine frontal view were obtained on all patients. The patient's abdominal anteroposterior dimension was recorded by the technician.

The technique for obtaining the axial pancreatic view is as follows:

1. The patient is given 8 ounces of a barium suspension and observed fluoroscopically until the barium reaches the ligament of Treitz.
2. He is placed supine with his left side against a vertical cassette.
3. The foot of the x-ray table is displaced away from the cassette so that the cassette and the patient's long axis form a 25° angle.
4. The patient's left side is elevated 20°.
5. A horizontal, 120 kvp, beam is directed perpendicular to the cassette, centering on L_2 and using a 40 inch focal-film distance.

The conventional upper gastrointestinal series were interpreted with the benefit of a clinical history by 2 of the authors (W.L.C., W.B.S.). Independently, 1 author (W.L.C.) analyzed the pancreatic body-tail space measurements of the 2 translateral and 2 axial pancreatic views without knowledge of the patient's name or clinical history. With these measurements, he calculated the pancreatic body-tail space index (*vide infra*). The frontal view was used only to help identify the ligament of Treitz on the supine translateral view.

The examinations were reviewed approximately 3 months after they were obtained. Clinical follow-up continued for an average of 6 months.

Finally, antrum-to-spine measurements were taken from expiratory supine translateral views on a randomized series of 100 normal patients with a body habitus distribution resembling the whole population.

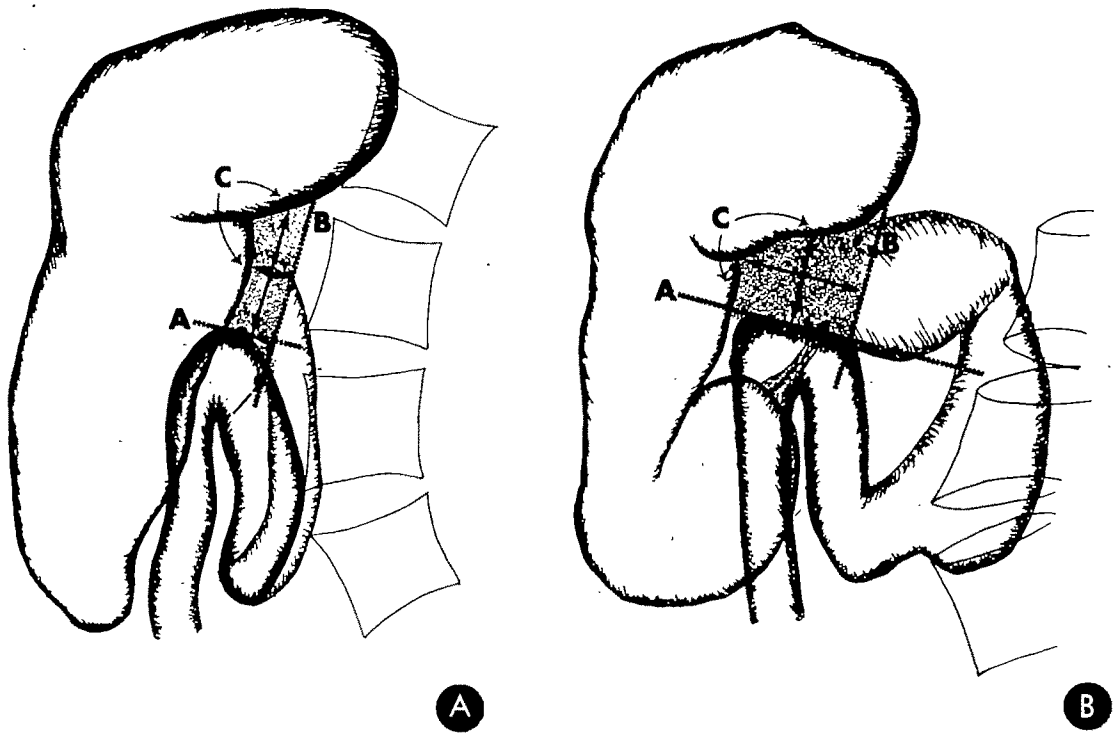

Fig. 1. The pancreatic body-tail space. (*A*) Supine translateral view. (*B*) Axial pancreatic view. Lines A, B, and C define the space; see text regarding their placement. The perpendicular arrows within the space represent the cephalocaudal and anteroposterior dimensions of the space. The multiplied product of these 2 dimensions is the pancreatic body-tail space index.

Similar measurements were made on all patients with pancreatic neoplasms, pseudocysts, ascites, and acute pancreatitis. This measurement was made according to the method described by Poppel *et al.*[7] and Hajdu.[8]

The pancreatic body-tail space indices for the 2 views, the antrum-to-spine measurements, and the results of the conventional upper gastrointestinal examination were analyzed using conventional multiphasic statistical methods, in order to determine the predictive value of each parameter.

THE PANCREATIC BODY-TAIL SPACE INDEX

The quantitative parameter evaluated for detecting pancreatic body-tail enlargement on the supine translateral and axial pancreatic views is the pancreatic body-tail space index. In both views it is obtained as follows:

1. The ligament of Treitz (duodenojejunal flexure) is identified. A line (line A in Figure 1, *A* and *B*) is drawn through the cephalic margin of the ligament of Treitz perpendicular to the anterior curvature of the spine.

2. The posterior surface of the ligament of Treitz is derived by assuming that the anteroposterior diameter is the same as the easily measured cephalocaudal diameter. Through this posterior point a line (line B) is drawn parallel to the anterior surface of the spine.

3. The posterior gastric body and fundus form a line (line C) which, with the previous 2 lines, encloses a space, the pancreatic body-tail space.

4. The anteroposterior and cephalocaudal dimensions of the space are measured through the middle of the space.

5. Early in our investigation it became apparent that the cephalocaudal dimension

(fundus to ligament of Treitz) was a far more reliable criterion for detecting enlargement than the uncorrected anteroposterior dimension. Disproportionate elongation of the anteroposterior dimension in the face of a normal cephalocaudal dimension was found not to reflect pancreatic enlargement, but probably a collection of redundant transverse mesocolon, colon, and small bowel loops.[13] Nevertheless, this dimension is of some value, for when it is quite small in the presence of a long cephalocaudal dimension, it suggests that no pathologic enlargement is present. Therefore, the following weighted convention was adopted. *If the anteroposterior dimension exceeds the cephalocaudal dimension by more than 5 mm., the accepted anteroposterior dimension is determined by adding 5 mm. to the cephalocaudal dimension.*

6. The pancreatic body-tail space index is the product obtained by multiplying the cephalocaudal and the corrected anteroposterior measurements together. This index is a rough approximation of the pancreatic cylinder cross sectional area. This simplification was selected over planimetric determination of the space's area in order to increase the likelihood of acceptance of the method by practicing radiologists.

RESULTS

Effect of respiratory phase and projection on the pancreatic body-tail space index. The mean and standard deviations of the pancreatic body-tail space index in 266 patients without evidence of pancreatic disease were calculated for the inspiratory and expiratory supine translateral and axial pancreatic views. There were no statistically significant differences on the 4 views in normal patients (Table I). The upper limit of normal was taken to be 14 cm., which is greater than 2 standard deviations above the mean in all views. The distributions of the index for all 4 views were skewed toward the left (Fig. 2, *A* and *B*).

Interestingly, the mean and standard deviations of the index in the 4 views in patients with retrocolic Billroth II gastrojejunostomies appear comparable to the normal population, although the sample size was insufficient for statistical validation. This is not unexpected since the landmarks which establish the index are unaltered: that is, the posterior gastric fundic wall is still in intimate contact with the pancreatic body-tail cylinder, and the ligament of Treitz remains unaltered. This is not the case for antecolic anastomoses where the colon is interposed between the pancreas and the stomach.

Effect of body habitus and projection on the pancreatic body-tail space index. Body habitus, as determined by the abdominal anteroposterior dimension, has significantly less effect on the pancreatic body-tail space index than on the antrum-to-spine distance. The index in the supine translateral view is more affected by body habitus than in the axial pancreatic view (Table II; and Fig. 3, *A* and *B*). Nevertheless, in both projections body habitus is relatively insignificant as compared to the effects of enlargement of the body and tail of the pancreas, and can be ignored in the assessment of pancreatic body-tail size.

Reproducibility of the pancreatic body-tail space index. Twenty-three patients, both normals and abnormals, were roentgenographed on 2 separate occasions. The intervals between examinations ranged from 1 day to nearly 1 year. In none of the

TABLE I

EFFECT OF RESPIRATORY PHASE AND PROJECTION ON THE PANCREATIC BODY-TAIL SPACE INDEX IN CLINICALLY NORMAL PATIENTS

View	Mean ($cm.^2$)	Standard Deviation ($cm.^2$)
Inspiratory supine translateral	4.3	4.3
Inspiratory axial pancreatic	4.6	4.6
Expiratory supine translateral	4.5	4.3
Expiratory axial pancreatic	4.3	3.9

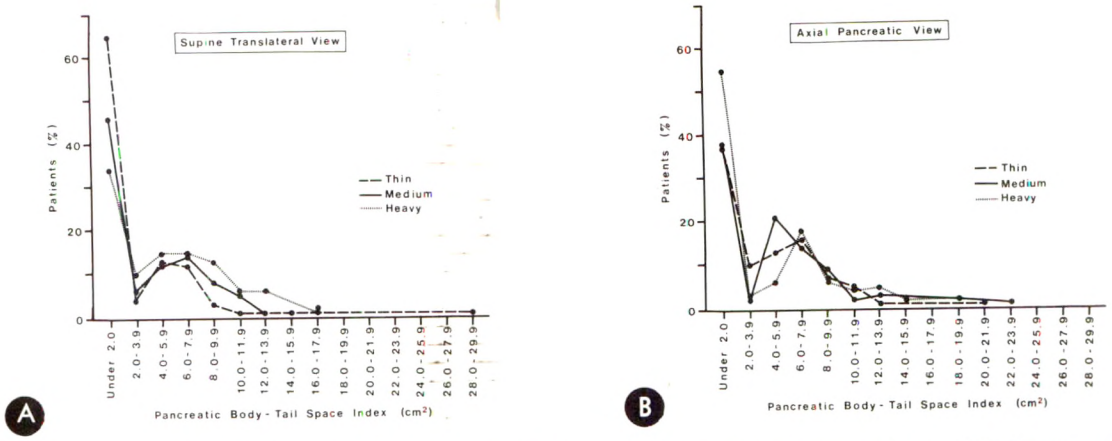

Fig. 2. The distribution of the pancreatic body-tail space index in 266 normal patients. (*A*) Expiratory supine translateral view. (*B*) Expiratory axial pancreatic view. All indices 14 cm.² or larger are greater than 2 standard deviations above the mean. The solid bars, therefore, represent the false positive results (9 patients in the supine translateral view, and 5 patients in the axial pancreatic view).

Table II

Effect of Body Habitus and Projection on the Pancreatic Body-Tail Space Index in Clinically Normal Patients

Anteroposterior Dimension	Expiratory Supine Translateral View		Expiratory Axial Pancreatic View	
	Mean (cm.²)	Standard Deviation (cm.²)	Mean (cm.²)	Standard Deviation (cm.²)
Thin (below 21 cm.)	3.0	2.9	4.0	3.3
Medium (21–24 cm.)	5.0	5.0	4.4	4.0
Heavy (over 24 cm.)	5.3	3.9	4.5	4.3

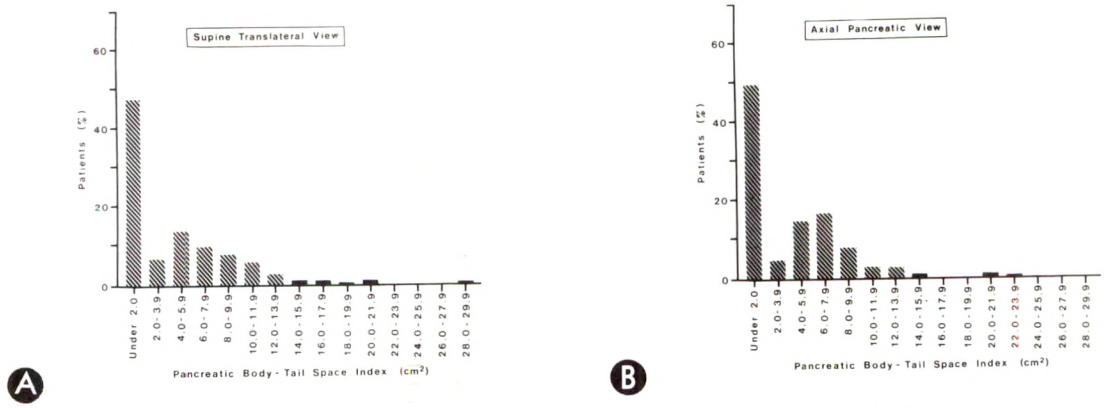

Fig. 3. The effect of body habitus on the pancreatic body-tail space index. (*A*) Supine translateral view. (*B*) Axial pancreatic view.

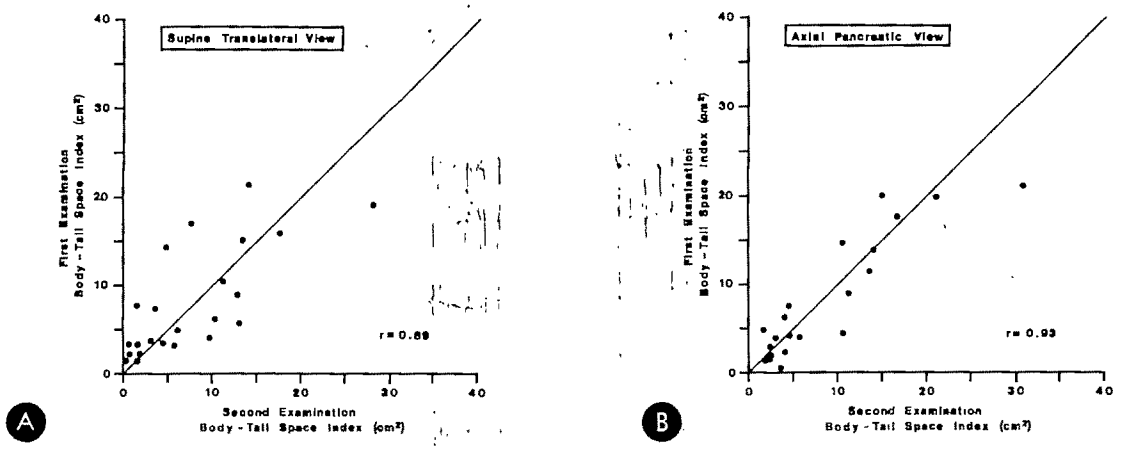

FIG. 4. Reproducibility of the pancreatic body-tail space index on 2 separate examinations. (*A*) Supine translateral view. (*B*) Axial pancreatic view. The coefficients of correlation (r) are indicated on the graphs.

cases was there evidence to suggest that a change in pancreatic size was likely in the interval. The 2 examinations were interpreted independently of one another. The distributions of the resultant indices were plotted for the expiratory supine translateral and axial pancreatic views (Fig. 4, *A* and *B*). The Pearson product moment coefficient, which is a quantitative measure of reproducibility, was calculated. This coefficient was 0.89 for the supine translateral view, indicating good reproducibility, and 0.93 for the axial pancreatic view, indicating excellent reproducibility. Despite the positional variation inherent in taking the axial pancreatic view, the resulting fluctuations in the index are surprisingly insignificant. Furthermore, the amount of barium in the stomach and the use of hypotonic agents seem to have an insignificant effect on the index.

The clinically abnormal populations. The clinically abnormal population was divided into 5 groups: (I) pancreatic neoplasms; (II) acute pancreatitis; (III) pancreatic pseudocysts; (IV) ascites; and (V) miscellaneous diseases.

All the neoplasms in Group I were proved surgically or at autopsy, both with respect to histology and location in the pancreas. All neoplasms were carcinomas except for 1 malignant histiocytoma and 1 secretin secreting adenoma. Group I was further divided into 3 subgroups: (I-A) involving the pancreatic body-tail or diffusely infiltrating the entire gland (12 cases); (I-B) involving the pancreatic body in the midline (2 cases); and (I-C) involving the head of the pancreas (9 cases).

Group II consisted of 92 patients with pancreatitis. Forty patients (Group II-A) were roentgenographed within 2 weeks of the onset of symptoms and had a documented serum amylase level over 100 Mayers-Killian units (normal less than 35). The other 52 patients (Group II-B) comprised a more heterogeneous group who had had an attack of documented pancreatitis, but did not fulfill the strict criteria of Group II-A. This heterogeneous subgroup consisted mainly of patients with clinically resolved acute pancreatitis as defined in the Marseilles classification.[8]

Group III consisted of 3 patients with pancreatic pseudocysts in the body-tail, documented ultrasonographically.

Group IV included 17 patients with massive ascites on physical examination. Early in our investigation it became apparent that these patients exhibited an enlarged pancreatic space, possibly because of fluid collecting in the lesser sac.

Group V was composed of 28 patients with miscellaneous upper abdominal masses or anatomic anomalies—conditions which

Table III

ABILITY OF THE PANCREATIC BODY-TAIL SPACE INDEX TO DIFFERENTIATE PANCREATIC BODY-TAIL ENLARGEMENT FROM THE NORMAL SIZE PANCREAS

Group	Diagnosis	Abnormal Pancreatic Body-Tail Space Index		
		Total Patients	Supine Translateral View	Axial Pancreatic View
I-A	Pancreatic body-tail neoplasms*	11	6	9
I-B	Carcinoma of the midline pancreatic body	2	0	0
I-C	Pancreatic head carcinoma	9	0	0
II-A	Acute pancreatitis	40	15	22
II-B	Pancreatitis	52	12	13
III	Pancreatic pseudocysts	3	3	3
IV	Ascites	17	7	11
	Normal patients	266	9 (3.3 per cent)	5 (1.8 per cent)

* In a twelfth patient, a carcinoma was detected on the axial pancreatic view, but this was excluded from the series since no supine translateral view was obtained.

might be expected to alter the landmarks of the pancreatic space.

Ability of the pancreatic body-tail space index to discriminate between normal patients and those with pancreatic body-tail enlargement. The pancreatic body-tail space indices for the expiratory supine translateral and axial pancreatic views were analyzed for their ability to discriminate between normal patients and those with pancreatic body-tail enlargement. All indices of 14 cm.^{2} or larger were considered abnormal. The results are shown in Tables III and IV. The axial pancreatic view showed more cases of carcinoma of the pancreatic body-tail (9 *versus* 6), acute pancreatitis (22 *versus* 15) and ascites (11 *versus* 7) than the supine translateral view and with fewer false positives (1.8 per cent *versus* 3.3 per cent). Both views detected the 3 large pan-

Table IV

PANCREATIC BODY-TAIL SPACE INDEX IN PATIENTS WITH MISCELLANEOUS UPPER ABDOMINAL MASSES OR ANATOMIC ANOMALIES (GROUP V)

Condition	Total Patients	Abnormal Supine Translateral View	Abnormal Axial Pancreatic View
Carcinoma of biliary system	3	0	0
Carcinoma of stomach	1	0	0
Retro-pancreatic lymphoma	3	0	0
Retro-pancreatic metastases	3	0	2
Unspecified retrogastric mass	1	1	1
Splenomegaly	6	3	2
Transverse mesocolic abscess	1	0	0
Transverse mesocolic hematoma	1	1	1
Upper abdominal aortic aneurysm	2	0	0
Lesser sac abscess	1	1	0
Calcified pancreatic head cyst	1	1	0
Massive left hydronephrosis	1	0	1
Multiple small bowel resections	1	0	1
Intestinal malrotation	3	1	1
Total	28	8	9

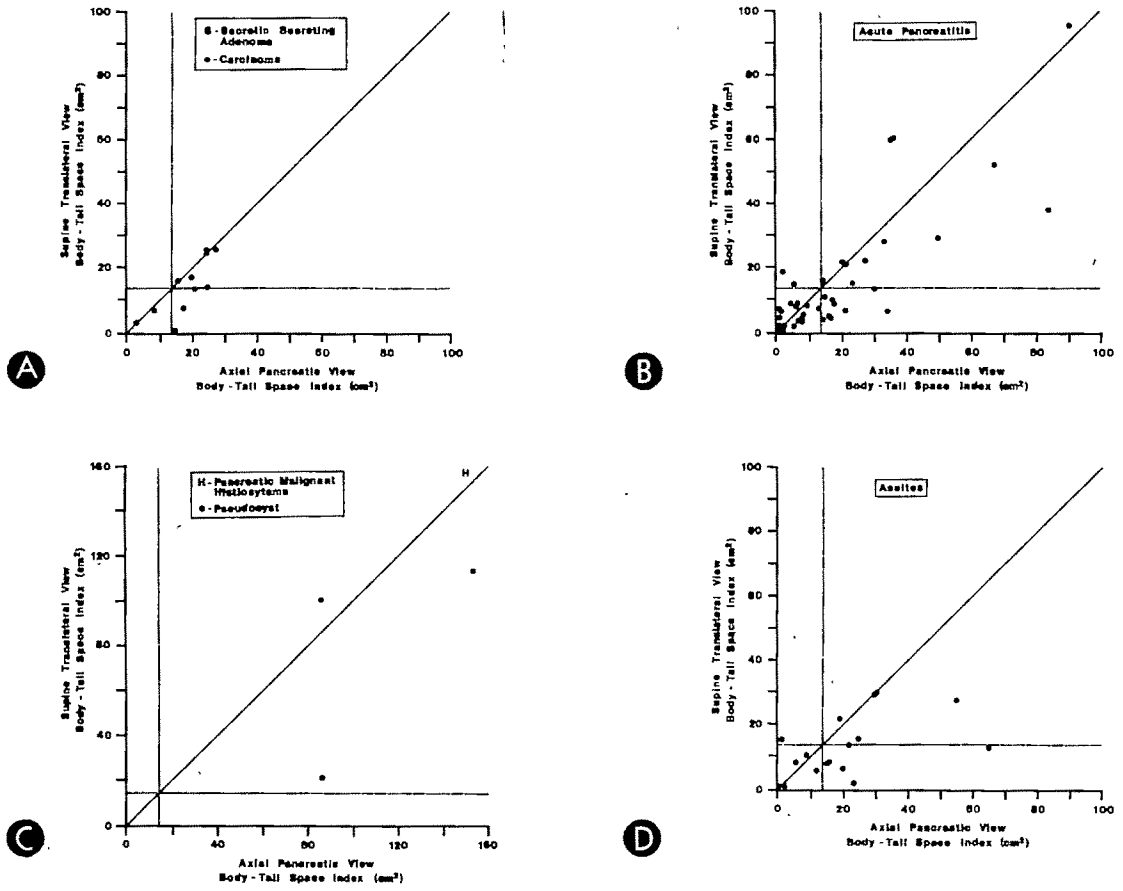

FIG. 5. Ability of the pancreatic body-tail space index to detect enlargement of the pancreatic body-tail. (*A*) Neoplasms (Group I-A). (*B*) Acute pancreatitis (Group II-A). (*C*) Pseudocysts (Group III), and a pancreatic histiocytoma. (*D*) Ascites (Group IV). The dotted lines represent the upper limits of normal for each view. Note that very large masses are detected with equal ease in the supine translateral and axial pancreatic views (*C*). In *A*, *B*, and *D*, there are many patients whose indices fall near the abscissa just to the right of the dotted vertical line. These patients are those whose degree of body-tail enlargement was moderate and, therefore, detected only on the axial pancreatic view.

creatic pseudocysts and neither view detected carcinoma of the pancreatic head, neck and midline body.

Figure 5, *A–D*, demonstrates plots comparing the indices in the supine translateral and axial pancreatic views for all patients in Groups I-A, II-A, III, and IV. The graphs show that masses producing very large indices (greater than 30 cm.^{2}) in 1 view are likely to produce comparable enlargement in the other view. But in 16 patients, the index calculated from the axial pancreatic view showed mild to moderate elevation (14 cm.^{2} to 30 cm.^{2}), while the supine translateral view remained normal. On the other hand, the supine translateral view showed elevations of the index in only 3 patients having normal indices on the axial pancreatic view.

Comparative discrimination of the pancreatic body-tail space index and the antrum-to-spine distance in the supine translateral view. In the supine translateral view, the ability of the pancreatic body-tail space index to discriminate between normal and enlarged pancreatic body-tails was compared with the results of the antrum-to-spine dimension. The index proved substantially more predictive of the presence of pancreatic body-tail neoplasms, pseudo-

TABLE V

COMPARATIVE ABILITY OF THE PANCREATIC BODY-TAIL SPACE INDEX AND THE ANTRUM-TO-SPINE DISTANCE TO DIFFERENTIATE PANCREATIC BODY-TAIL ENLARGEMENT FROM THE NORMAL SIZE PANCREAS IN THE SUPINE TRANSLATERAL VIEW

Diagnosis	Total Patients	Abnormal Antrum-to-Spine Distance	Abnormal Pancreatic Body-Tail Space Index*
Pancreatic body-tail neoplasms (Group I-A)	11	2	6
Acute pancreatitis (Group II-A)	40	7	15
Pseudocyst (Group III)	3	1	3
Ascites (Group IV)	17	10	7
Normal patients		5/100 (5.0 per cent)	9/266 (3.3 per cent)

* Refers only to the supine translateral view.

cysts and acute pancreatitis, and with a lower false positive rate, 3.3 per cent *versus* 5.0 per cent (Table v). Ascites, on the other hand, was more often detected with the antrum-to-spine dimension. Perhaps this latter observation results from the floating position of the gastric antrum above the ascitic collection in the greater sac.

Comparative accuracy of the axial pancreatic view and the conventional upper gastrointestinal examination. The pancreatic body-tail space index in the axial pancreatic view was significantly more predictive of pancreatic body-tail enlargement than was the conventional upper gastrointestinal series (Table vi). In patient Groups I-A, II-A, and III, the axial pancreatic view detected pancreatic body-tail enlargement in 61 per cent of cases compared to 15 per cent for the upper gastrointestinal series. As expected, the axial pancreatic view was not useful for diagnosing enlargement of the head of the pancreas. Evidence of pancreatic head enlargement was present in 42 per cent of upper gastrointestinal series in Groups I-A, II-A, and III.

Forty-six patients in Groups I-A, II-A, and III underwent both upper gastrointestinal series and axial pancreatic views. In only 2 cases was the axial pancreatic view normal in the face of an abnormal upper gastrointestinal series. Both cases were patients with acute pancreatitis in whom abnormalities of the second portion of the duodenum were seen.

TABLE VI

COMPARATIVE ABILITY TO DETECT ENLARGEMENT OF THE PANCREATIC BODY-TAIL USING THE AXIAL PANCREATIC VIEW VERSUS THE CONVENTIONAL UPPER GASTROINTESTINAL EXAMINATION

Diagnosis	Axial Pancreatic View	Upper Gastrointestinal Series	
		Tail Abnormalities	Head Abnormalities
	No. Per Cent	No. Per Cent	No. Per Cent
Pancreatic body-tail neoplasms (Group I-A)	10/12 (83)	1/12 (8)	5/12 (42)
Pancreatic midline body carcinoma (Group I-B)	0/2	0/2	0/2
Acute pancreatitis (Group II-A)	22/40 (55)	3/31 (10)	12/31 (39)
a. With enlarged index (22 patients)	—	3/19 (16)	10/19 (53)
b. With normal index (18 patients)	—	0/12 (0)	2/12 (17)
Pseudocyst (Group III)	3/3	3/3	3/3

DISCUSSION

This study was undertaken to test our clinical impression that significant improvement in detecting pancreatic body-tail enlargement could be achieved by supplementing the upper gastrointestinal series with the axial pancreatic and supine translateral views. On each view, a "pancreatic body-tail space" may be identified (Fig. 1, *A* and *B*) that corresponds to the cross sectional area of the pancreatic body-tail. This space is reproducible from examination to examination, and its size is little affected by differences in body habitus. Moderate pancreatic body-tail enlargement increases the size of the pancreatic body-tail space, even though the over-all retrogastric space may not be noticeably altered. The axial pancreatic and supine translateral views are most sensitive when taken in full expiration.

The calculation of the pancreatic body-tail space index on the axial pancreatic view is the single most accurate method for detecting pancreatic body-tail enlargement on the barium meal examination. Both the axial pancreatic and supine translateral views are clearly superior to the conventional upper gastrointestinal series in this regard.

A generally accepted observation has been that the upper gastrointestinal series is more sensitive in detecting enlargement of the pancreatic head than the pancreatic body-tail. Our data show that when diffuse pancreatic enlargement is present, the axial pancreatic view is more sensitive for detecting the enlargement of the pancreatic body-tail than is the upper gastrointestinal series for detecting abnormalities of the pancreatic head. And while the 2 studies are complementary, in only 2 of 46 patients did the upper gastrointestinal series reveal evidence of diffuse pancreatic disease when the axial pancreatic view was normal. The axial pancreatic view is also significantly more sensitive than the antrum-to-spine distance, another measurement that has been used to diagnose pancreatic enlargement.

A variety of disease processes were included which might cause enlargement of the retrogastric space. Ascites, splenomegaly, and midgut malrotation often cause an increase in the pancreatic body-tail space index. These conditions can usually be suspected on the supine frontal roentgenogram taken in conjunction with the axial pancreatic and supine translateral views. When ascites, splenomegaly, or midgut malrotation is present, an increased body-tail space index is not a reliable indicator of pancreatic body-tail enlargement.

Diagnostic accuracy was better for neoplasms (10/12 cases) than for acute pancreatitis (22/40 cases). This probably reflects the fact that not all cases of acute pancreatitis are accompanied by significant swelling of the gland. Also, swelling may be transient or focal, and therefore be missed at the time of roentgen examination. Specific pathologic data on the frequency of pancreatic enlargement in acute pancreatitis are not available. In addition, our sample of patients with body-tail neoplasms is admittedly small. The 10/12 detection rate may not remain quite so high as more cases are accumulated. Nonetheless, the superiority of the axial pancreatic and supine translateral views over the conventional upper gastrointestinal series and the antrum-to-spine distance seems well established.

It is currently our policy to obtain a "pancreatic series" on any patient referred for upper gastrointestinal examination for suspected pancreatic disease. This "pancreatic series" consists of axial pancreatic, supine translateral, and supine frontal abdominal roentgenograms. These views, plus conventional or hypotonic examinations of the duodenal sweep, permit the roentgenographic diagnosis of most pancreatic enlargements.

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The authors wish to thank Dr. Andre

Varma of the Columbia University School of Public Health for his assistance with the statistical analysis.

REFERENCES

1. BERANBAUM, S. L., and JACOBSON, H. G. Right angle roentgenography of gastrointestinal tract. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1958, *86*, 933–944.
2. ENGEL, A., and LYSHOLM, E. New roentgenological method of pancreas examination and its practical results. *Acta radiol.*, 1934, *15*, 635–651.
3. HAJDU, N. Role of translateral films in barium meal. *Clin. Radiol.*, 1968, *19*, 33–46.
4. HERBERT, W. W., and MARGULIS, A. R. Diagnosis of retroperitoneal masses by gastrointestinal roentgenographic measurements: computer study. *Radiology*, 1965, *84*, 52–57.
5. MANI, J. R., ZBORALSKE, F. F., and MARGULIS, A. R. Carcinoma of body and tail of pancreas. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 429–446.
6. POOLE, G. J. New roentgenographic method of measuring retrogastric and retroduodenal spaces: statistical evaluation of reliability and diagnostic utility. *Radiology*, 1970, *97*, 71–81.
7. POPPEL, M. H., SHEINMEL, A., and MEDNICK, E. Procurement and critical appraisal of width diameter of midline retrogastric soft tissues. AM. J. ROENTGENOL. & RAD. THERAPY, 1949, *61*, 56–60.
8. SARLES, H. Proposal adopted unanimously by participants of symposium on pancreatitis at Marseilles, 1963. *Bibl. Gastroenterologica*, 1965, *7*, 7–8.
9. SCHULTZ, E. H., JR. Measurements of retrogastric space. *Radiology*, 1965, *84*, 58–65.
10. SEAMAN, W. B., SORABELLA, P. A., and CAMPBELL, W. L. Roentgen detection of enlargement of body and tail of pancreas using supine translateral projection. *Radiology*, 1974, *111*, 529–533.
11. SHEIMMEL, A., and MEDNICK, E. A. Roentgen diagnosis of upper abdominal retroperitoneal space-occupying lesions. AM. J. ROENTGENOL. & RAD. THERAPY, 1951, *65*, 77–92.
12. SORABELLA, P. A., CAMPBELL, W. L., and SEAMAN, W. B. Axial pancreatic view: new approach for recognizing enlargement of body and tail of pancreas. *Radiology*, 1974, *111*, 535–542.
13. WHALEN. J. P., EVANS, J. A., and SHANSER, J. Vector principle in differential diagnosis of abdominal masses: left upper quadrant. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 104–118.

NEW THOUGHTS CONCERNING XANTHOGRANULOMATOUS PYELONEPHRITIS (X-P)*

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ABSTRACT:

A clinical and roentgenographic analysis of 13 patients with pathologically proved xanthogranulomatous pyelonephritis (X-P) has demonstrated that many previously accepted truisms associated with this disease may not be valid. As a result of this study it is suggested that X-P:

1. Does have a predominant female distribution.
2. May arise relatively acutely.
3. Can be associated with a well-functioning kidney.
4. May destroy the kidney and collecting system.
5. Does not demonstrate neovascularity.
6. Can be distinguished angiographically from hypernephroma.
7. May be associated with diabetes.

Other important facts were again observed:

1. X-P is still often associated with staghorn calculi and urinary tract obstruction.
2. *Proteus mirabilis* is the main offending organism.

THE following thoughts and observations have been associated with xanthogranulomatous pyelonephritis (X-P):
X-P:

1. has no predominant sex distribution;
2. is often associated with calculi;
3. occurs as a long standing chronic disease;
4. is associated mainly with a nonfunctioning kidney;
5. does not destroy the collecting system;
6. demonstrates neovascularity as a roentgenographic feature;
7. cannot be distinguished angiographically from adenocarcinoma;
8. is not associated with other diseases; and
9. *Proteus mirabilis* is the main offending organism.

We have demonstrated by analyzing 13 cases of X-P that many of these observations are incorrect.

In this report we will evaluate the findings and roentgenographic features observed in these 13 patients. By so doing we propose to refute certain concepts, support some, and introduce others.

MATERIAL

Of the 13 patients with X-P, 11 were females and 2 were males. Ages extended from 14 to 72 years with the majority older than 40 (Table 1).

A history of urinary tract infection that had lasted from months to years was elicited in most patients. One patient, however presented with an acute infection with no previous history of urinary tract infection. In another case, we were able to document the onset of the infection 1 month prior to operation.

All patients were studied by intravenous

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–29, 1974.
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pyelography and angiography. Retrograde pyelography was performed in 2 patients who demonstrated nonvisualization of the involved collecting system on the excretory urogram. Calculi were present in 8 patients, and in 8 patients (not the same 8) the disease was localized as either a unilocular or multilocular mass. The remaining 5 kidneys were diffusely involved with infection. The diagnosis of X-P was confirmed on pathologic examination following surgical removal of the kidney in all patients except 2. In 1 a biopsy was performed because the disease was too extensive to be removed; it had grown cephalad to involve the diaphragm. In the other patient, the diagnosis of X-P was made angiographically. At operation it was felt that she had cancer, and a frozen section of a biopsy of her kidney was reported as such. She was signed out of the hospital as " . . . renal carcinoma . . . " even though the final sections showed chronic inflammation. She died 2 years later and at necropsy, the diagnosis of X-P was made. The disease had spread to involve her duodenum and diaphragm.

PATHOLOGY

Xanthogranulomatous pyelonephritis is an aggressive infection of the kidney char-

TABLE I

DATA ON 13 CASES

Sex	Age	Calculus	Diabetes	Post-partum	Kidney Involved	Function	Category: A. Local unilocular multilocular B. Diffuse	Organism	Epinephrine Response *—Not done	Comments
O.G. F	55	—	+	+	R	+	multilocular-hypervascular		*	polycystic kidneys
J.K. F	16	—	+	+	R	+	unilocular avascular	*Proteus m.*	*	
K.B. M	14	—	—	—	R	+	unilocular avascular	*E. coli*	*	
B.N. F	25	ureteral	+	—	L	+	unilocular avascular	*Proteus m.*	*	developed cutaneous fistula
L.T. F	33	—	—	—	L	+	unilocular vascular		+	inoperable
A.H. F	55	staghorn	+	—	L	+ (poor)	unilocular hypervascular		+	pre-op. Dx-XP
A.D. M	72	staghorn	—	—	L	+	unilocular hypervascular		*	
W.M. F	37	staghorn	—	—	R	+	multilocular-hypervascular	*Proteus m.*	*	pre-op. Dx-Ca
R.M. F	52	staghorn	—	—	R	+ (poor)	multilocular hypervascular	*Proteus m.*	*	pre-op. Dx-XP
F	64	—	+	—	R	+ (poor)	diffuse-hypervascular		*	1. Pre-op. Dx-XP 2. Biopsy Dx-Ca 3. Autopsy Dx-XP
M.P. F	63	staghorn	+	—	L	+ (poor)	diffuse-hypervascular	*Proteus-Klebsiella*	*	pre-op. Dx-XP
F	28	staghorn	—	—	L	—	diffuse-hypervascular		*	
D.W. F	46	staghorn	+	—	L	—	diffuse-hypervascular	*Proteus m.*	*	
Totals										
11-F 2-M	—	8	7	2	6-R 7-L		6 localized 4 diffuse 3 multilocular		2	

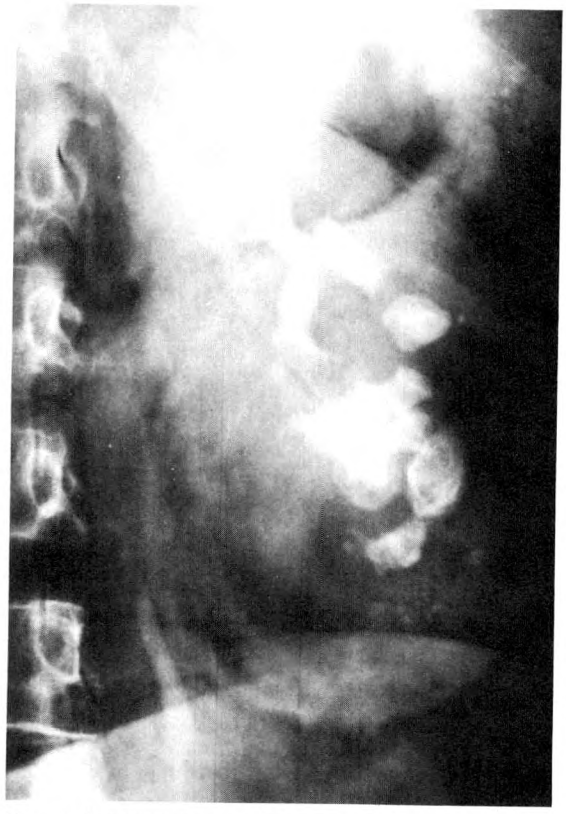

FIG. 1. Intravenous pyelogram reveals good opacification of the superior collecting systems. Inflammatory mass is present within the lower pole containing multiple calculi.

acterized by abscess formation surrounded by inflammatory foam, plasma, and multinucleated giant cells as well as an intense collagenous and fibrous reaction. One can differentiate this form of abscess from others by its yellow color owing to the numerous large lipid contained cells and multinucleated giant cells. The affected kidney is usually enlarged and surrounded by a thick fibrous capsule. Microscopically, abscesses are usually multiple with necrotic centers but become more cellular and fibrotic toward the margin. Immediately adjacent to the necrosis are chronic inflammatory cells; that is, plasma cells, lymph cells, and histiocytes laden with lipid, the so-called xanthoma cells. Peripherally, fibrous tissue and collagenous tissue surround the abscesses; this tissue contains blood vessels. The number of vessels present varies from lesion to lesion. The over-all structural architecture of the vessels appears normal, *e.g.*, the entire wall consists of the usual intima, muscularis, and adventitia. However, these vessels are frequently conglomerate and distorted in appearance, as well as hypertrophied.

ROENTGENOGRAPHIC FEATURES

On excretory urography, we observed enlarged kidneys or mass lesions within the involved kidney. The renal outline was often indistinct because of perirenal inflammatory response. Eight involved kidneys contained calculi, 6 of which were staghorn. In all 8 patients with localized disease, the diseased kidney showed some degree of function (Table 1). In 6, good visualization (function) of the collecting system was present (Fig. 1), whereas the urograms in the remaining 2 showed only poor function. In the part of the kidney that was involved with infection, the calyces were elongated and separated, and occasionally amputated. In 3 of the patients with diffuse involvement, some degree of calyceal opacification was present, although somewhat diminished. Two involved kidneys showed nonfunction. Retrograde pyelograms of these 2 patients showed calyces that were dilated and eroded.

Vascular patterns of the involved kidneys varied according to extent of involvement. We classified the localized lesions as vascular if parenchymal opacification occurred within the lesion during the nephrographic phase, or avascular if no parenchymal opacification was observed. Six of these cases with localized disease were unilocular; 2 were multilocular. In all of these lesions, blood vessels were displaced and rearranged around and within the affected part of the kidney (Fig. 2*A*). The more vascular the lesion, the more distorted the vessels appeared. In general, all the vessels tapered normally in caliber and demonstrated no arteriovenous communications or early venous filling (Fig. 3). While these distorted and rearranged vessels showed an appearance somewhat akin to tumor vascu-

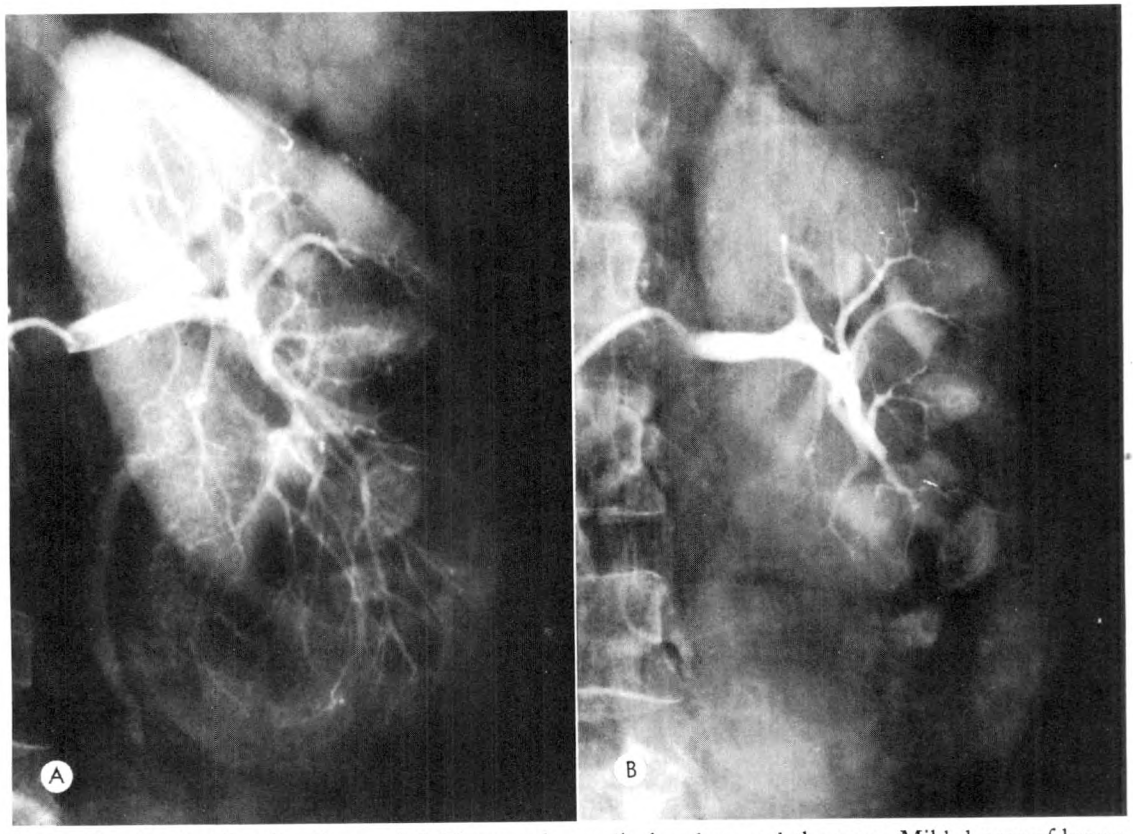

Fig. 2. Localized unilocular disease. (*A*) The vessels are displaced around the mass. Mild degree of hypervascularity is noted. (*B*) Epinephrine study. The vessels supplying infected area are completely contracted.

larity, in most cases we could distinguish between the 2. In 2 patients with localized, unilocular disease, we injected intra-arterial epinephrine. All vessels supplying the affected part of the kidney contracted (Fig. 2*B*). This diagnostic method unfortunately was not utilized in any of the remaining patients. This positive response is contrary to the negative results reported previously.[6]

In all our cases that were vascular, the appearance of the vascularity was in our opinion mostly due to distortion and rearrangement of the normally present vessels. Possibly, additional vessels which are not normally visible enlarge and become visible on the angiograms, probably in response to the increased demand for blood caused by the inflammatory response (Fig. 4).[7]

DISCUSSION

Surprisingly little attention has been given to the question of sex predominance in X-P. Nevertheless, Anhalt *et al.*[1] after reviewing the literature, noted that 70 per cent of the patients were female. The same authors added 4 more patients to the series; all female. Noyes and Palubinskas[8] also noted a slight female preponderance in their series. To further support this, 11 of our 13 patients were female. All of our patients were from general hospitals. The sex distribution is therefore random and not selective. It appears then that women may be more prone to infection with X-P.

The association of X-P and calculi is so common that many consider its presence and accompanying obstruction as necessary features for the diagnosis. The incidence of calculi has been quoted as high as 70 per cent.[1] This finding was present in 8 of our 13 patients (approximately 60 per cent). Most of the calculi (6 of 8) were staghorn, although in 1 patient a stone was lodged

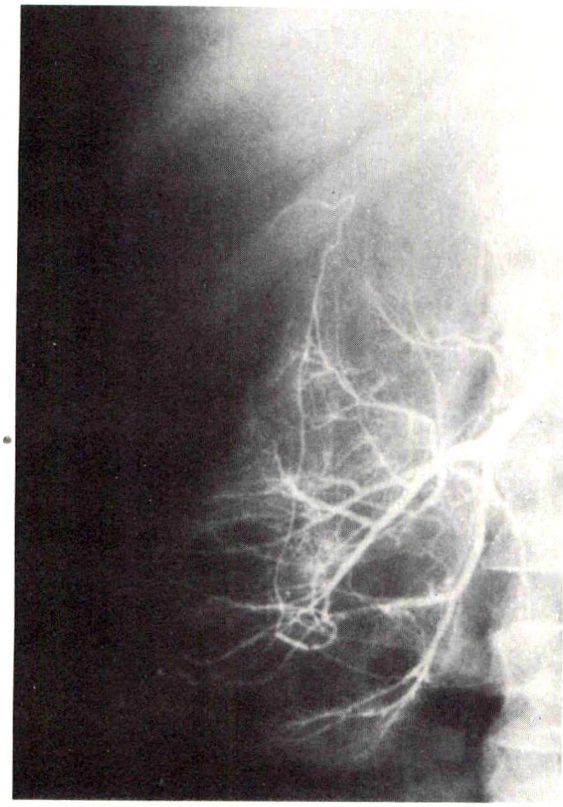

Fig. 3. Diffuse disease. Moderately vascular lesion with multiple vessels coursing somewhat irregularly in direction. The vessels are basically distorted and rearranged and show none of the changes associated with tumor vascularity.

within the proximal ureter and in another, calculi were identified within the lesion itself. There was an apparently increased incidence of staghorn calculi in patients with local involvement (Table 1).

In most cases the evolution of the xanthogranulomatous process developed over periods ranging from months to years. Because of this, it has been believed that this disease originates as a long-standing, low grade infection with subsequent tissue destruction and release of certain lipids that stimulate the histiocytic response.[1] In most of our patients the infection had persisted for many months. One patient with polycystic kidneys was studied clinically and roentgenographically and exhibited no signs or symptoms of urinary tract infection. Three months later she became pregnant and was aborted for medical reasons. Immediately thereafter she developed chills and fever. Excretory urography and angiography showed that the right kidney had enlarged and was slightly more vascular than it was on the study 3 months previously (Fig. 4). Another 16 year old woman developed an acute infection in her right kidney immediately post partum. Both of these patients were diabetic and both kidneys when removed surgically were involved with X-P. Consequently, it is possible that the disease may arise relatively quickly and that other factors beside the age of the infection may be responsible for the development of the disease.

Another feature previously considered highly significant in X-P is a non-functioning kidney.[1–4] This had been described with diffuse as well as local disease. Nevertheless, all 8 of our patients with localized or segmental involvement of the kidney demonstrated little or *no* functional impairment. In fact, the presence of staghorn calculi in 5 of these patients had little effect upon the ultimate function. This seems quite understandable since most subacute or chronic abscess, X-P included, when walled off behave only as space occupying masses.[5] An acute exacerbation of the disease, however, with extension beyond the abscess wall could eventually be responsible for loss of function. Even in the 5 cases with diffuse renal involvement, 3 demonstrated some degree of function. The concept that a non-functioning kidney is a necessary manifestation of xanthogranulomatous pyelonephritis is, in our experience, not valid.

Localized, encapsulated X-P may distort only the calyces without invading the neighboring collecting system. However, in some situations, particularly with diffuse involvement, the disease can erode and destroy the calyces as was demonstrated in our 2 patients on whom retrograde pyelography was performed. Extension beyond the kidney has also been noted, and in 1 of our cases the inflammatory process extended across the midline, involving all the

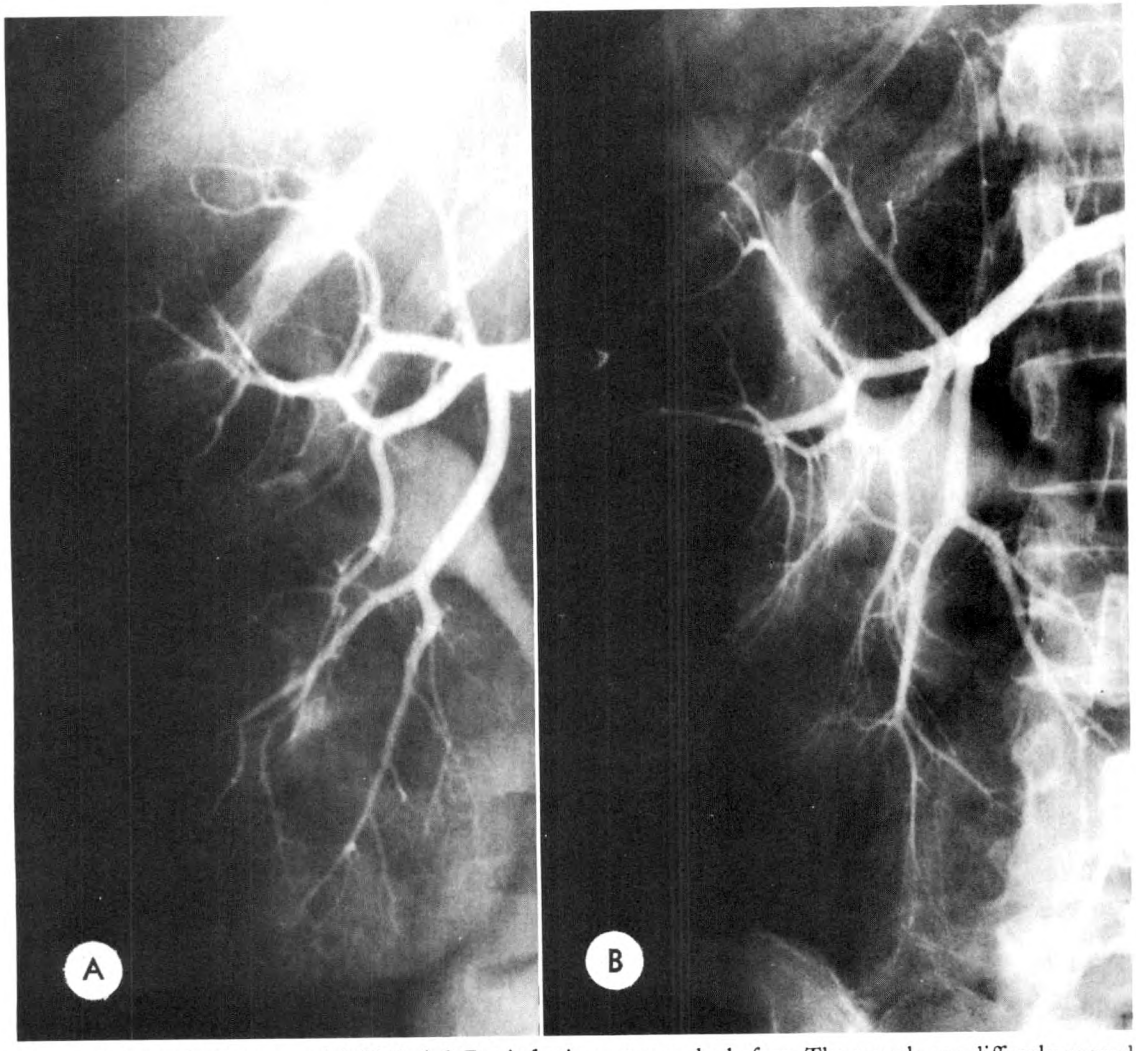

Fig. 4. Polycystic disease—with X-P. (*A*) Pre-infection—2 months before. The vessels are diffusely spread and thinned. (*B*) Post-infection—1 month following onset of renal infection. The kidney is enlarged and the vessels further spread apart and thinned. Additional smaller branch vessels are now apparent.

perirenal structures as well as the diaphragm. In another it invaded the duodenum and diaphragm.

The question of whether other underlying diseases are associated with or are responsible for xanthogranulomatous pyelonephritis has not been previously alluded to. Nevertheless, 7 of our patients were diabetics. This is a surprisingly high incidence. Since diabetics are more prone to infection and suppuration, it is conceivable that X-P may be another complication added to chronic infection in these patients. The underlying changes in lipid metabolism may also contribute to the formation of X-P. Two of our patients with diabetes presented with X-P in the immediate postpartum period. In 1 the course of the disease lasted for only 1 month prior to nephrectomy. This span is short in comparison to that seen in most of our patients and in others recorded in the literature. Although no definitive conclusion can be established in this small series, further investigation regarding the association of the 2 diseases is definitely warranted.

Proteus mirabilis was the most common organism cultured. *E. coli* and *Klebsiella*

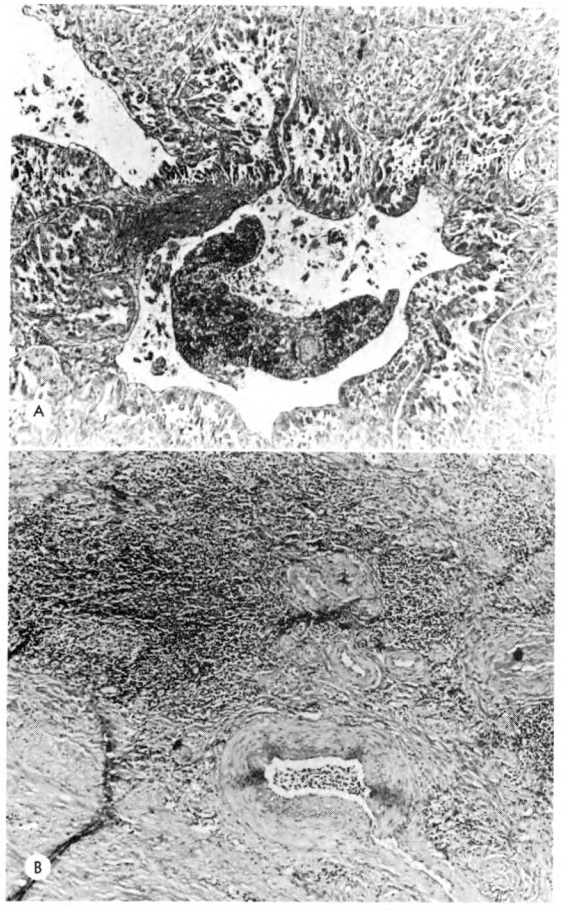

FIG. 5. Photomicrographs showing pathology. (*A*) Tumor vessel cross section in a renal adenocarcinoma consists basically of endothelium surrounded partially by fibrous connective tissue. (*B*) Cross section of a vessel present in X-P demonstrates a normal architecture. The endothelium is surrounded by muscularis and adventitia.

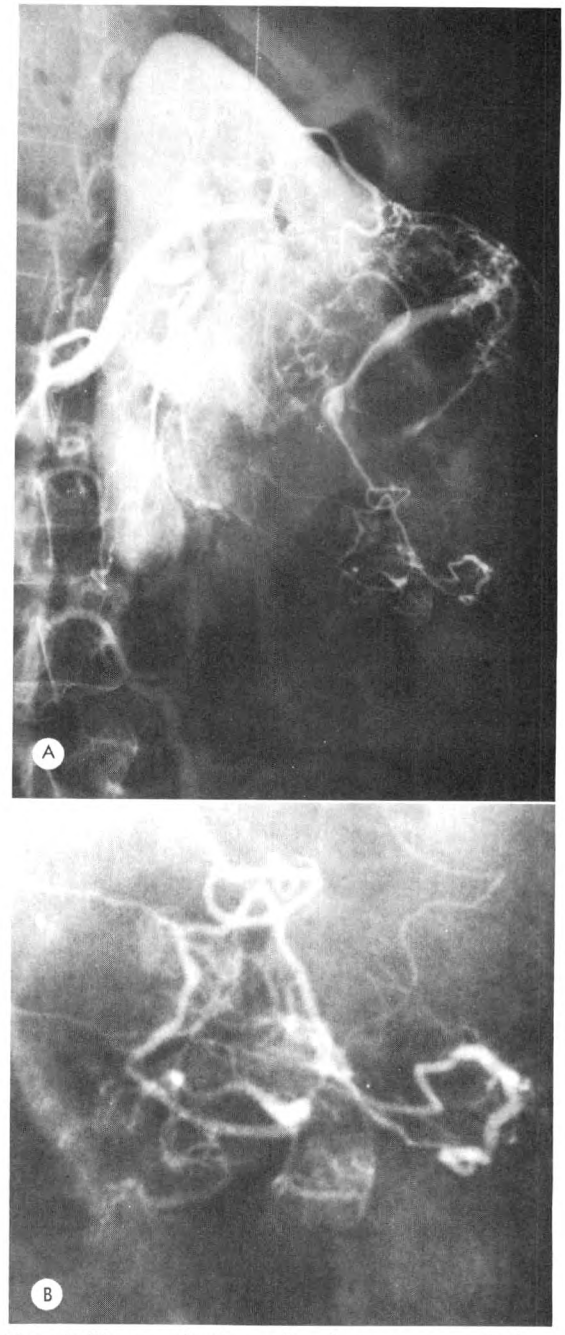

FIG. 6. Tumor. Angiography shows neovascularity. (*A*) Early arteriographic study demonstrates irregular vessels with early venous filling. (*B*) A detailed examination of the tumor vessels reveals extremely tortuous vessels that run in a purposeless direction. The vessels widen and narrow irregularly.

were also obtained. The essential feature in all these patients is a chronic suppuration with an organism of low virulence. *Proteus mirabilis* when injected experimentally into rabbits with elevated lipids has induced xanthogranulomatous response.[1] However, inappropriate antibiotic administration or weak body defenses may add to the problem.

Most authors insist that neovascularity is an integral part of the response of the kidney to X-P. It has also been the general contention of most authors that because of this neovascularity, X-P cannot usually be

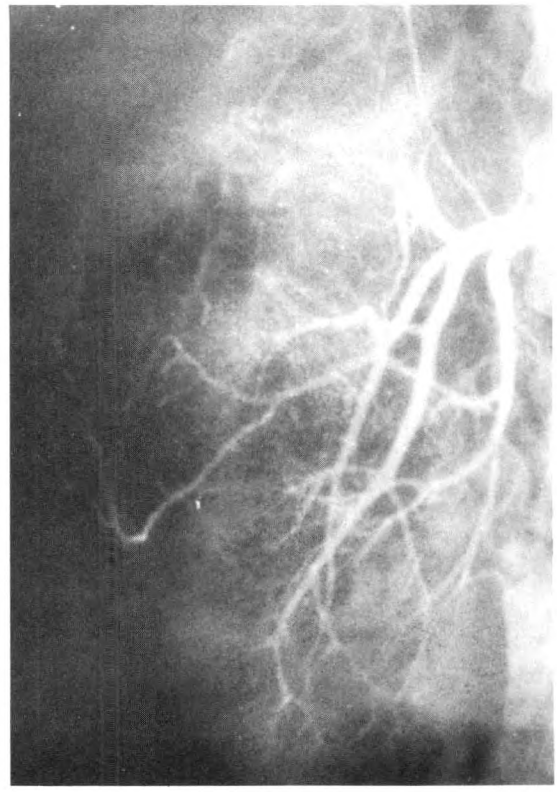

Fig. 8. Localized multilocular disease. This is a hypervascular lesion that may be confused with tumor. Careful observation reveals vessels that, although slightly directed in random fashion, taper relatively normally as they extend toward the periphery. Also, with this degree of vascularity tumor should demonstrate other features anticipated; *e.g.*, arteriovenous communication, puddling or early venous filling.

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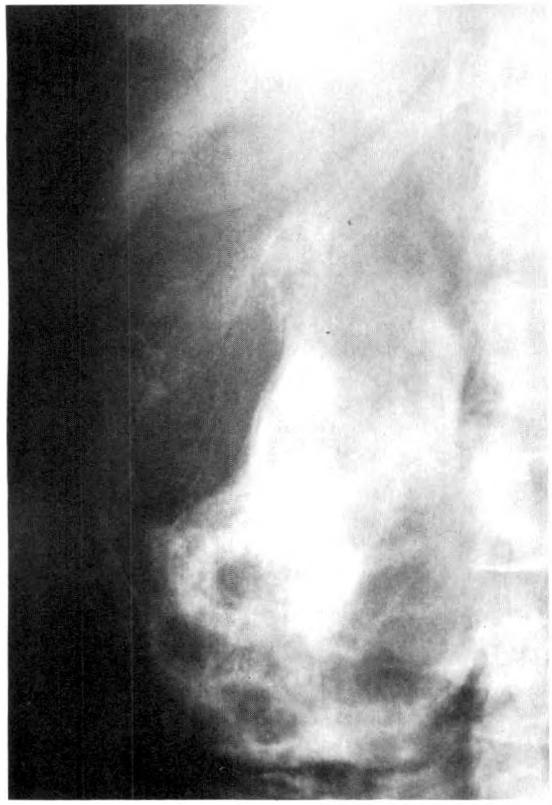

Fig. 7. Nephrogram X-P reveals blotchy, somewhat irregular opacification. This is opacification within a somewhat distorted renal parenchyma. (Same arteriogram in Figure 3 shows that surrounding lucent structure compressing upper pole area is peri-renal extension of the disease.)

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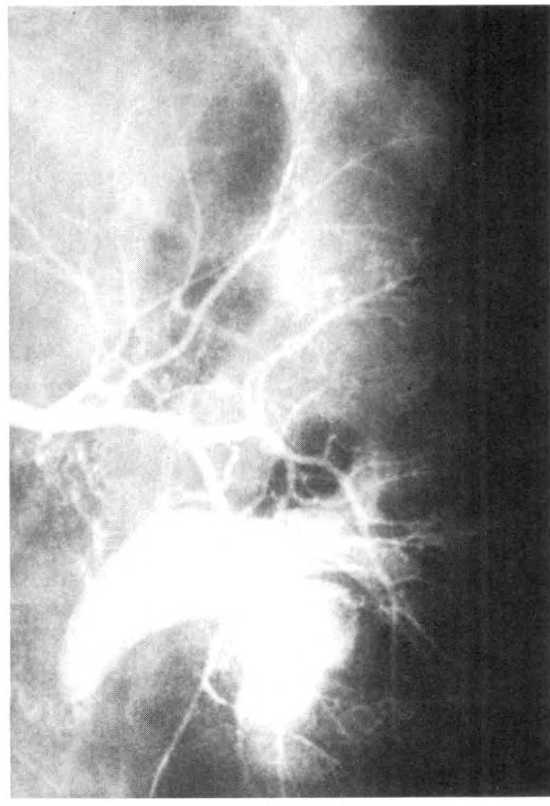

Fig. 9. Diffuse X-P. Angiographic study of a patient with diffuse involvement reveals marked elongation and spreading of the vessels. Small vascular twigs and early branches somewhat irregular in shape are noted.

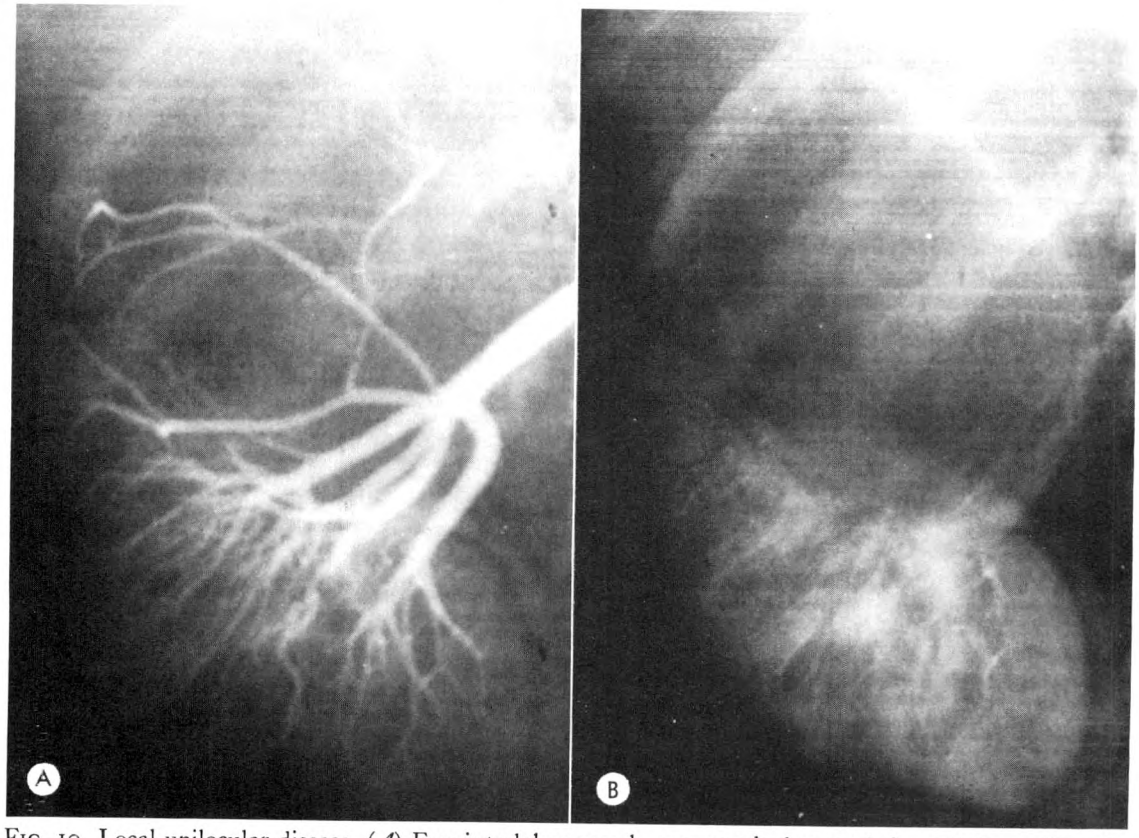

FIG. 10. Local unilocular disease. (*A*) Few interlobar vessels are stretched around the periphery of a relatively avascular mass in the upper pole. (*B*) Nephrogram reveals an avascular lucent mass surrounded by a thick wall capsule.

differentiated from adenocarcinoma of the kidney.[2,6,11]

A cross section of a vessel from a kidney involved with adenocarcinoma shows that the vessel consists only of endothelium partially surrounded by fibrous connective tissue (Fig. 5*A*). By contrast, comparative studies of vessels present in X-P show basically "normal" vessels, that is, the endothelium is surrounded by a muscularis composed of hypertrophic, smooth muscle and an adventitia. This observation was confirmed by staining multiple sections of carcinomas and X-P's with Masson's trichrome stain that differentiates fibrous connective tissue from smooth muscle. These observations indicate that the vessels present within the tumor are basically new vessels or true neovascularity because their walls are composed of fibrous connective tissue and contain no smooth muscle. Since smooth muscle neither proliferates nor regenerates, the vessels associated with X-P are not new vessels: *X-P forms no neovascularity.*

If X-P produces no neovascularity then what have observers been mistaking for it?

Does inflammatory vascularity fulfill the same criteria angiographically as neovascularity and as a result is it indistinguishable from neovascularity?

Angiographically, neovascularity is characterized by extremely tortuous vessels that are completely purposeless in direction, the lumens of which do not taper normally but rather widen and narrow in a disorderly fashion. Neovascularity is associated with puddling, tumor staining and early venous filling (Fig. 6).

For the purpose of discussion of the angiographic findings we have divided X-P into 2 main categories: (1) localized, either uni- or multilocular; and (2) diffuse.

If one compares a hypervascular localized

lesion encountered in X-P to a renal cell carcinoma, one notes that the vessels in X-P often taper toward the periphery, are usually not purposeless in direction, produce no puddling, staining or early venous filling. Although X-P may present an appearance suggesting staining, careful observation will show that this appearance is due to opacification of renal parenchyma that has been distorted by the inflammatory response (Fig. 7). Moreover, vessels supplying X-P may contract when epinephrine is injected into them. Although, the vessels involved with the multilocular variety of X-P are somewhat purposeless in direction and appear to narrow and widen somewhat, the degree and appearance are remarkably different from those in a tumor (Fig. 8).

Finally, diffuse lesions should never be confused with adenocarcinoma of the kidney, for not only does the over-all pattern of these vessels differ in appearance between these diseases, but adenocarcinoma almost never involves the entire kidney. Some vascular patterns produced by squamous cell carcinoma may, however, resemble those produced by X-P (Fig. 9; and 10).[9]

Nevertheless, the avascular, solitary mass lesions (X-P) cannot be definitively distinguished from necrotic avascular adenocarcinomas on angiographic findings alone. Other roentgenologic and clinical findings must be applied in these cases. This premise also holds true for abscesses of the kidney without xanthomatous changes.[5,10]

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REFERENCES

1. Anhalt, N. A., Kawood, C. D., and Scott, R., Jr. Xanthogranulomatous pyelonephritis: comprehensive review with report of four additional cases. *J. Urol.*, 1971, *105*, 10–17.
2. Becker, J. A. Xanthogranulomatous pyelonephritis: case report with angiographic findings. *Acta radiol.*, 1966, *4*, 139–144.
3. Elliott, C. B., Johnson, H. W., and Balfour, J. A. Xanthogranulomatous pyelonephritis and perirenal xanthogranuloma. *Brit. J. Urol.*, 1968, *40*, 548–555.
4. Gingell, J. C., Roylance, J., Davies, D. R., and Penry, J. B. Xanthogranulomatous pyelonephritis. *Brit. J. Radiol.*, 1973, *46*, 99–109.
5. Himmelfarb, E. H., Rabinowitz, J. G., Kinkhabwala, M. N., and Becker, J. A. Roentgen features of renal carbuncle. *J. Urol.*, 1972, *108*, 846–851.
6. Kahn, P. C., and Wise, H. M., Jr. Simulation of renal tumor response to epinephrine by inflammatory disease. *Radiology*, 1967, *89*, 1062–1064.
7. Lagergren, C., Lindbon, A., and Soderberg, G. Hypervascularization in chronic inflammation demonstrated by angiography. *Acta radiol.*, 1958, *49*, 442–452.
8. Noyes, W. E., and Palubinskas, A. J. Xanthogranulomatous pyelonephritis. *J. Urol.*, 1969, *101*, 132–136.
9. Rabinowitz, J. G., Kinkhabwala, M., Himmelfarb, E., Robinson, T., Becker, J. A., Bosniak, M., and Madayag, M. M. Renal pelvic carcinoma—angiographic re-evaluation. *Radiology*, 1972, *102*, 551–554.
10. Rabinowitz, J. G., Kinkhabwala, M. N., Robinson, T., Spyropoulos, E., and Becker, J. A. Acute renal carbuncle: roentgenographic clarification of medical enigma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 740–748.
11. Vinik, M., Freed, T. A., Smellie, W. A. B., and Weidner, W. Xanthogranulomatous pyelonephritis: angiographic considerations. *Radiology*, 1969, *92*, 537–540.

THE SIGNIFICANCE OF THERMOGRAPHY IN THE DIAGNOSIS OF ACUTE ABDOMINAL DISEASE*

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ABSTRACT:

Thermography appears to be a useful supporting aid to classical methods of examination in the clarification of the problems of acute abdominal disease. In a study of 100 patients thermography was in support of the true diagnosis in acute appendicitis in 63 per cent, in acute biliary tract disease in 59 per cent, and, in an attempt to differentiate between tumor and periappendicular abscess, assistance was rendered by thermography in 53 per cent of patients. However, some uncertainty exists because carcinoma often produces in its environment an inflammatory reaction, which causes an increase of temperature visible in the thermogram. In the present series thermography produced a finding supporting the correct diagnosis in 59 per cent. Errors occurred most often in obese patients, who have thick abdominal covers. Thermography also has an obvious application in the localizing of postoperative suppurative foci.

THE patient with acute abdominal disease is occasionally highly problematic to the examining physician: in spite of the development of medicine and the improvement of diagnostics in all areas, new methods of diagnosis aiding the acute abdominal patient have been few. It is true that owing to advances in anesthesia, understanding of fluid balance, and antibiotic treatment the mortality in appendicitis, for instance, has greatly decreased in the recent decades; it is also a fact, however, that we are continually losing patients due to appendicitis. This is by no means always caused by the patient's tardiness. Part of the appendicitis mortality also has to be assigned to diagnostic error.

Thermographic examination has spread in medical use during the most recent decade. The beginning was made when in 1964 the Canadian surgeon Ray Lawson[10] published his breast cancer studies. He demonstrated that breast cancer is warm and observable by a temperature measurement. Thermography has been extensively applied together with mammography in the diagnosis of mammary tumor.[5,6] The accuracy of thermography in breast tumors has been about 75–90 per cent in different series.

Thermography is also applied with fair success in examinations for various circulatory disorders[9,13] and rheumatoid changes.[14]

In supplementation of other methods, thermography is used, although with less reliable results, in the examination of venous thrombosis, thyroid nodules,[8] and in obstetrics for determining the location of the placenta,[1] etc.

Our search revealed only 1 thermographic investigation concerning acute abdominal disease, made in 1968 by Buwalda[3] with a comparatively small series.

It was our desire to determine if thermography has a significant place in the diagnosis of acute appendicitis, acute cholecystitis, and abscesses in the abdominal cavity.

MATERIAL AND METHOD

A total of 100 patients with acute abdominal disease was examined during the years 1970–1973 by thermography at the Second Department of Surgery, Helsinki

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University Central Hospital.

The apparatus used in the studies was an AGA Thermovision Model 551, by the aid of which the infrared radiation emitted by the patient was measured. The temperature measurement was made from the patient's abdominal skin with the aid of a mirror placed at an angle of 45 degrees. The patient was kept for about 10 minutes prior to the examination with the area in question free of clothes in order to eliminate the thermal pattern produced by the clothing.

The temperature of the region presenting symptoms was measured and compared with the surrounding areas and the corresponding, contralateral location. The isothermal curves could be evaluated to give the temperature difference betweeen the comparison areas in degrees Centigrade. Owing to the fairly strong basic thermal pattern, only differences exceeding 1°, or preferably 2°, can be interpreted as pathological. In violent inflammations temperature differences up to 4–5° may be noted.

The 100 patients with acute abdominal disease examined by thermography displayed the following distribution according to disease groups:

Group I. Biliary group (acute)	41 cases
Group II. Appendicitis group	27 cases
Group III. Abscess/Tumor group	32 cases

Sixty-three of the patients examined were female and 37 were male.

The age distribution in the different groups was as follows:

Age	Number in Biliary Group	Appendicitis Group	Abscess/Tumor Group	Total
Under 29	0	13	2	15
20–39 years	5	4	4	13
40–59 years	19	7	11	37
Over 60	17	3	15	35
Total	41	27	32	100

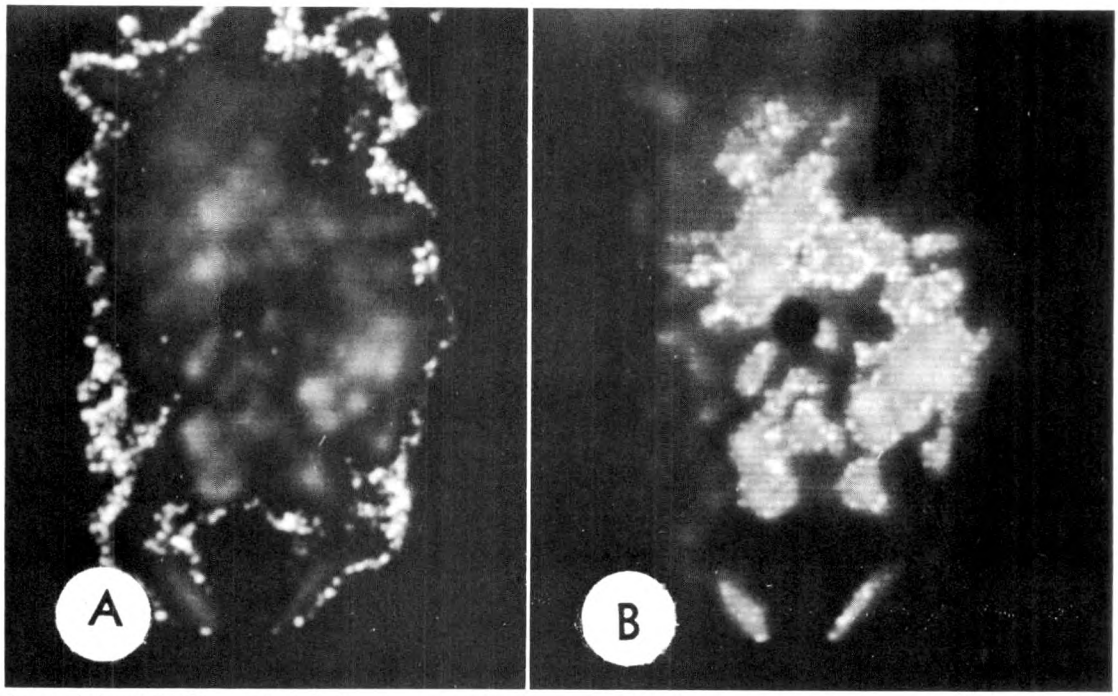

Fig. 1. (*A* and *B*) The patient had acute appendicitis, and in the thermographs a hot spot can be seen on the right lower abdomen (low on your right in the figure).

After the final diagnosis had been established, the series was analyzed separately for each group. A temperature difference of 2° or higher was considered as supporting the presence of inflammation.

I—Biliary Group (acute). This group was comprised of 11 male and 30 female patients who had been admitted to the emergency ward owing to abdominal distress. The relation of thermography to the conventional means of examination, including history, clinical status, and leukocyte count, is shown by the following table.

Relation of thermography and diagnosis.

	In Support	Without Significance	Misleading	Total
History				
Negative	1	5	0	6
Distinct	3	6	0	9
Strong	16	7	3	26
Clinical status				
Negative	0	4	0	4
Distinct	7	7	2	16
Strong	13	7	1	21
Leukocyte count				
Under 8,000	3	4	0	7
8,000–10,000	3	5	0	8
Over 10,000	14	9	3	26

The table reveals that the history of the patient failed to corroborate the diagnosis of acute biliary inflammation in 6 cases and the clinical examination in 4, while the leukocytes were not elevated in 7 patients. Thermography proved misleading in 3 cases.

One case of empyema of the gallbladder with leukocytosis of 11,000, body temperature 38°C., and a severe disease both anamnestically and clinically, was interpreted as a clear case of inflammatory biliary disease. The temperature difference in thermography was zero, however. Operation revealed gallbladder empyema.

Two other patients had clear-cut acute gallbladder inflammation (distinct leukocytosis and obvious disease both anamnestically and clinically). Furthermore, 1 of them already suffered from peritonitis when the operation was undertaken. In spite of this no temperature differences were elicited. All 3 patients with erroneous result were obese and had thick abdominal covers.

Results of thermography in biliary patients:

Finding in support of the diagnosis (average temperature difference 2–4°)	24 patients (59 per cent)
No significance in regard to diagnosis	14 patients (34 per cent)
Misleading	3 patients (7 per cent)

In none of the cases was thermography the sole examination supporting the diagnosis.

II—Appendicitis Group (Fig. 1, *A* and *B*). This group consists of 27 patients, 17 women and 10 men. Half of the patients were young (under 20 years) and 10 patients were older than 40. Diagnosis of acute appendicitis was made preoperatively in all cases. The results of examination were as follows.

Type of Examination	Number of Patients
History	
Negative	3
Suspect	9
Distinct	15
Clinical examination	
Negative	5
Suspect	4
Distinct	18
Leukocyte count	
Under 8,000	5
8,000–10,000	5
Over 10,000	17

Results of thermography, compared with the true operative diagnosis:

Type of Inflammation	Result of Thermography		
	In Support	Without Significance	Misleading
Perforation	1	1	0
Gangrenous	4	0	1
Acute	9	1	2
Innocent	3	4	1
Total	17 (63 per cent)	6 (22 per cent)	4 (15 per cent)

The results show that almost exactly the same result was obtained by thermography in the unambiguous cases as by the examination procedures commonly available in emergency admission conditions (history, clinical examination and leukocyte count). In 2 out of 3 patients the thermographic examination was in support of the true diagnosis, that is, the temperature difference was 2° or higher. The 3 cases with misleading thermography all presented a distinct history of appendicitis, distinct clinical findings and leukocytosis, but no temperature differences occurred in spite of this. On the other hand, an innocent appendix was discovered in 1 case even though the temperature difference was as high as 5°.

III—Abscess/Tumor Group (Fig. 2; 3; 4, *A* and *B*; and 5, *A* and *B*). This group was studied in order to find out whether thermography may possibly furnish aid in the differential diagnosis of abscesses and tumors. Of the 32 patients of this group, 16 were male and 16 were female.

The aid derived from thermography in the diagnosis was as follows.

In support	16 patients (50 per cent)
Without significance	11 patients (35 per cent)
Sole source of diagnosis	1 patient (3 per cent)
Misleading	4 patients (12 per cent)

We consider thermography the sole source of diagnosis in 1 case, a 52 year old man with distinct signs suggesting a periappendicular abscess, elevated temperature, and a leukocyte count of 13,900. A roentgen examination was also made, but it revealed no specific findings. No temperature differences were noted on thermography. On these grounds we believed the resistance palpable in the lower right abdomen to be a tumor. This suspicion was later confirmed at operation.

There were 9 tumors of cecum and ascending colon in which a very clear thermographic finding supporting tumor diagnosis was obtained. All had palpable resistance in the lower right abdomen. Thermography

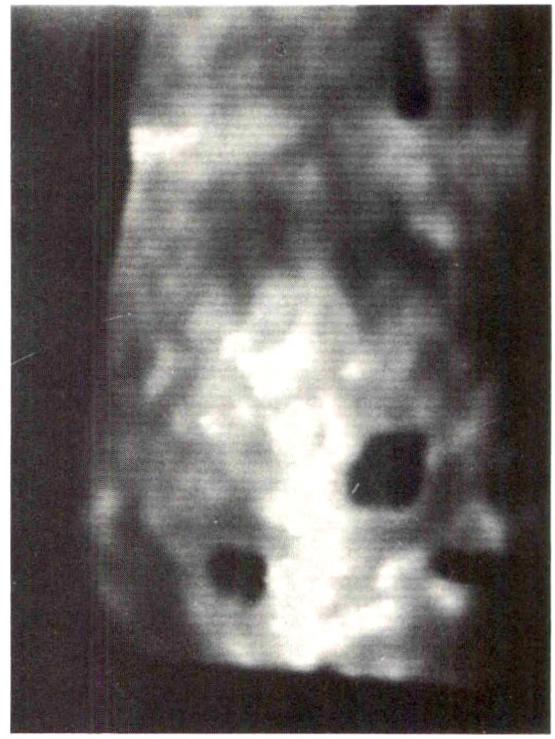

Fig. 2. A left-sided subphrenic abscess was also disclosed by thermography (in the figure, a hot spot caused by the abscess, high on your left side).

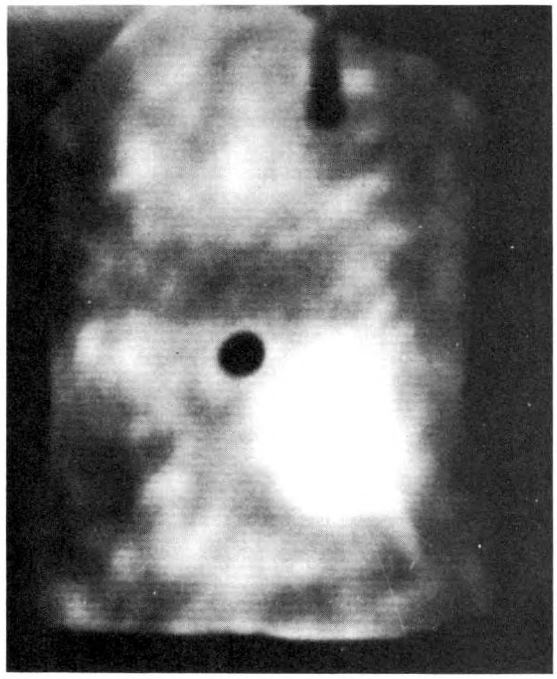

FIG. 3. Thermography clearly reveals the hot spot from a periappendicular abscess.

elicited no temperature difference. In tumors elsewhere in the colon, a thermographic finding supporting the diagnosis was also obtained in 3 cases. Out of 4 cases of periappendicular abscess, 1 produced no distinct temperature difference, and in 1 further case the difference was only 1.5°.

The thermographic examination was in support of the tumor *versus* abscess diagnosis in half of the patients. It has to be noted, however, that thermography was misleading in one-eighth of the cases. In all 3 groups the patients for whom misleading thermographic results were recorded were appreciably obese and had thick abdominal covers.

DISCUSSION

Thermography has been used rather sparingly as an aid in the diagnosis of acute abdominal disease. Buwalda,[3] in 1968, found a positive thermographic study in examinations for acute appendicitis in not

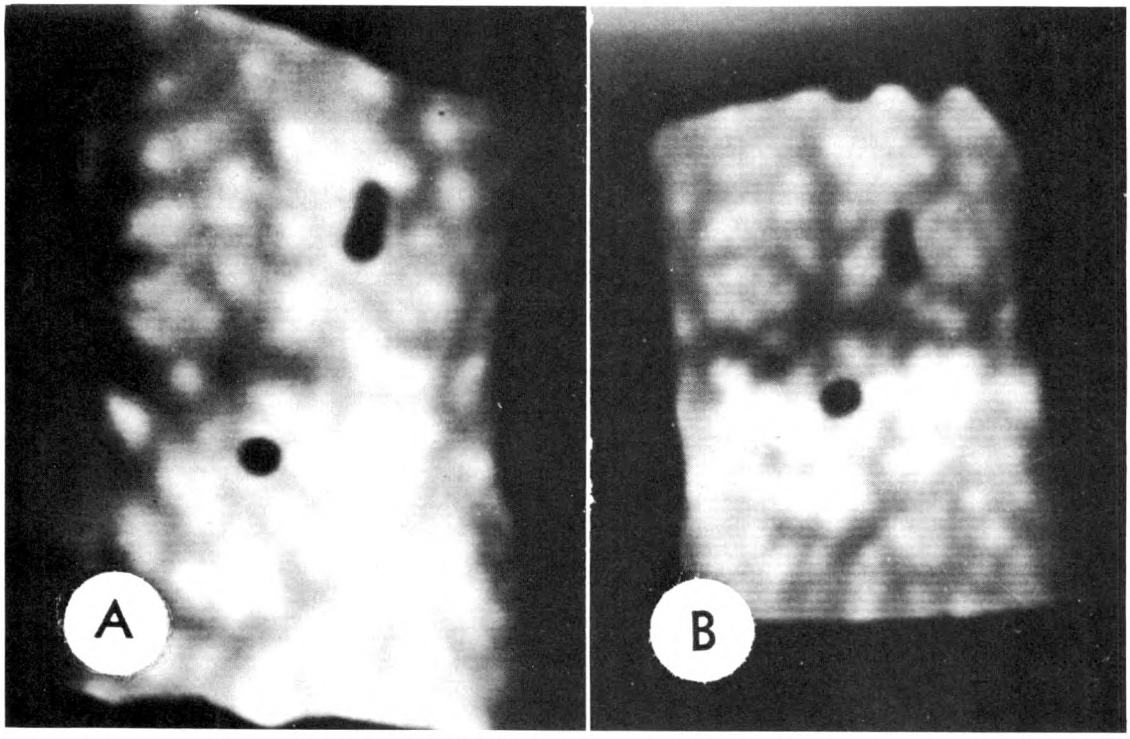

FIG. 4. A periappendicular abscess (*A*) in the acute stage; and (*B*) after 1 month, when the temperature differences on the lower abdomen have already leveled out.

quite one-half (47 per cent) of cases. In the present study we obtained thermographic results in support of the diagnosis in 63 per cent. This was based on our criterion that there should be a temperature at least 2° higher in the appendix region. The number of positive findings was of the same order as that obtained by conventional emergency case procedures (history, clinical examination and leukocyte count). It is believed that the localization of the appendix is significant for the thermographic finding.[3]

The abdominal region is not the best suited to thermographic examinations, since it even normally presents an ample thermal pattern caused by subcutaneous tissues. Heat reflections on the surface may even be produced by major vessels at greater depth. Such deep reflections are better visible in lean than in obese patients.

Examinations by thermography with respect to acute cholecystitis are impeded by the abundant vascularization of the liver. However, Mansfield *et al.*[11] in 1970 clarified the presence and diagnosis of hepatic metastases with the aid of thermography, obtaining a result as high as 70 per cent. In our series of 41 biliary patients the diagnosis received support from thermography in 59 per cent. This, too, is of the same order as the diagnostic assurance obtainable from history, clinical examination, and simple laboratory tests. In 3 cases the thermographic finding was misleading. All these patients were obese and therefore had thick abdominal covers. A subcutaneous fat layer 1 cm. in thickness is considered critical in view of thermography.

The differentiation of periappendicular abscess and tumor is frequently exceedingly difficult, and we believe that this is one of the most rewarding applications of thermography. Diagnosis of carcinoma is quite often, and fatefully, delayed because a tumor palpable in the lower abdomen is believed to be a periappendicular abscess.[12]

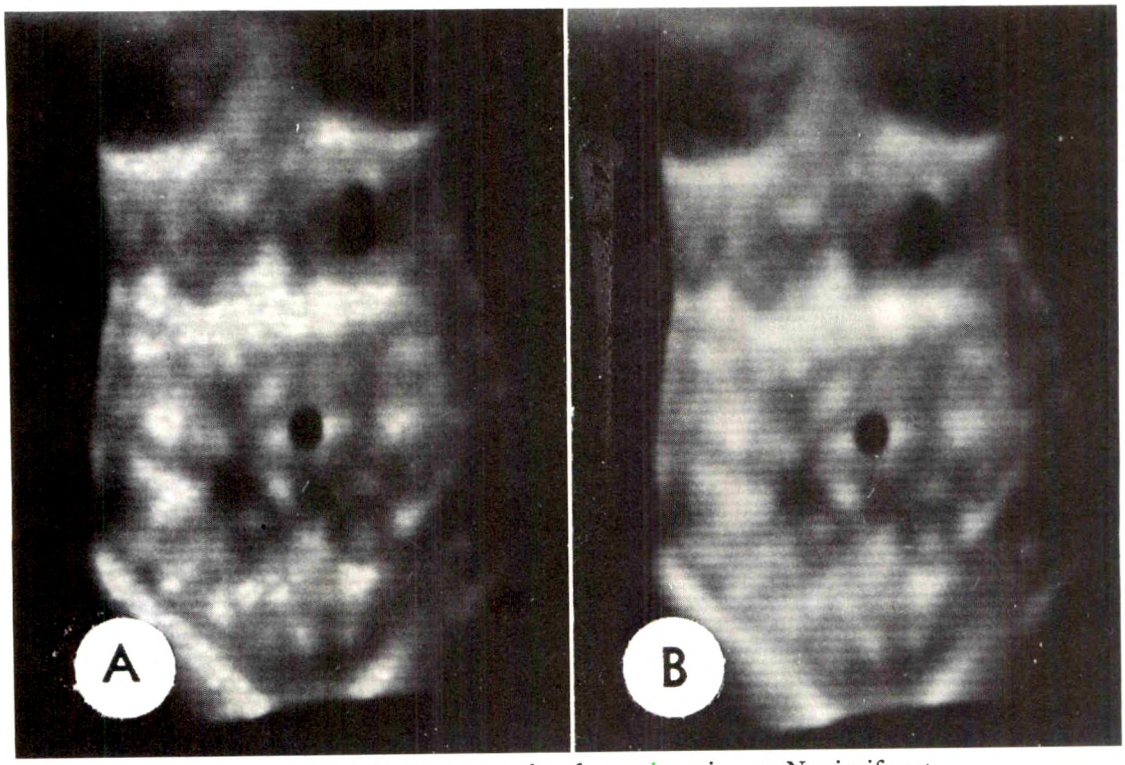

Fig. 5. (*A* and *B*) Thermographs of a cecal carcinoma. No significant temperature difference can be noted.

It should be kept in mind that fever is a rather important symptom also in cancer of the large intestine. The mass palpable with a carcinoma may sometimes be a result of inflammation. This inflammatory reaction may even produce clinical symptoms in the patient.[2]

A frequent error occurs in the diagnosis of cecal carcinoma when a patient has fever, abdominal pain, and a mass palpable in the right lower abdomen believed to be a periappendicular abscess. Antibiotic treatment results in disappearance of the "tumor" mass which may have been an inflammatory reaction caused by the carcinoma. Therefore, patients with a periappendicular abscess should be subjected to a roentgenographic examination and the patient should not be dismissed until the symptoms have completely vanished or the patient has been operated on. Erroneous diagnosis of periappendicular abscess delayed the diagnosis of carcinoma by an average 6.5 months.[12]

Cancer of the large intestine often causes symptoms resembling appendicitis, and in such cases the diagnosis of appendicitis is quite often made. In addition, the large intestinal neoplasm may even itself produce acute appendicitis. Cancer in the large intestine has been found to contribute to the genesis of acute appendicitis by the following mechanisms:

(1) The lumen is occluded by the cancerous tissue itself or by the surrounding edema.[4]
(2) When a cecal carcinoma ulcerates or becomes infected, the inflammation spreads to the adjacent organ, the appendix.
(3) Obstruction caused by cancer produces tension and expansion not only in the cecum, but also in the appendix. The stasis, in its turn, produces vascular disturbances and inflammation.[15]

This close relationship of appendicitis and inflammation with carcinoma interferes considerably with the usefulness of thermography in the differential diagnosis. However, in more than half of the cases (53 per cent) the thermographic finding was in support of the abscess *vs.* tumor differentiation. In 1 patient, furthermore, thermography was the sole examination which supported the diagnosis, although it should be noted that in 12 per cent of cases the thermographic examination was misleading. In all groups the patients involved in the misleading findings were remarkably obese and had thick abdominal covers; these characteristics clearly appear to restrict the use of thermography.

One possible application area of thermography which was not clarified is the localizing of postoperative abscesses. In our opinion it appears as if thermography possesses significance in the diagnosis of the acute abdomen, as an aid to the existing examinations.

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REFERENCES

1. AANTAA, K. Location of placenta: comparison between radiographie, ultrasound, thermography and isotopes. *Acta radiol.*, 1971, Suppl. 303.
2. BRODERS, A. C., PHILLIPS, C., and STINSON, J. C. Neoplasms of large bowel. *Surg. Clin. North America*, 1952, *32*, 1511–1524.
3. BUWALDA, G. Possible application of thermography in acute abdominal condition. Medical Thermography, Boerhaave Course, March 28–30, 1969, Leyden.
4. COLLINS, D. C. Study of specimens of human vermiform appendix. *Surg., Gynec. & Obst.*, 1955, *101*, 437–452.
5. FURNIVAL, I. G., STEWART, H. J., WEDDEL, J. M., DOVEY, P., GRAVELLE, I. H., EVANS, K. T., and FORREST, A. P. Accuracy of screening methods for diagnosis of breast disease. *Brit. M. J.*, 1970, *4*, 461–463.
6. GERSHON-COHEN, J., HERMEL, M. B., and MURDOCK, M. G. Thermography in detection of early breast cancer. *Cancer*, 1970, *26*, 1153–1156.
7. HOSSAIN, M. A. Unrecognized carcinoma of caecum presenting as acute appendicitis or appendix abscess. *Brit. M. J.*, 1962, *11*, 709–716.

8. Karpman, H. A. Thermography in diagnosis of thyroid nodule. *J.A.M.A.*, 1971, *216*, 1646–1647.
9. Kormano, M., Kahanpää, K., Svinhufvud, U., and Töhti, E. Thermography of varicocele. *Fertil. & Steril.*, 1970, *21*, 558–564.
10. Lawson, R. N. Early applications of thermography. *Ann. N. Y. Acad. Sc.*, 1964, *121*, 31–33.
11. Mansfield, G. M., Farrel, C., and Asbell, S. Use of thermography in detection of metastatic liver disease. *Radiology*, 1970, *95*, 696–698.
12. Peltokallio, P. Acute appendicitis associated with carcinoma of colon. *Dis. Colon & Rectum*, 1966, *9*, 453–461.
13. Thiers, H., Schmitt, M., and Fagolle, J. Thermographic data in Raynaud's syndrome: initial result in Lyon. *Bull Soc. franç. dermat. et syph.*, 1970, *77*, 462–464.
14. Viitanen, S-M., and Laaksonen, A. L. Thermography in juvenile rheumatoid arthritis. *Acta rheum. scandinav.*, 1970, *16*, 91–98.
15. Wilson, E. Carcinoma of colon associated with appendicitis. *Dis. Colon & Rectum*, 1964, *7*, 348–353.

PRACTICAL SIGNIFICANCE OF GASTRIC RUGAL FOLDS*

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ABSTRACT:

A clinical pilot study oriented toward the practical value of gastric fold assessment offers the following: (1) Younger patients (below 60 years) with carcinomas may have rugose stomachs as opposed to the more usual presentation in the elderly. (2) Some proportionality of gastric fold to acid output may exist in normal patients or cases with peptic disease of the distal stomach or duodenum. In any given case, however, acid secretory capacity may be very broad. (3) Proportionality of fold pattern within the stomach itself or with respect to the duodenum, as noted in the literature, is emphasized. (4) There exists a possible upper limit of "physiologic" fold thickness of 4 to 5 mm. beyond which an additional pathologic process may be inferred. (5) Hypertrophic gastritis and gastric body ulcer disease may illustrate this progression and possibly share similar underlying mucosal pathology and rugal characteristics.

CORRELATIVE statements about gastric and duodenal folds have surfaced intermittently: concerning "duodenitis" in the 1930s, atrophic gastritis or advanced gastric atrophy in the 1950s and the Zollinger-Ellison (ZE) syndrome since 1964. More recently, similar speculations have been advanced concerning other lesser degrees of peptic diathesis with or without roentgenographic ulceration, *e.g.*, hypertrophic hypersecretory gastropathy (HHG) of Stempien.[22] Applied still more broadly, assessment of acid secretory capacity has been claimed possible by simple observation of gastric and duodenal folds.[8,18]

At first, a retrospective review of rugal appearance and presumptive acid output was initiated indirectly by comparing a collected series of gastric carcinomas with random cases of gastric ulcers (GU) and duodenal ulcers (DU) to verify the stated usefulness of rugal fold assessment. Later, a direct fold to acid correlation became possible with a more regular use of acid stimulation testing. While still considered a tentative pilot study and founded chiefly on clinical or roentgenographic diagnosis, the findings to date may stimulate additional insights, especially in the light of literature review.

METHOD

A total of 33 gastric carcinomas collected from Lankenau Hospital's American College of Radiology Index over the past 5 years was compared with an additional 47 cases of GU or DU obtained randomly from the same files. Later, 42 ulcer cases examined by the clinical Gastrointestinal Laboratory during the past year were added to this first study group.

The more direct acid to rugal fold assessments of the second study group were based upon a standard 50 mg. histalog stimulation test on a total of 98 patients who had had recent recoverable upper gastrointestinal examinations (UGI).

In both study groups a representative overhead film from the standard UGI was retained for comparison with the others, the prone right oblique usually being the most satisfactory. Upper gastric body and fundal rugal folds fell at first into 5 or 6 general visual types. With the later case group having acid stimulation data, estimated measurements of average anterior

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body rugae were made by overlaying a clear plastic ruler. Admittedly inexact and occasionally hampered by poor barium adherence to the mucosal surface, these measurements are probably as meaningful as the visual grading cited in the literature and are offered for their heuristic value.

Initial "visual gradings" are described as classifications O-V and are depicted in Figure 1, *A–I*):

O. Fundal and greater curvature folds are readily effaced with gastric distention,

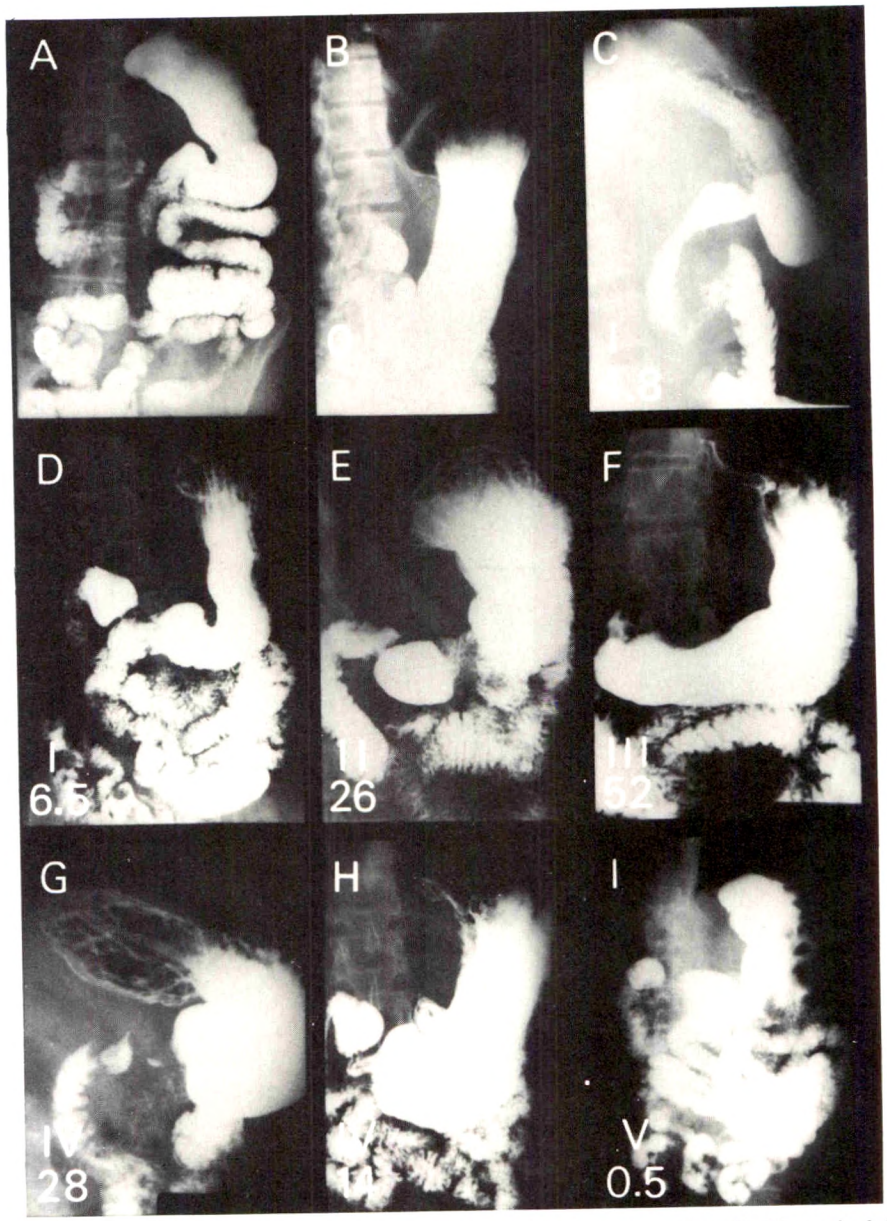

Fig. 1. *Gastric Rugal Classifications and Acid Outputs.* Classifications from O to V. Numerical figure represents the "maximal output" in mEq./hr., under standard 50 mg. histalog stimulation. Patients *A* and *B* are achlorhydric: *A* is an elderly male with known pernicious anemia, *B* is a 31 year old female. Note postapical DU in patient *F*, and DU in patient *G*. Note "bland" duodenum contrasting with heavy gastric folds of patients *H* and *I*. (Patient *H*, with endoscopic and biopsy diagnosis of "chronic gastritis," resembles HHG of Stempien but has normal acidity. Patient *I* has probable Menetrier's disease.)

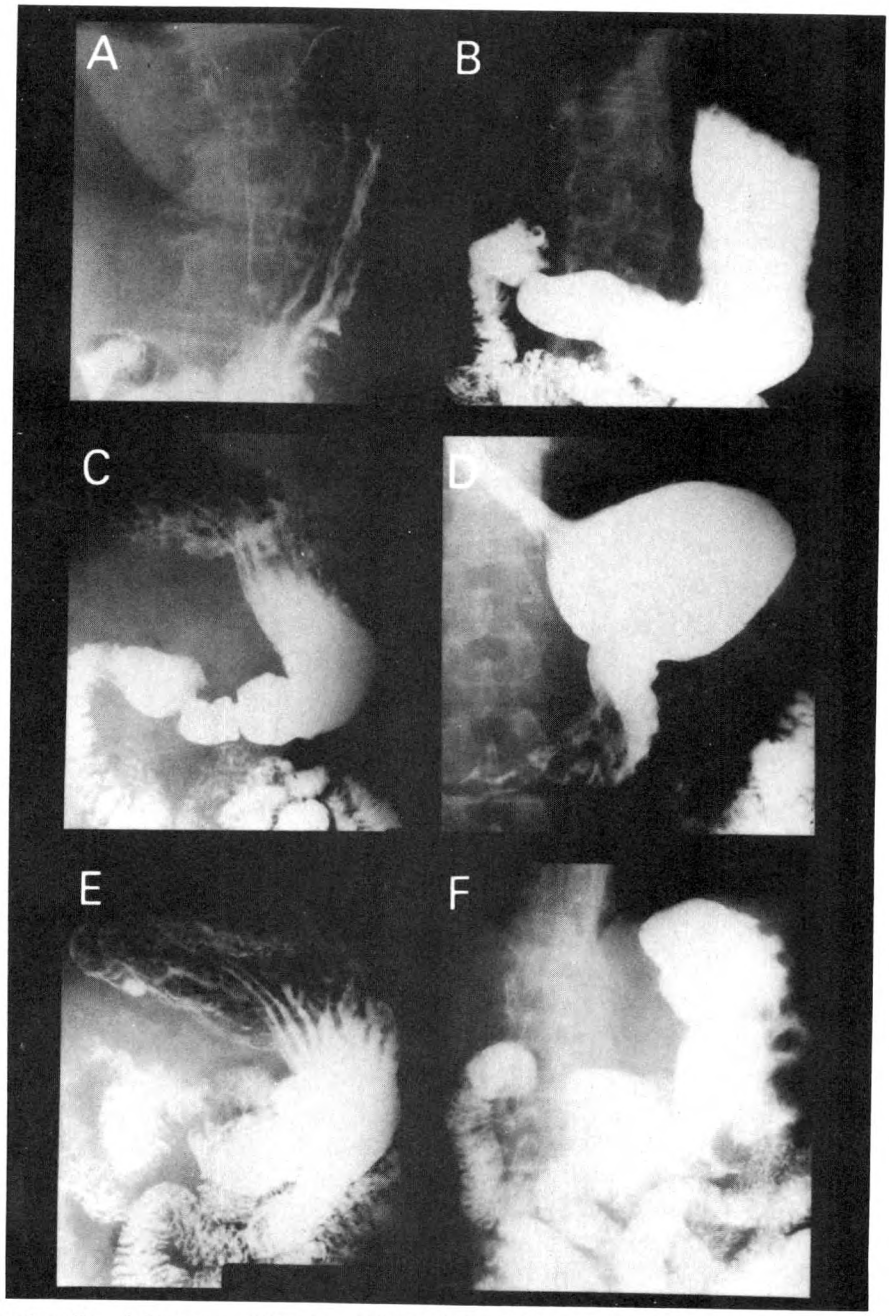

FIG. 2. *Proportionality of Gastric and Duodenal Folds.* (*A* and *B*) Focally thickened gastric body folds with respect to bald fundus and loss of wall compliance in the filled state, proved carcinoma in an achlorhydric elderly patient. (*C* and *D*) Focally thickened and nodular antral folds with respect to body, "chronic antral gastritis" on biopsy, acid productivity 16 mEq./hr. (*E*) Associated coarse duodenal folds (without ulcer) and heavy rugae, 22 mEq./hr. acidity output. (*F*) Normal duodenum, extremely heavy gastric folds, probable Menetrier's, 0.5 mEq./hr. acidity output.

those remaining appearing sparse, thin, 2 mm. or less in thickness. (When incompletely distended, some of this group interestingly have paradoxically thickened rugae out of character with their easy effacement, possibly a manifestation of mural hypotonia.)

I and II. Gastric folds are of low profile,

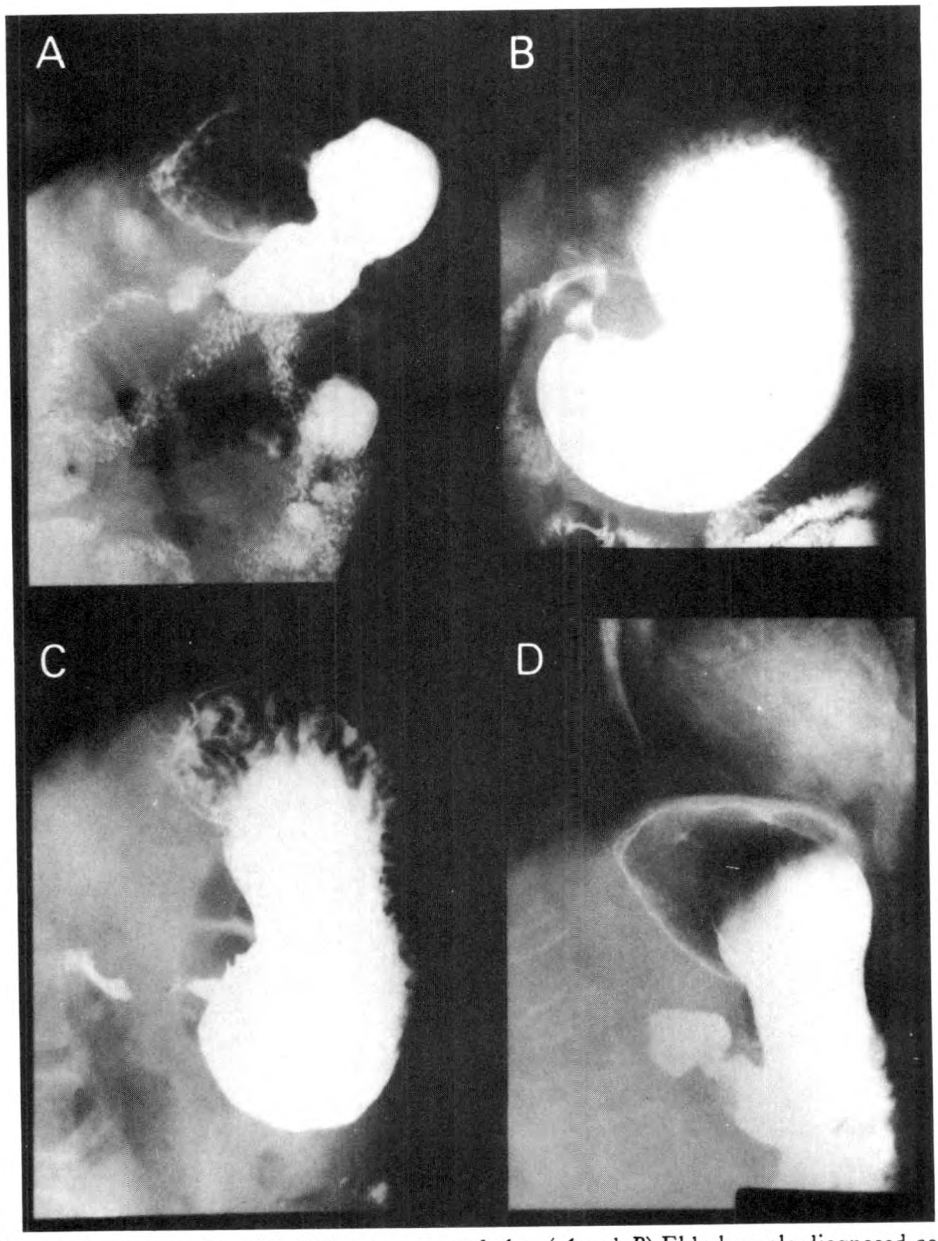

Fig. 3. *Gastric Carcinoma, Rugal Fold Appearance and Age.* (*A* and *B*) Elderly male diagnosed as "chronic distal antral scarring," 4 years later proved carcinoma. Note atrophic stomach. (*C*) A 45 year old male with antral carcinoma. (*D*) A 90 year old female, ? antral cancer, refused further work-up.

rather thin, straight, but of increased width in the higher grade.

III. Clearly coarsened fundal and body folds, more tortuous and thicker than I or II.

IV. Still more marked coarsening of the rugae, tending now to dominate the visual effect.

V. Massive rugal prominence with folds not readily measurable, likely more than 1 cm. thickness.

RESULTS

A variable degree of duodenal fold coarsening was apparent in the classifications from II–IV with or without a diagnosable duodenal ulcer. This duodenal appearance was assumed secondary to the

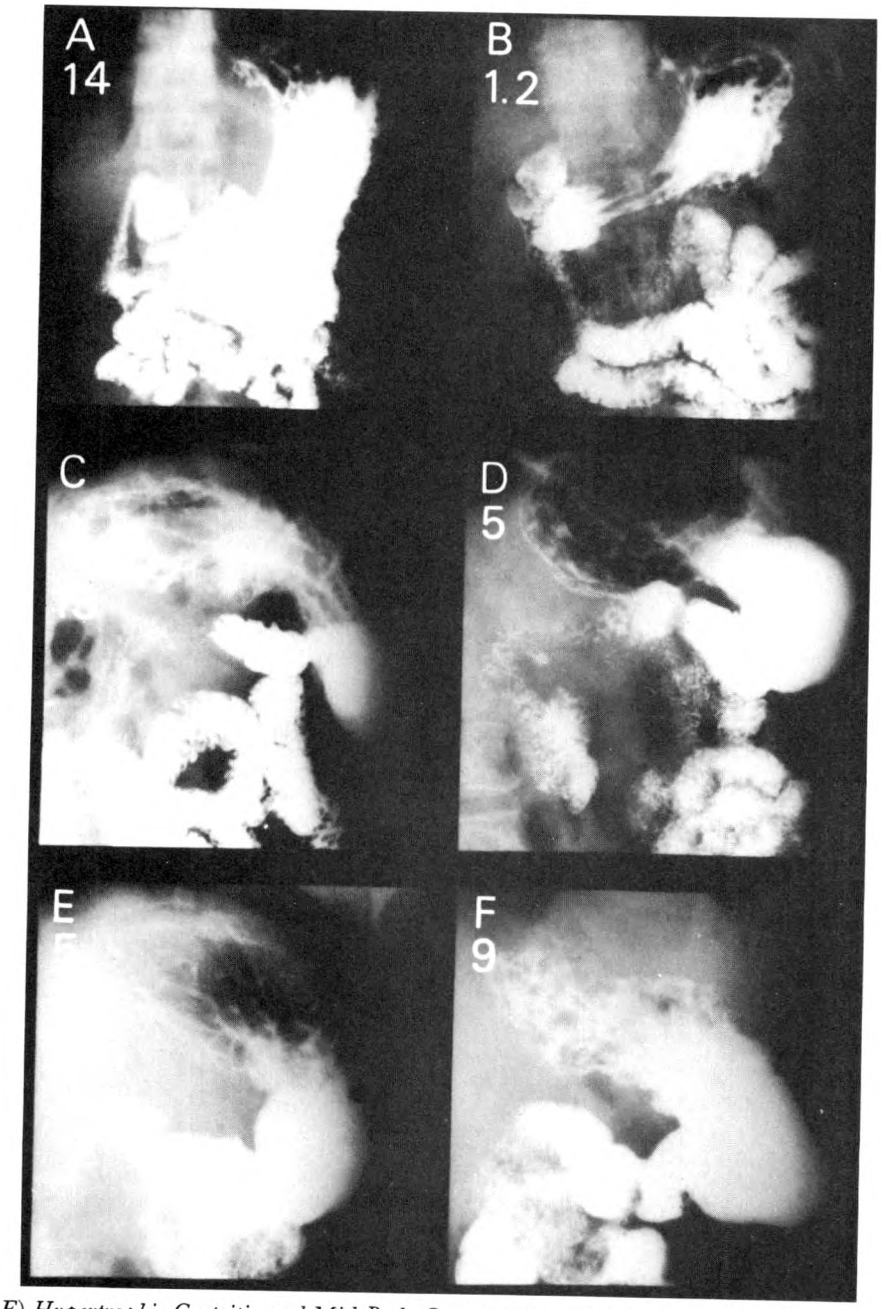

FIG. 4. (*A–F*) *Hypertrophic Gastritis and Mid-Body Gastric Ulcer Disease.* All patients show rather low acid output but unusually prominent rugal patterns of their acid secreting mucosa. Patients *E* and *F* have mid-body gastric ulcers and may have similar underlying mucosal disease. Note the duodenum in all cases is normal.

primary fundal rugal pattern and was not specifically studied. Likewise, possible abnormality in gastric secretions, *e.g.*, increased mucus, exudate, or excessive quantity, made manifest by poor barium mucosal adherence, seemed to be a meaningful roentgenographic finding but was too difficult to assess specifically.

General fold relationships emerge. Rugal coarsening and thickness are greatest in the acid secreting portion of gastric mucosa, the fundus and body, with antral

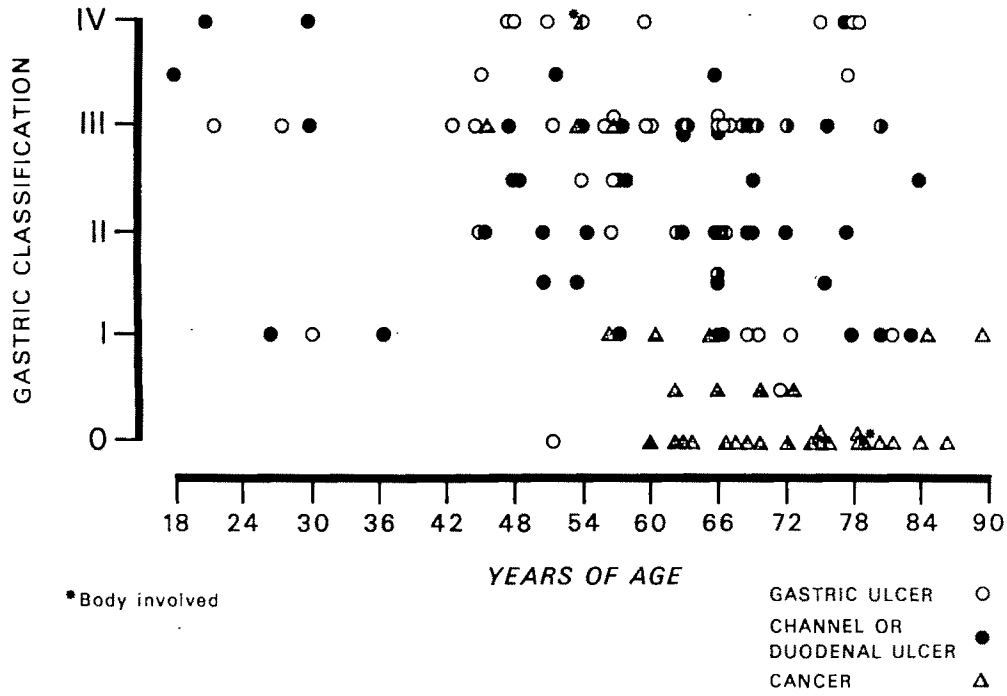

FIG. 5. *Collected Random "Peptic" Cases vs. Accumulated Gastric Cancers.* Visual gastric fold classification with respect to age. Skewed position of cancers in patients 60 years or older. Generally more prominent rugal appearance in GU cases, see text.

folds equal to the fundal rugae in the lowest gastric categories. Proportionality of duodenal and gastric folds, emphasized in the literature,[3,7,18,20,23,25] also applies to the gastric antrum. These fold relationships described above have apparent diagnostic value. A local area of gastric fold exaggeration was seen to reflect tumor or inflammation in specific cases (Fig. 2, *A–D*). Hyperrugosity with a "bland" duodenum suggested an underlying chronic mucosal disease, possibly of diverse etiology, but with low acid output as contrasted with the "reactive" duodenum reflecting a more normal or increased acid output (Fig. 2, *E* and *F*). Gastric atrophy consistently cautioned a high index of suspicion of any lesion, especially the diagnosis of "chronic peptic scarring" (Fig. 3, *A*, *B* and *D*).

Graphing the known carcinomas *vs.* random DU and GU cases (Fig. 5) indicates to no surprise that older patients with cancers did fall into the lowest "atrophic" grouping. Below 60 years, however, cancers may affect the more rugose types (Fig. 3*C*). The peptic cases overlap but GU or combined ulcer cases tend toward greater rugosity, particularly in the older population.

Still, additional possible useful patterns emerge from the second group of patients when acid stimulation data can be graphed with respect to rugal gradings and measurements, particularly when the patients are separated into various diagnostic groupings. (The 50 mg. histalog is less than the "maximal" dosage used in the literature but allows a relative index of acid capacity within the patient group tested.)

"*Gastritis*" cases (Fig. 6) fall into the extremes of fold classifications. "Atrophic" cases include a number of carcinomas and one pernicious anemia. "Hypertrophic"

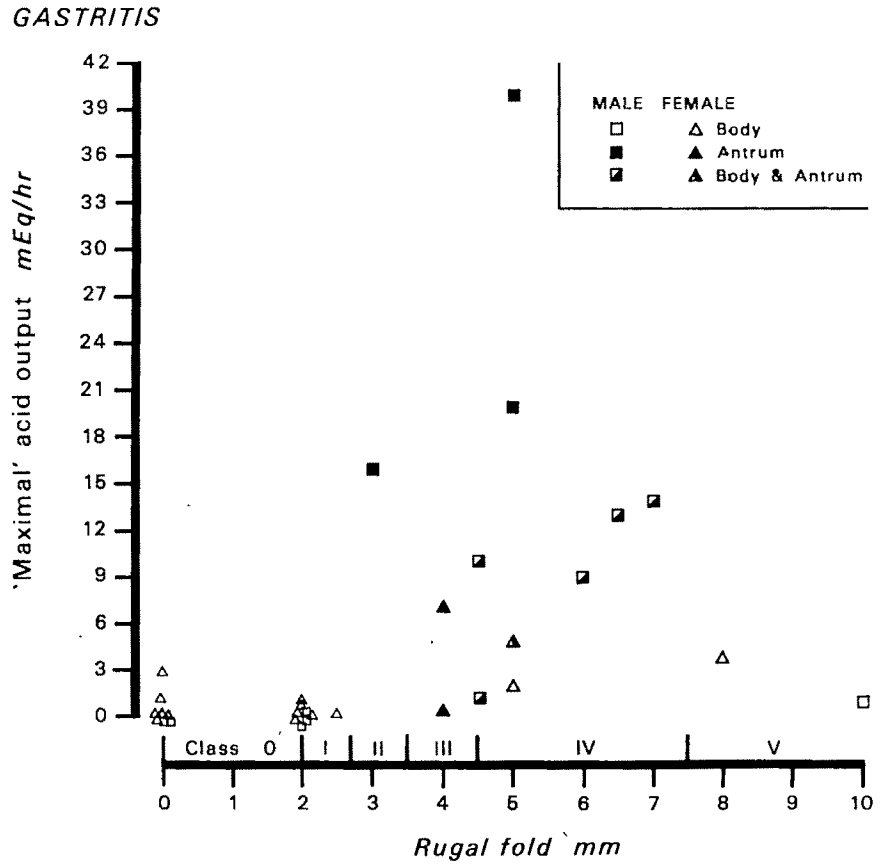

FIG. 6. *Gastritis*. Atrophic and hypertrophic types. Note the elevated acid in patients with antral disease and progressive low to absent acid output with gastric fundal and body mucosa involvement, the acid bearing areas of gastric mucosa.

cases affected the acid-bearing mucosa, included the antrum, or involved the antrum alone. There is no general progression of acid output with increasing body and fundal folds but, in fact, a tendency to the reverse beyond folds of 5 mm. Those patients having solely antral disease have the greatest acid production and seemingly fit into the duodenal-channel ulcer progression below. Patients with greater degrees of fundal and upper body findings tend to separate in a skewed manner, more in keeping with the scattergram of midbody gastric ulcers (Fig. 4, *A–F*).

Duodenal and channel ulcer cases (Fig. 7) do tend toward a general fold to acid scattergram having a steep slope although in any particular case a given fold appearance has a wide range of acid productivity.

Miscellaneous "normal" cases (Fig. 8), without peptic disease, have a slope similar to DU but a less extreme peak of acid output.

GU cases (Fig. 9) show a scattergram with a less definable slope apparently composed of a mixed population. Antral ulcers predominate in the lower gastric grades and have the higher outputs similar to antral gastritis. Angularis cases lie more midway between antral and mid-body ulcers. The group with mid-body ulcers is skewed toward the lower acid outputs and the most prominent rugal patterns, in this respect, falling into the fundal and upper body "hypertrophic" population.

Figure 10 summarizes the various group characteristics. Over-all, the relative acidity output seems to peak from Class II to

early Class IV. It may be that 4–5 mm. thickness of the upper body folds is, in fact, the "maximal physiologic" fold thickness beyond which an additional pathologic process may be inferred. (Females may have more acidity proportional to fold dimensions, although this could reflect more histalog per body weight.)

DISCUSSION

Many indications exist in the medical literature that mucosal parietal cell mass does correlate with maximal acid output stimulated by a variety of agents.[2,4,12,15,17,19,21] This relationship holds with conditions having both increased parietal cell mass and in states with variable degrees of glandular atrophy.[2,12] Increased parietal cell mass, in Z-E particularly, is a result of greater glandular length and actual encroachment of fundal type mucosa into the antrum.[19] Roentgenographically, the Z-E rugal appearance is characterized by increased rugosity and gastric secretions. In advanced gastric atrophy, roentgenographic criteria emphasizing an absence of folds and fundal hypotonia are stated to be accurate in up to 75–80 per cent of cases.[9,17]

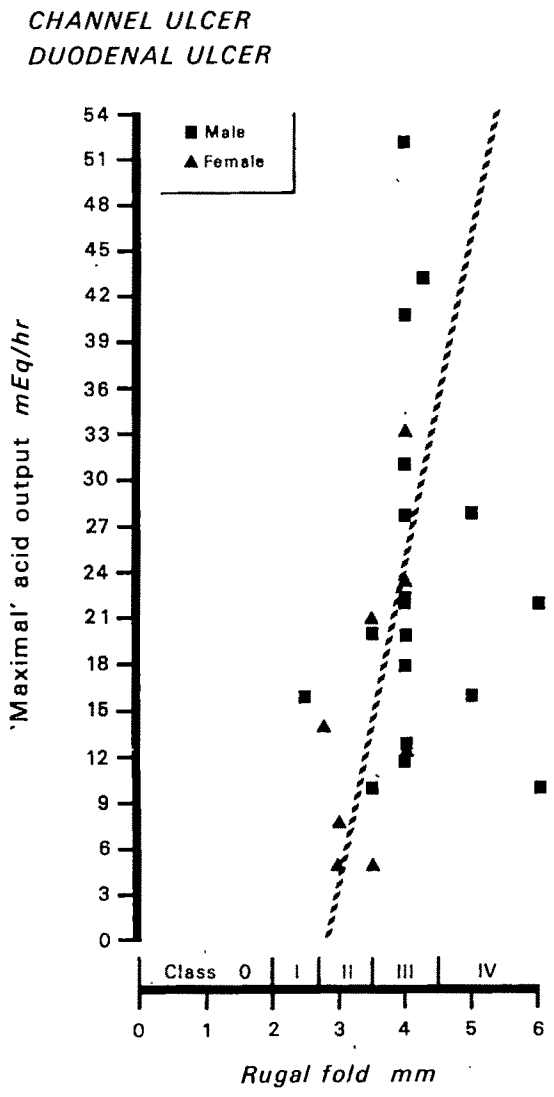

FIG. 7. *Duodenal and Channel Ulcers.* Steeply sloped general fold width to acid output progression. There is, however, a wide range of potential acid outputs especially in classifications II and III.

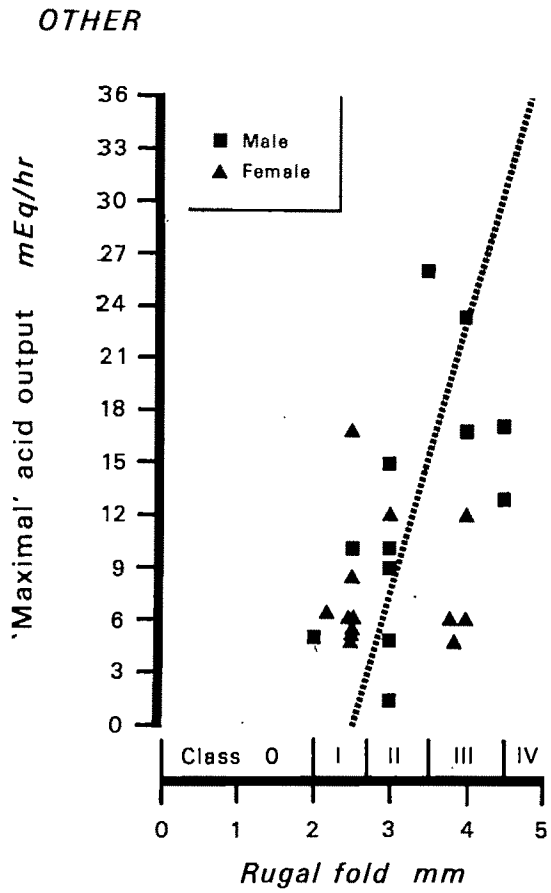

FIG. 8. *Miscellaneous Cases.* Other than with peptic disease. Slope progression and scattergram very similar to Figure 7 but with less peak of acid output.

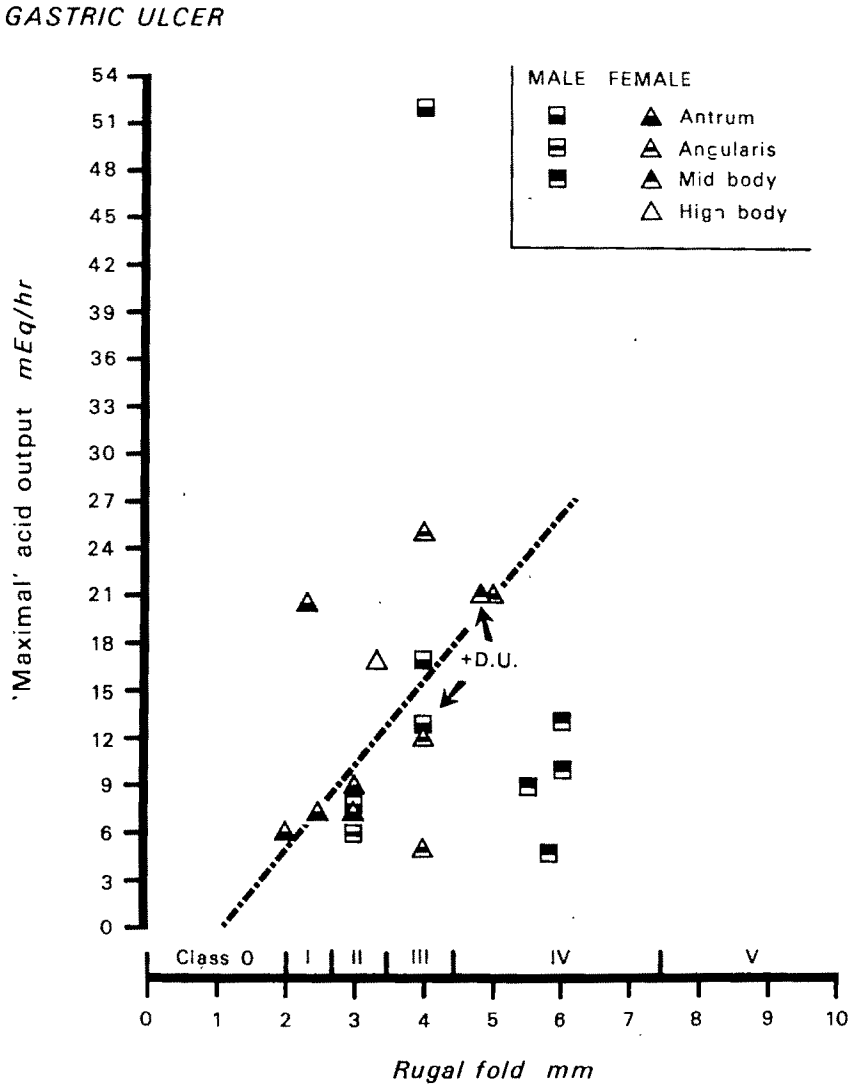

FIG. 9. *Gastric Ulcer Disease.* Most heterogeneous scattergram composed of a mixed population. A progression line is drawn primarily through the "angularis" cases. Note tendency of mid-body GU to skew toward heavier fold and acid output in a manner similar to hypertrophic gastritis. Antral ulcers may follow a progression akin to antral gastritis. (One patient with a high body ulcer does not fit into this pattern, but no additional cases are available.)

Perhaps fittingly, Stempien and more recently Tan *et al.*,[28] have discussed cases of peptic diathesis, often without roentgenographic ulceration, having thickened, nodular rugosity and usually increased secretions. Re-emphasis of thickened, nodular duodenal folds,[7,20] possibly to be differentiated from simple Brunner gland hypertrophy,[10] has assumed significance as part of this hypersecretory pattern, although this is subject to argument.[8] Finally the, roentgenographic perspective offered by Burns and Laws[3] and Moghadam *et al.*[18] claims that in all intervening cases a useful general acid to fold relationship exists. (It should be noted, however, that Burns and Laws found their 13 GU patients more difficult to classify, and not regularly conforming to their thesis. Moghadam *et al.* do not separate their GU and DU cases.)

The pilot study tends to confirm their general thesis while it cautions an inherent

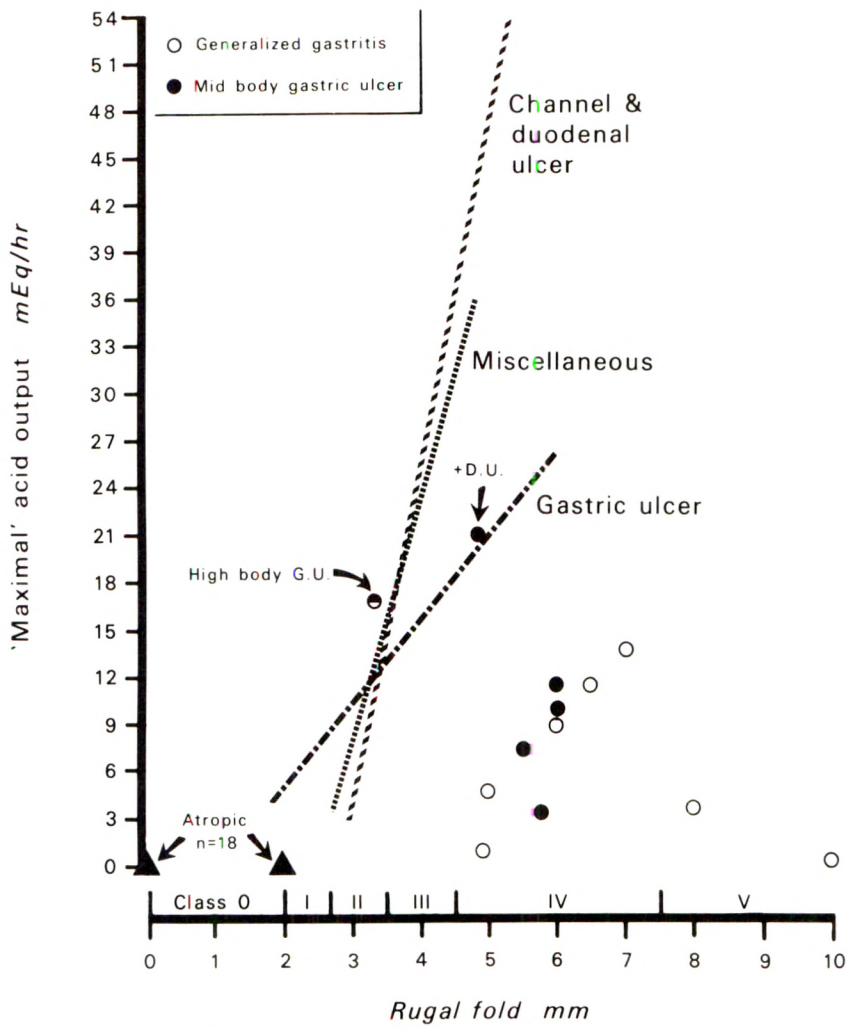

Fig. 10. *Summary of Figures 6–9.* Note the similarity of DU channel ulcer slopes with patients having no apparent ulcer disease, as opposed to the GU group of cases. Note particularly the skewed but similar positions of mid body GU and upper gastric hypertrophic cases. Maximal "physiologic" fold thickness may be 4–5 mm.

weakness. It points toward an additional possibly useful insight.

It is now recognized that gastric and duodenal peptic diseases may result from fundamentally different pathophysiologic events. GU disease may be largely a by-product of decreased mucosal resistance,[1,24] a loss of hydrogen ion barrier created by a variety of toxic agents (*e.g.*, bile reflux,[11] aspirin,[22] alcohol, uremia,[5] etc.), to be distinguished from the hyperacidity-induced duodenal (and probably antral) ulcers. Histologically, a substratum of chronic gastritis having variable degrees of mucosal glandular atrophy has been repeatedly shown in GU patients.[1,15,24] The gastritis is multifocal, predominantly of the acid-bearing mucosal areas, associated with "pylorocardial" expansion of inflamed antral mucosa into the gastric body.[1,15] The GU itself is stated to lie within or near this mucosal "transition zone,"[15] on the antral side, implying a greater degree of generalized mucosal pathology, as the GU lies higher in the stomach. Whereas DU patients have a variable degree of antral

gastritis, the fundal mucosa superiorly is relatively normal[6] or has an increase in glandular size typified in the extreme by Z-E.

It would be simplistic therefore to assume rugal folds indicate acid productivity in these basically different pathologic conditions. More logically, as seen in the scattergram data above, it may be more useful to group GU cases involving the antrum with duodenal cases, and the midbody ulcer cases with those patients having "hypertrophic" gastritis. (Both latter conditions presumably have diverse etiologies and chiefly need differentiation from disease of a diffuse infiltrative malignant nature.)

We now come to the difficult and often frustrating diagnostic problem of "chronic gastritis." From a roentgenographic standpoint, it must be recognized that this truly nonspecific entity is inherently complicated by multiple competing mucosal, and probably submucosal, changes potentially able to produce a spectrum of roentgenographic findings. Glandular atrophy and mucosal fibrosis may coexist with an "interstitial" chronic cellular infiltration within various depths of the lamina propria or submucosa or may compete with actual "proliferative" superficial epithelial or foveolar mitotic activity which can develop into localized pseudopolypoid changes,[14] adenomas, or even an "intestinal type" gastric carcinoma.[15] Initially, at least in the pre-atrophic phase, one may expect an increase in gastric rugosity as witnessed by Robert's study[21] of alcoholic gastritis. (Menetrier's disease may represent a primary mucosal disturbance with or without an inflammatory component.[15])

True physiologic fold thickness, and very broadly, acid secretory capacity, may be maximal at 4–5 mm., beyond which an additional pathological process may be inferred, especially if not supported by other indirect signs of increased acid output. This perspective, along with recognition of the other principles of fold proportionality and clinical examples above, may allow a deeper understanding of a given gastric fold pattern.

A final overview should include other variables participating in formation of the rugal pattern:

1. The gastric mural tone or wall "compliance" probably creates the number and height of the mucosal and submucosal pleatings. (The factor of hypotonia has been advanced in the diagnosis of gastric atrophy, and increased tone is suggested in chronic antral gastritis.)

2. Submucosal thickness and pliancy itself must be critical, although rarely studied without full thickness biopsies.[15] Its role in infiltrative malignancy and in extensive fibrotic states is well recognized.

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REFERENCES

1. AUKEE, S. Gastritis and acid secretion in patients with G.U.D.U. *Scandinav. J. Gastroenterol.*, 1972, *7*, 541–547.
2. BARON, J. H. Clinical applications of gastric secretion measurements. *Clin. Gastroenterol.*, 1973, *2*, 293–314.
3. BURNS, G. P., and LAWS, J. W. Radiologic assessment of gastric acid secretion. *Lancet*, 1966, *1*, 70–72.
4. CARD, W. I., and MARKS, I. N. Relationship between acid output of stomach following maximal histamine stimulation and parietal cell mass. *Clin. Sc.*, 1960, *19*, 147–163.
5. DAVENPORT, H. W. Gastric mucosal barrier. *Digestion*, 1972, *5*, 162–165.
6. DELUCA, V. A., JR., and SPIRO, H. M. Gastric mucosa in acute duodenal ulcer. *A.M.A. Arch. Int. Med.*, 1963, *3*, 363–366.
7. FRASER, G. M., PITMAN, R. G., LAWRIE, J. H., SMITH, G. M. R., FORREST, A. P. M., and RHODES, J. Significance of radiological finding of coarse mucosal folds in duodenum. *Lancet*, 1964, *2*, 979–982.
8. GELZAYD, E. A., GELFAND, D. W., and RINALDO, J. A., JR. Nonspecific duodenitis: distinct clinical entity? *Gastrointest. Endosc.*, 1973, *19*, 131–134.
9. JAMES, W. B., MELROSE, A. G., DAVIDSON, J. W., and RUSSELL, R. I. Radiologic diagnosis of gastritis. *Gut*, 1965, *6*, 372–375.
10. KAPLAN, E. L., DYSON, W. L., and FITTS, W. T., JR. Relationship of gastric hyperacidity to hyperplasia of Brunner's glands. *A.M.A. Arch. Surg.*, 1969, *98*, 636–639.

11. KIM, S. K., ROGERS, L. S., and HEITZMAN, R. E. Reflux alkaline gastritis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *115*, 271–274.
12. KONTUREK, S. J., and URBAN, A. Correlation between gastric acid secretion and histology of fundic and antral gland area. *Scandinav. J. Gastroenterol.*, 1964, *4*, 463–468.
13. KORMAN, M. G., HANKY, J., and STRICKLAND, R. G. Progressive increase in functional G cell mass with age in atrophic gastritis. *Gut*, 1973, *14*, 549–551.
14. KRONE, C. L., and GELFAND, M. D. Gastritis presenting as multiple polyposis of stomach. *Gastroenterology*, 1970, *57*, 703–708.
15. LAMBERT, R. Chronic gastritis. *Digestion*, 1972, *7*, 83–126.
16. MACDONALD, W. C. Gastric biopsy: a critical evaluation. *Gastroenterology*, 1967, *53*, 143–170.
17. MARTIN, J. F., O'BRIEN, T. F., JR., HOLLEMAN, I. L., WALL, G. H., and DUQUE, J. L. Roentgenographic signs in atrophic gastritis and gastric atrophy. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *94*, 343–352.
18. MOGHADAM, M., GLUCKMANN, R., and EYLER, W. R. Radiologic assessment of gastric acid output. *Radiology*, 1967, *89*, 888–892.
19. NEUBURGER, P. Parietal and chief cell populations in four cases of Z. E. syndrome. *Gastroenterology*, 1972, *62*, 937–942.
20. RHODES, J., LAURIE, J. H., and EVANS, K. T. Coarse duodenal folds in patients with peptic ulcer. *Gut*, 1968, *9*, 609–611.
21. ROBERTS, D. M. Chronic gastritis, alcohol, and non-ulcer dyspepsia. *Gut*, 1972, *13*, 768–774.
22. SUGAWA, C., LUCAS, C. E., ROSENBERG, B. F., RIDDLE, J. M., and WATT, A. J. Differential topography of acute erosive gastritis due to trauma, sepsis, ethanol and aspirin. *Gastrointest. Endosc.*, 1973, *19*, 127–131.
23. TAN, D. T. D., STEMPIEN, S. J., and DAGRADI, A. E. Clinical spectrum of hypertrophic hypersecretory gastropathy. *Gastrointest. Endosc.*, 1971, *18*, 69–74.
24. THOMAS, E., HALL, P., and HISLOP, I. G. Histology of gastric mucosa in chronic gastric ulcer. *Am. J. Digest. Dis.*, 1972, *17*, 683–688.
25. VENKATESWARAN, P. S. Gastric acid secretion in chronic renal failure. *Brit. M. J.*, 1972, *4*, 22–23.

MINIMAL DEVIATION HEPATOMA*

A NEW ENTITY

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ABSTRACT:

The term minimal deviation hepatoma has been applied to a specific clinical, pathological, and angiographic entity. It is a tumor, composed of atypical cells, which is neither frankly malignant nor so well organized as a benign tumor. This lesion occurs in females with otherwise normal livers and has a relatively benign course, although life threatening hemorrhage may occur. The diagnosis may be strongly suggested in the presence of hypervascular or hypovascular discrete masses without evidence of frank malignancy, with a defect in the colloid liver scan and with normal liver function tests. Surgical resection when possible and aggressive chemotherapy may be corrective in many cases.

A NUMBER of recent reports[2–4,9,10,11,14,22] have appeared indicating an apparent increase in the incidence of seemingly benign liver tumors occurring exclusively in young females with no previous history of liver disease. A confusing galaxy of terms has been applied to these lesions. These range from hamartoma to adenoma and benign hepatoma. In addition, there has been an implicit etiologic association between oral contraceptives and these tumors in that all previously reported patients had received these hormones.

We are presenting 4 new cases of similar liver tumors, all in women without previous liver disease. We are introducing the term "minimal deviation hepatoma" (MDH) which we feel best describes these lesions of behavior intermediate between the obviously benign and the frankly malignant. Only 1 of these 4 patients had been exposed to hormonal therapy.

REPORT OF CASES

CASE 1. M.R. is a 35 year old Puerto Rican female who presented with acute right upper quadrant pain. She had no previous history of liver disease, hepatitis, alcohol intake, or gallbladder disease. She was on no medication and was the mother of 3 living, normal children. On the day of admission her hematocrit dropped from 43 to 30 per cent and a tender right upper quadrant fullness became apparent. Her liver function tests were normal as were her chest roentgenogram and electrocardiogram. The alpha fetoprotein was negative.

A colloid scan of the liver showed multiple intra-hepatic defects. Barium enema and upper gastrointestinal examinations were negative. A selective hepatic arteriogram demonstrated multiple vascular hepatic masses and a subcapsular, right-sided hematoma (Fig. 1, *A–D*).

The patient was explored and was found to have multiple, firm masses in the right and left lobes of the liver. Biopsy of one of the lesions was initially interpreted as hamartoma, but was later felt to represent a minimal deviation hepatoma.

She did well postoperatively but had a persistent low-grade fever and right pleural effusion. Complete work-up of the pleural fluid and blood was negative. She did not respond to antibiotics, but her fever disappeared dramatically following Indocin 25 mg. *q. i. d.*

She received no chemotherapy or radiation therapy and was discharged without symptoms but a persistent anemia with a hematocrit of 37 per cent.

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.

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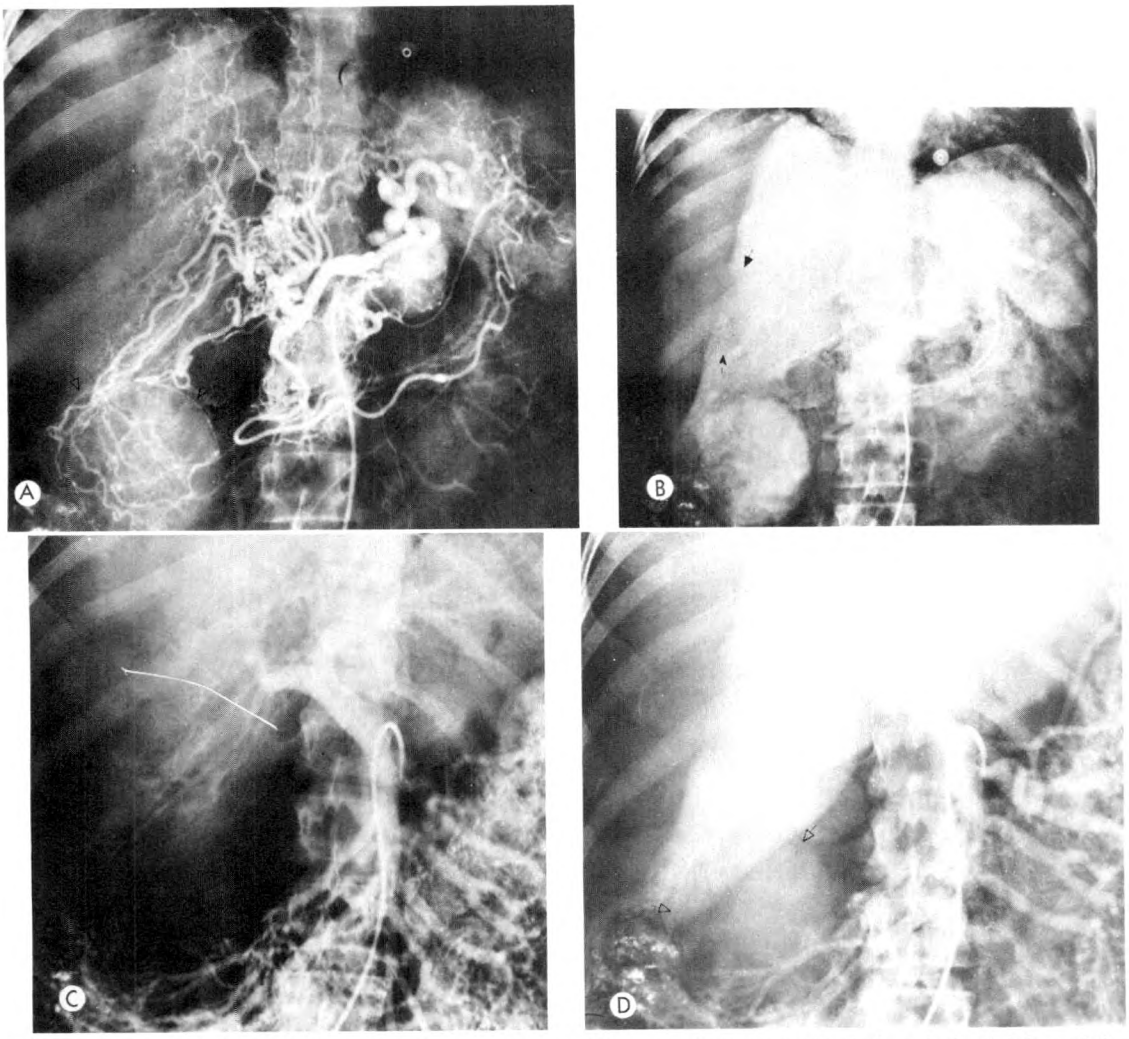

Fig. 1. Case 1. (*A*) Arterial phase of celiac arteriogram. There are hypervascular lesions in both lobes of the liver. One lesion is pedunculated from the inferior right lobe (arrows) with peripheral arteries coursing centripetally. (*B*) The large subcapsular hematoma associated with a peripheral lesion (arrows) is best seen in the capillary phase. (*C*) Portal venous flow is hepatopetal and the portal radicles are patent. (*D*) Late portal phase shows no evidence of venous laking or arteriovenous shunting. The negative blush of the pedunculated lesion (arrows) confirms its arterial, rather than portal venous, blood supply.

The patient chose to return to Puerto Rico to be closer to her family. At last report she is alive and well 15 months after her diagnosis without further therapy.

Case II. E.M. is a 30 year old white female who was entirely well until 3 days prior to her admission when she was admitted to another hospital with acute, severe epigastric pain, fever of 102° and anemia with a hematocrit which fell from 36 to 31 per cent in 24 hours. Her white blood cell count was 17,000, SGOT 312, alkaline phosphatase 136 and LDH 1300 units. Bilirubin was 0.8 and amylase 32 milligrams per cent. Alpha fetoprotein was negative.

She had no significant past medical history but had been on birth control pills for 5 years following the birth of her third child. She underwent exploratory laparotomy where a huge mass involving both lobes of the liver was seen. There was obvious, extensive hemorrhage into the lesion. Biopsy showed only necrotic liver cells.

She was transferred to this hospital for further work-up and treatment. A liver scan done with technetium 99^{m} sulfur colloid showed

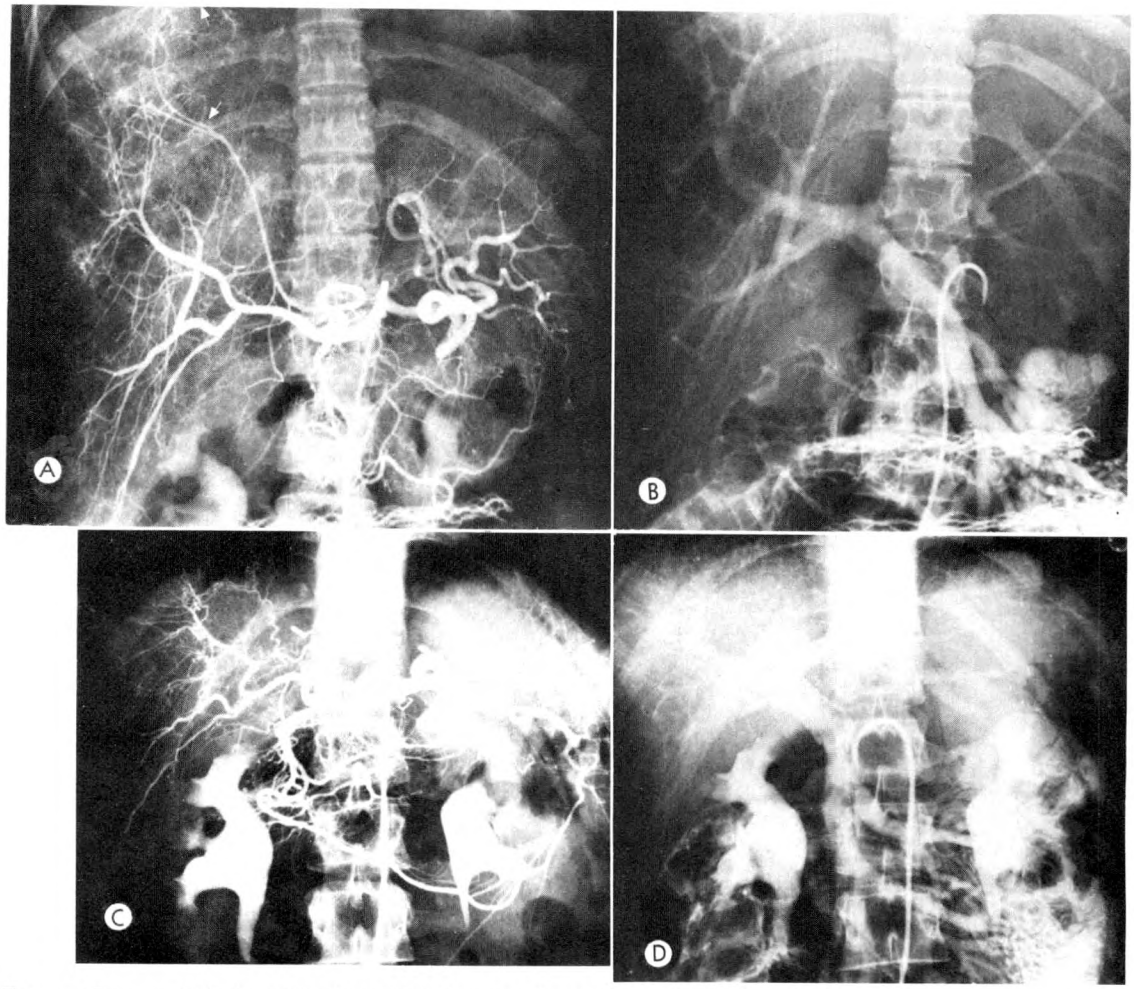

FIG. 2. Case 11. (*A*) Arterial phase of celiac arteriogram showing enlarged liver with avascular mass extending into the dome of the right lobe (arrows). (*B*) Portal venous phase confirming a mass between the right and left lobes. The portal vein radicles are patent. (*C*) Follow-up angiogram 15 months later demonstrates a normal size liver with no appreciable mass or neovascularity in the region of the previous abnormality (surgical clip). (*D*) Portal veins remain normal.

a huge 14 cm. "cold" area between the right and left lobes. The hepatic arteriogram demonstrated a hypovascular mass with peripheral neovascularity and normal portal veins (Fig. 2, *A* and *B*).

On the twentieth hospital day, after marked clinical improvement she underwent surgical exploration in an attempt to resect the lesion. The mass involved the right and left lobes of the liver and was markedly hemorrhagic and necrotic. The major bulk of the mass was indeed hematoma between the hepatic lobes. Some firm areas of tumor were present, and biopsy of these areas confirmed the diagnosis of minimal deviation hepatoma. The lesion was felt to be unresectable and following evacuation of the hematoma the abdomen was closed.

Following operation she did remarkably well. Her temperature returned to normal and all enzymes and liver function tests became normal. It was felt that the large hematoma had produced hepatic vein compression and a congested liver. She was discharged 5 weeks after admission.

Ten days later she was re-admitted for a course of chemotherapy with 5- Fluorouracil and BCNU which she tolerated well. A second course of chemotherapy was given 8 weeks later.

She became completely asymptomatic, and 8 months following surgery, she was re-admitted for liver scan and arteriography. The former

showed a persistent small interlobar defect where the tumor had been, and the angiogram showed marked resolution toward normal with disappearance of frank neovascularity.

A third angiogram, 15 months after her original diagnosis, was unchanged (Fig. 2, *C* and *D*). During the past year she has remained asymptomatic and in apparent good health. Her only therapy has consisted of surgical drainage of the hematoma and 2 courses of 5-FU and BCNU.

Case III. H.P. is a 34 year old Black female who was in good health until 1 month before her final admission when she underwent a total abdominal hysterectomy for uterine fibroids and chronic pelvic inflammatory disease. At the time of operation an 8×10 cm. liver mass was palpated. The wound was closed, and a subsequent liver scan showed a large defect in the right lobe of the liver. An arteriogram showed a 13 cm. avascular lesion involving the right inferior portion of the liver (Fig. 3, *A–C*). Liver function studies showed bilirubin 0.6 milligrams per cent, alkaline phosphatase 155, SGOT 325, and LDH 650 units. Her hematocrit was 37 per cent and white blood cell count 5,000. Alpha fetoprotein was negative. She had no palpable mass, was asymptomatic, and gave no history of past medication or birth control pills.

An open liver biopsy revealed a solid, hemorrhagic, necrotic tumor of the liver. Frozen section showed only necrotic tissue. A larger resectional biopsy was done, and the abdomen closed. Permanent sections revealed a minimum deviation hepatoma.

Accordingly, 3 days later, a total resection of the lesion was successfully carried out, and the diagnosis of necrotic minimal deviation hepatoma confirmed. She had an uneventful recovery and has remained asymptomatic for the past 9 months. Her blood chemistries are now normal and follow-up liver scans show no evidence of tumor.

Case IV. A.R. is a 67 year old mother of 16 children from the Dominican Republic who presented with anorexia and epigastric pain. She had no significant past medical history. Her epigastric pain was sudden in onset and very severe. Her private physician felt a firm mass in the epigastrium and referred her for treatment.

Her physical findings were unremarkable except for an 8 cm. firm epigastric mass. Her white blood cell count was 8,200, hematocrit 38 per cent, SGOT 89, alkaline phosphatase 72 units and bilirubin 1.3 milligrams per cent. Alpha fetoprotein was negative. Barium studies were negative. A liver scan was not performed. Celiac and superior mesenteric arteriography showed 2 large vascular mass lesions involving both lobes of the liver (Fig. 4, *A–C*). There was no arteriovenous shunting, and the portal veins were normal.

An open liver biopsy was performed on 1 of the tumor masses. There was considerable difficulty in achieving hemostasis since the tumor was extremely vascular and friable. Pathology revealed a well differentiated tumor compatible with a minimal deviation hepatoma. She did well after biopsy and was followed for 1 year without progression of the lesion or recurrence of pain. She was been lost to follow-up for the past year.

Pathologic Considerations

The anatomical unit of the human liver is an ill-defined lobule which is composed of hepatic cells arranged in 2 cell thick plates. Between the cell plates run the sinusoids, modified capillaries lined with a specialized endothelial cell called the Kupffer cell. These cell plates and sinusoids converge toward the central veins. The portal spaces are located at the periphery of each lobule. They are composed of radicles of the portal vein, the hepatic artery and the bile duct. Bile flows from the cells to the canaliculi between adjacent cells and then peripherally to the duct in the portal space. Portal venous and hepatic arterial blood flows from the portal spaces centrally, mixing in the sinusoids, to the hepatic vein. This orderly arrangement is altered by any growth disturbance or neoplastic process within the liver. The following discussion of lesions of hepatocellular origin is an attempt to clear up some of the confusing terms.

A general definition of a hepatic adenoma is a benign neoplasm, usually solitary and well circumscribed if not encapsulated, which is composed of "normal" to slightly atypical hepatic cells vaguely arranged in cords without lobular architecture and devoid of portal tracts, central veins, and bile ducts. Grossly, it is tan to light brown,

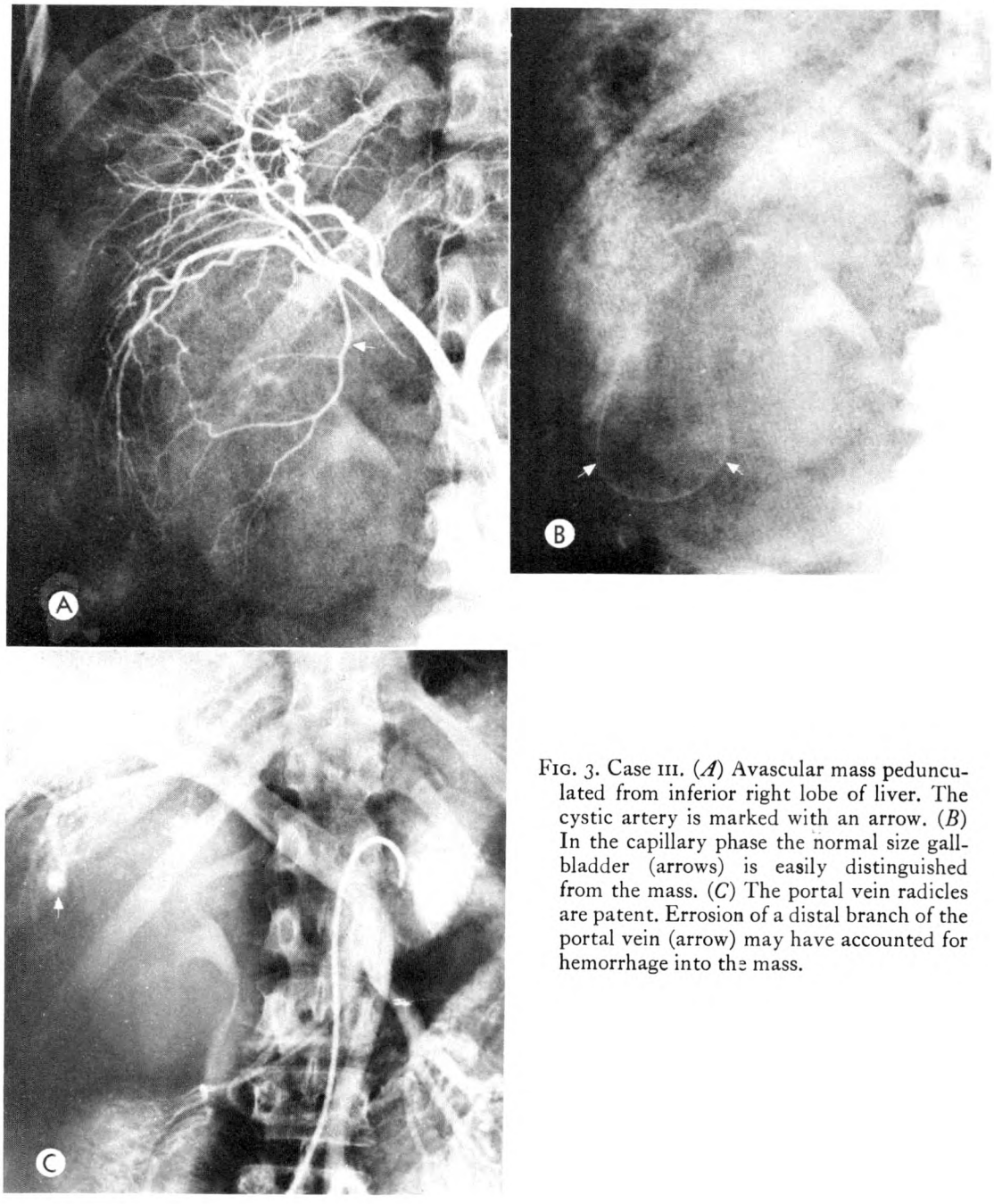

FIG. 3. Case III. (*A*) Avascular mass pedunculated from inferior right lobe of liver. The cystic artery is marked with an arrow. (*B*) In the capillary phase the normal size gallbladder (arrows) is easily distinguished from the mass. (*C*) The portal vein radicles are patent. Errosion of a distal branch of the portal vein (arrow) may have accounted for hemorrhage into the mass.

projects from the surface of the liver and may be pedunculated. The adjacent liver should be normal. Such a lesion occurring in a cirrhotic liver is extremely rare, if it occurs at all. However, somewhat similar lesions are seen in cirrhosis and correspond to the nodular appearance of the liver in that disease. Occasionally, these nodules may grow to a large size and present a problem of diagnosis. They are more correctly termed regenerating nodules. Although there is cellular proliferation with loss of lobular architecture (as is true with an adenoma) they tend to have bile ducts at the periphery. To make the diagnosis of adenoma in cirrhosis, there should be com-

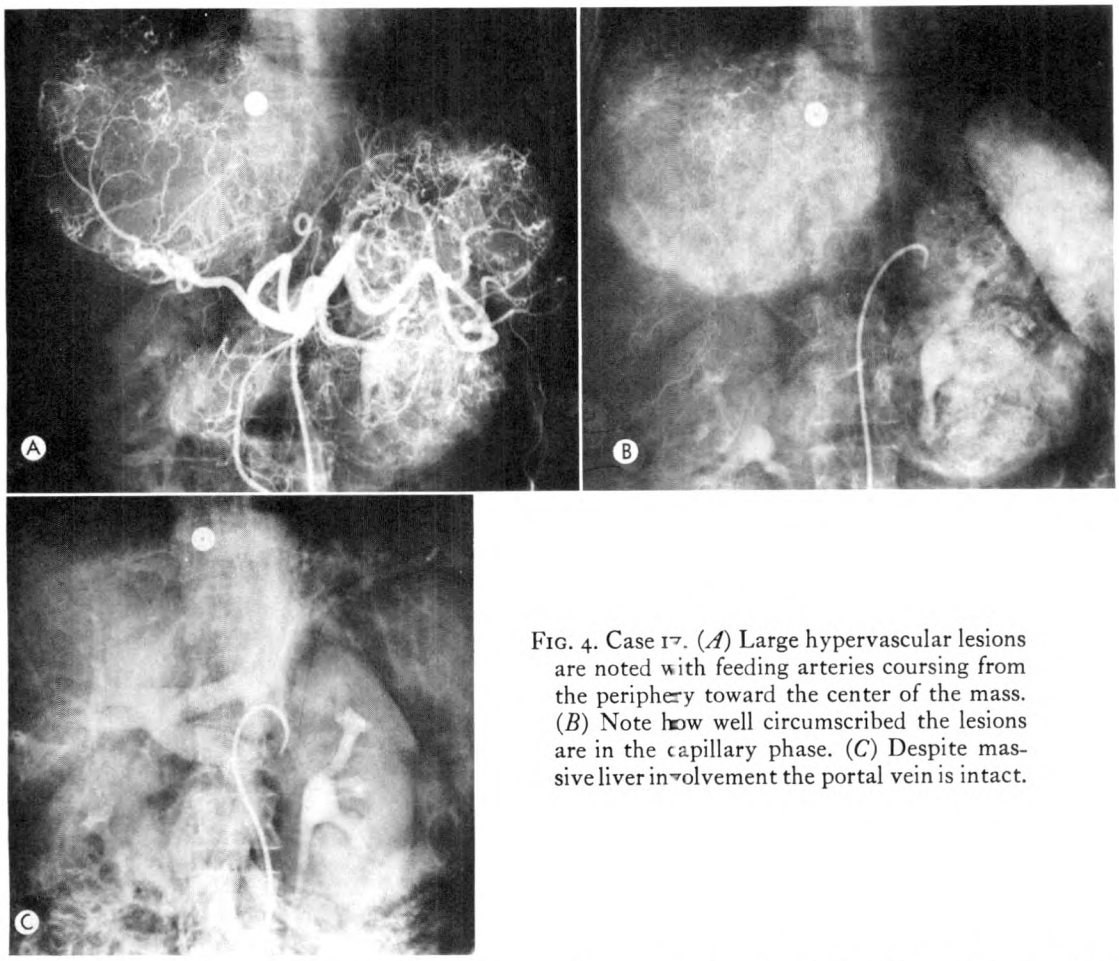

Fig. 4. Case 17. (*A*) Large hypervascular lesions are noted with feeding arteries coursing from the periphery toward the center of the mass. (*B*) Note how well circumscribed the lesions are in the capillary phase. (*C*) Despite massive liver involvement the portal vein is intact.

plete absence of any such ducts.

Occasionally, masses will be encountered in an otherwise normal liver which in one area of the lesion will fulfill the criteria for an adenoma while in other areas there will be a proliferation of bile ducts and excessive fibrous tissue. Using strict criteria, these cannot be called true adenomas and, according to Popper and Schaffner[16] should be called solid hamartomas. These improperly organized structures straddle the line between a malformation and a neoplasm.

In addition to the 2 above-mentioned benign masses, focal areas of benign hyperplasia occasionally develop in otherwise normal livers and differ slightly from the nodular regeneration seen in cirrhosis. Since there has been no known tissue insult, it is difficult to accept them as reparative phenomena (*i.e.*, regenerating nodules). Like the adenoma, they may be subcapsular or pedunculated and occur most often in women and children. Grossly, there is a central stellate mass of connective tissue from which radiate fibrous strands separating the proliferating cells into pseudolobules. The cells everywhere are related to bile ducts and their accompanying connective tissue, in contrast to the true adenoma. This lesion is called focal nodular hyperplasia (FNH) and seems to be a distinct entity.

A frankly malignant tumor of hepatocellular origin (malignant hepatoma) will exhibit mitotically active atypical cells replacing adjacent parenchyma, or, in cirrhotic livers, replacing pseudolobules between fibrous tracts. Grossly, it is soft and friable with a great variety of colors due to a mixture of bile secretion, necrosis, fatty change and hemorrhage. Tissue and vascular invasion is common.

A hepatocellular lesion which does not meet the strict criteria for one of the benign lesions and is not frankly malignant might be classified as an intermediate tumor or carcinoma *in situ* since the experimentalists have developed such a tumor with only slightly atypical cells, which lacks local invasiveness and distant metastases.[17] The term "minimal deviation hepatoma" has been used to describe this tumor, which differs the least from the normal tissue in terms of its biochemical and enzymatic potential, for the purposes of designing a simple cancer model.[13,17] It became apparent that this description includes the biological behavior of the tumor (*i.e.*, growth potential) as well as biochemical aberrations.[6]

It is our contention that most of the recently reported cases of relatively benign liver tumors with minimally atypical cytology best fit the description of the minimal deviation hepatoma. To our knowledge no such tumor has regressed spontaneously, a point used to justify aggressive therapy. The fate of these lesions if left untreated is not known, but no one has observed a frankly malignant transformation of this tumor (*i.e.*, metastasizing behavior). Thus, minimal deviation hepatoma seems to be a good term to describe the lesion which currently is being treated as a low grade hepatocellular carcinoma.

ROENTGENOGRAPHIC FEATURES

The angiographic features of the 4 cases herein reported are notably variable. Lesions which were hypervascular had large feeding arteries which began peripherally in the mass and coursed centripetally toward the center (Fig. 1*A* and 4*A*). Bizarre permeating neovascularity seen in malignant hepatomas was not observed. The lack of venous laking, arteriovenous shunting and portal vein invasion also distinguished these lesions from their malignant counterparts. Other masses were principally avascular (Fig. 3). Exploration in these instances revealed either central necrosis or intrahepatic and subcapsular hematomas. Single and multiple nodules were seen with both types and each mass was well demarcated from adjacent normal liver tissue in the capillary phase of the angiogram. Some lesions were located deep in the liver substance while others were subcapsular or pedunculated and associated with subcapsular hematomas.

Angiographic features of benign liver masses (FNH, adenoma, hyperplastic nodule, solid hamartoma and benign hepatoma) have been described elsewhere.[7,12,16] Hyper vascular, well circumscribed lesions lacking bizarre neovascularity, venous laking, and vascular invasion are general descriptive terms applied to these tumors. The similarity of this pattern with those we have observed in MDH further supports the contention that we are dealing with similar lesions with only minor variations in their histology.

Scintillation scanning theoretically would be of some advantage in differentiating MDH from the various other "benign" lesions and malignant hepatomas. Agents used to detect RE system function (*i.e.*, technetium 99^{m} sulfur colloid) show decreased uptake in all these lesions.[12,19] There should be better uptake in FNH, regenerating nodules, and solid hamartomas, which have more connective tissue, than in adenomas which have only proliferating hepatocytes. On the other hand, agents reflecting hepatocyte function (*i.e.*, I^{131} rose bengal) should demonstrate better uptake in a proliferating lesion of functioning hepatocytes like the adenoma than in the other lesions made up of a mixture of both. The difference in uptake, however, is quite variable due to the relative amount of functioning tissue with respect to the adjacent normal liver tissue. Also, well differentiated hepatocellular carcinomas have been shown to have some function with these studies.[20] Blood pool agents such as indium 113^{m} simply reflect the vascularity of the mass with no further differentiation. Gallium[67] has been shown to have a specificity for malignant hepatoma and may be useful in distinguishing that lesion.[21]

In summary, radionuclide scanning is very sensitive in detecting masses in the

liver but is relatively nonspecific in differentiating the lesions.

DISCUSSION

The cases reported here and the 19 previously reported cases represent a distinct clinical and pathological entity.

The clinical setting is similar in that the patients are usually female with no known pre-existing liver disease and in a young age group. The liver function studies are normal or minimally deranged. Fever indicates an underlying necrosis and anemia may reflect blood loss. Hemorrhage and shock may occur with the rupture of a superficial mass. One patient presented with a Budd-Chiari like syndrome due to compression of hepatic veins from a deep seated mass (Case 11). The usual presenting complaint, however, is a falling hematocrit or an enlarging mass.

The question of hormonal influence has been raised. The effect of hormones on the liver has been studied experimentally and clinically. The changes consist of development of abnormal bile canaliculi causing cholestasis.[1] Ultrastructure changes in the mitochondria[8] and dilatation of the periportal sinusoids similar to peliosis hepatis[18] have been noted. No cellular atypia or mitoses has been produced. Although the association has been reported, a cause and effect relationship has not been established.

The roentgenologic features include a defect in the colloid liver scan. Although the various scanning agents are very sensitive in detecting the masses, they have not been very helpful in differentiating these lesions from other masses. Angiography is helpful in the diagnosis of these lesions and in the planning of resection. The pattern may be either hypervascular or hypovascular and the lesions may be deep or peripheral and pedunculated. The lack of frankly malignant features and the frequent association of necrosis and subcapsular hematoma aid in the diagnosis. Although the angiographic pattern can be suggestive in the proper clinical setting, it is by no means pathognomonic.

Pathologically, these masses are discretely separated from normal adjacent liver substance and no tissue or vascular invasion is seen (Fig. 5). The cells are proliferating in sheets with loss of lobular architecture and polarity of blood flow. Cellular atypia is common but mitotically active cells are absent.

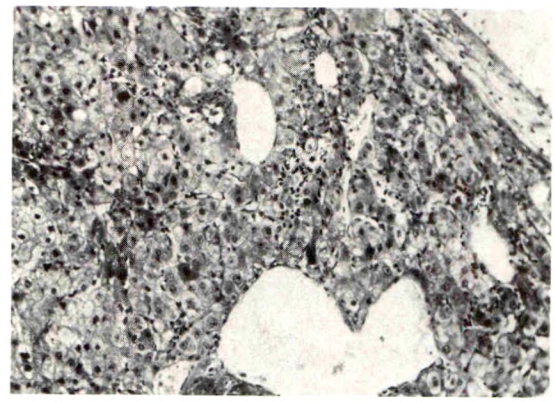

Fig. 5. Histologic section is representative. There is loss of the lobular arrangement with proliferation of atypical cells. The lesion lacks cellular mitosis and tissue and vascular invasion.

A benign course ensues although acute complications of hemorrhage and necrosis can be life threatening. It should be noted that 4 out of 19 previously reported cases died following an attempt at radical resection. Response to surgery or chemotherapy seems to be good, although the follow-up is limited. Regression of the masses on chemotherapy was noted in 1 of our cases (Case 11). Spontaneous regression of tumors has never been observed, a fact used to justify therapy. At the present time therapy should be directed at limited resection of accessible lesions, since radical resection may carry a morbidity greater than that of the lesion itself. Non-resected masses showing cellular atypia may be given a trial of chemotherapy with the hope of regression and cure. Untreated lesions still carry the hazard of acute rupture and hemorrhage.

We feel that many, if not all, of the cases of liver tumors in young women being currently reported belong in this "intermediate" category of minimal deviation hepatoma. Our reasons for choosing that term have been outlined in the pathological section of this manuscript. Briefly, one should

apply the strict pathologic criteria suggested by Edmondson[5] before classifying a benign hepatocellular lesion of the liver as an adenoma or benign hepatoma. Not all pathologists are in agreement with these criteria and, indeed, Popper and Schaffner[16] prefer the term solid hamartoma since usually there is a mixture of cellular and glandular elements. If in addition there is cellular atypia then MDH should apply. No one has observed one of these lesions to progress to a frankly malignant hepatocellular carcinoma, although experimentally such alterations have occurred. Perhaps in time this question can be answered clinically since there are cases now being reported where resection of the lesion has not been possible.

To differentiate these lesions clinically is impossible. When presented with a previously healthy female patient with a right upper quadrant mass, epigastric pain, a defect in the liver scan, or a hemoperitoneum, the diagnosis of MDH should be considered and verified by angiography.

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REFERENCES

1. ADLERCREUTZ, H., and TENHUNEN, R. Some aspects of interaction between natural and synthetic sex hormones and liver. *Am. J. Med.*, 1970, *49*, 630–648.
2. ALBRITTON, D. R., TOMPKINS, R. K., and LONGMIRE, W. P., JR. Hepatic cell adenoma: report of four cases. *Ann. Surg.*, 1974, *180*, 14–19.
3. BAUM, J. K., HOLTZ, F., BOOKSTEIN, J. J., and KLEIN, E. W. Possible association between benign hepatomas and oral contraceptives. *Lancet*, 1973, *2*, 926–929.
4. CONTOSTAVLOS, D. L. Benign hepatomas and oral contraceptives. *Lancet*, 1973, *2*, 1200.
5. EDMONDSON, H. A. Tumors of the Liver and Intrahepatic Bile Ducts. Armed Forces Institute of Pathology, Washington, D. C., 1958, pp. 18–22.
6. FARBER, E. On concept of minimal deviation in study of biochemistry of cancer. *Cancer Res.*, 1968, *28*, 1210–1211.
7. GOLDSTEIN, H. M., NEIMAN, H. L., MENA, E., BOOKSTEIN, J. J., and APPLEMAN, H. D. Angiographic findings in benign liver cell tumors. *Radiology*, 1974, *110*, 339–343.
8. GORODISCH, S., PEREZ, V., DE MARTIER, J., NICHOLSON, R., and DI PAOLA, G. Oral contraceptives: long-term use produces fine structural changes in liver mitochondria. *Science*, 1969, *165*, 805–807.
9. HORVATH, E., KOVACS, K., and ROSS, R. C. Ultrastructural findings in well-differentiated hepatoma. *Digestion*, 1972, *7*, 74–82.
10. KELSO, D. R. Benign hepatomas and oral contraceptives. *Lancet*, 1974, *1*, 315–316.
11. KNAPP, W. A., and RUEBNER, B. H. Hepatomas and oral contraceptives. *Lancet*, 1974, *1*, 270–271.
12. MCLOUGHLIN, M. J., and GILDAY, D. L. Angiographic and colloid scanning of benign mass lesions of liver. *Clin. Radiol.*, 1972, *391*, 377–391.
13. MORRIS, H. P. Studies on development, biochemistry and biology of experimental hepatomas. *Advances Cancer Res.*, 1965, *9*, 227–302.
14. O'SULLIVAN, J. P., and WILDING, R. P. Liver hamartomas in patients on oral contraceptives. *Brit. M. J.*, 1974, *3*, 7–10.
15. PALUBINSKAS, A. J., BALDWIN, J., and MCCORMACK, K. R. Liver-cell adenoma: angiographic findings and report of case. *Radiology*, 1967, *89*, 444–447.
16. POPPER, H., and SCHAFFNER, F. Liver: Structure and Function. McGraw-Hill Book Company, Inc., New York, 1957, pp. 587–612.
17. POTTER, U. R. Biochemical Studies on Minimal Deviation Hepatoma in Cellular Control Mechanisms and Cancer. Elsevier Publishing Co., Amsterdam, 1964, pp. 190–210.
18. POULSEN, H., and WINKLER, K. Liver disease with periportal sinusoidal dilatation. *Digestion*, 1973, *8*, 441–442.
19. SACKETT, J. F., MOSENTHAL, W. T., HOUSE, R. K., and JEFFERY, R. F. Scintillation scanning of liver cell adenoma. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 56–60.
20. SHOOP, J. D. Functional hepatoma demonstrated with rose bengal scanning. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *107*, 51–53.
21. SUZUKI, T., HONJO, I., HAMAMOTO, K., KOUSAKA, T., and TORIZUKA, K. Positive scintiphotography of cancer of liver with Ga^{67} citrate. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 92–103.
22. TOUNTAS, C., PARAKEVAS, G., and DELIGEORGI, H. Benign hepatomas and oral contraceptives. *Lancet*, 1974, *1*, 1351–1352.

A NEW SYNDROME OF LEFT VOCAL CORD PARESIS AND ESOPHAGEAL DIVERTICULUM DUE TO MEDIASTINAL FIBROSIS*

By M. A. LERNER *and* R. KATZ
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ABSTRACT:

The association of left vocal cord paresis with an esophageal diverticulum pointing to the left at the aorto-bronchial level constitutes a syndrome resulting from mediastinal fibrosis in that region. Demonstration of this type of diverticulum in a case of unexplained left cord paresis establishes mediastinal fibrosis as its cause.

VOCAL cord paresis is twice as frequent on the left as on the right due to the long intrathoracic course of the left recurrent laryngeal nerve, beginning with its loop between the aortic arch and the left main bronchus (Fig. 1). Of the numerous lesions impairing conduction in this nerve, only one passing mention implicating peri-

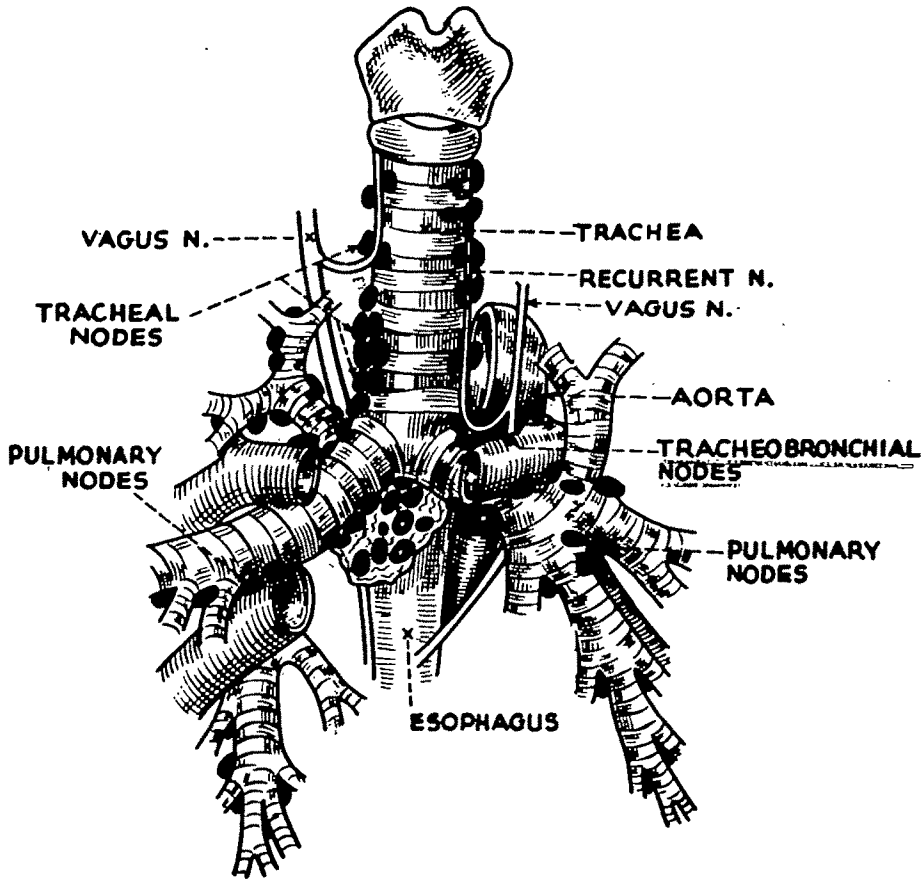

FIG. 1. Left tracheobronchial lymph nodes in relation to the aortic arch, left recurrent laryngeal nerve, and the esophagus (posteriorly). (From Morris, *Human Anatomy*.)

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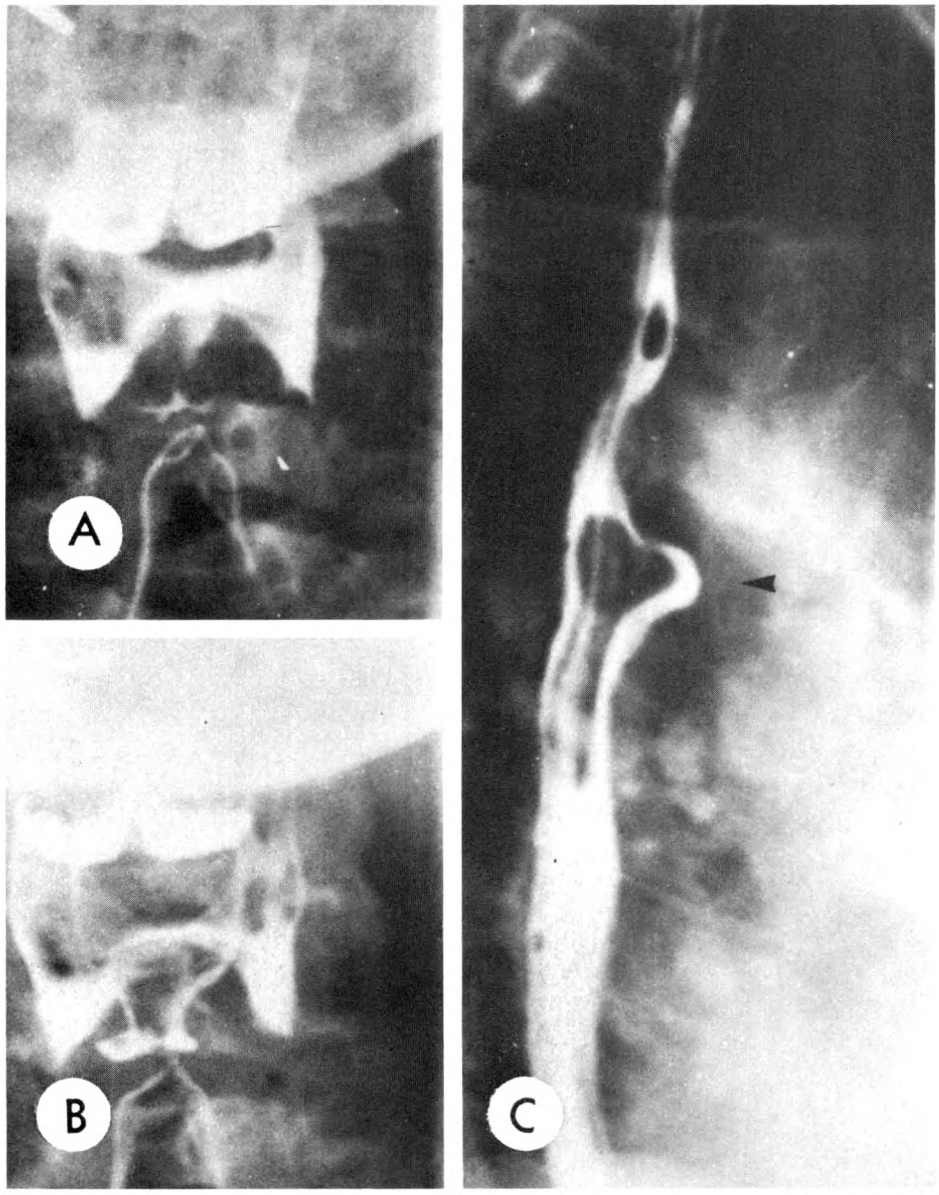

FIG. 2. (*A* and *B*) Laryngography. (*A*) Obliquity of laryngeal fissure in Valsalva maneuver. (*B*) Phonation with left cord and vestibular fold in "cadaveric position" of recurrent nerve paresis, and right cord meeting it to the left of the midline. (*C*) Diverticulum of esophagus at aorto-left-bronchial level.

lymphadenitis has been traced.[2] On the other hand, in several series, the etiology of left vocal cord paresis is listed as idiopathic in up to 25 per cent of cases.

Mediastinal fibrosis accompanying chronic lymphadenitis, usually tuberculous, is the commonest cause of acquired diverticulum of the middle third of the esophagus. Adherence of the esophagus to the lymph nodes anteriorly leads to formation of a traction diverticulum as the aortic arch, unfolding in later life, draws the esophagus backwards.[3]

In a patient with both hoarseness and discomfort on swallowing, left vocal cord paresis and a middle third esophageal di-

verticulum were demonstrated (Fig. 2, *A–C*). Since vocal cord paresis usually leads to examination of the mediastinum and the esophagus, another case soon came to light. A search was then initiated for left cord paresis among all patients found to have esophageal diverticulum. Analysis of the results is presented in Figure 3. The connection between the two conditions becomes apparent when the diverticula are considered with regard to the precise location. With the more frequent interbronchial location no cord paresis was encountered, whereas cord paresis was discovered in 4 out of 5 diverticula pointing to the left at the aorto-bronchial level (Fig. 4). Actual development of the diverticulum was observed in a case of left cord paresis followed for 4 years (Fig. 5, *A* and *B*).

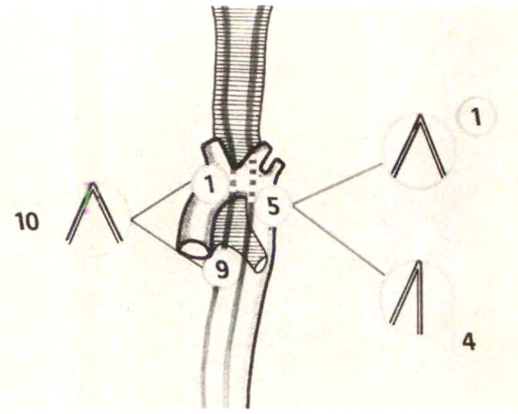

Fig. 3. Fifteen esophageal diverticula. Four out of 5 pointing to the left at the aorto-bronchial level had left vocal cord paresis.

Significantly there was no cord paresis associated with a diverticulum at the aorto-bronchial level but pointing to the right.

The importance of lymph nodes in close

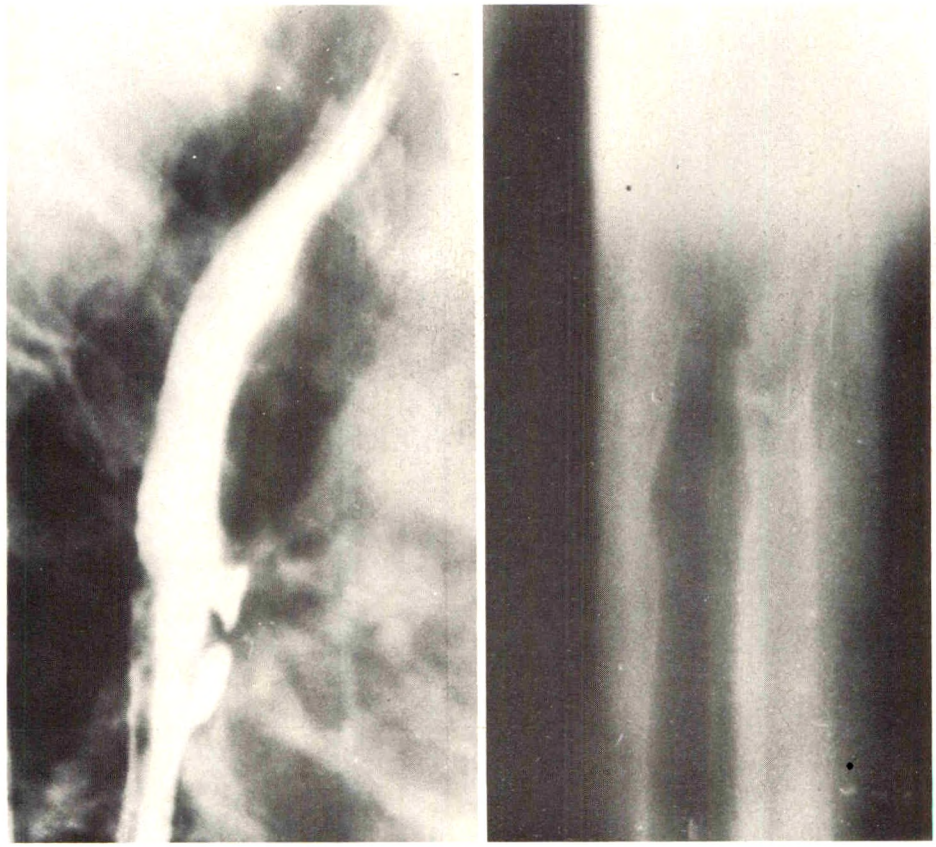

Fig. 4. Left cord paresis discovered in patient with diverticula at interbronchial (subcarinal) and aorto-bronchial levels.

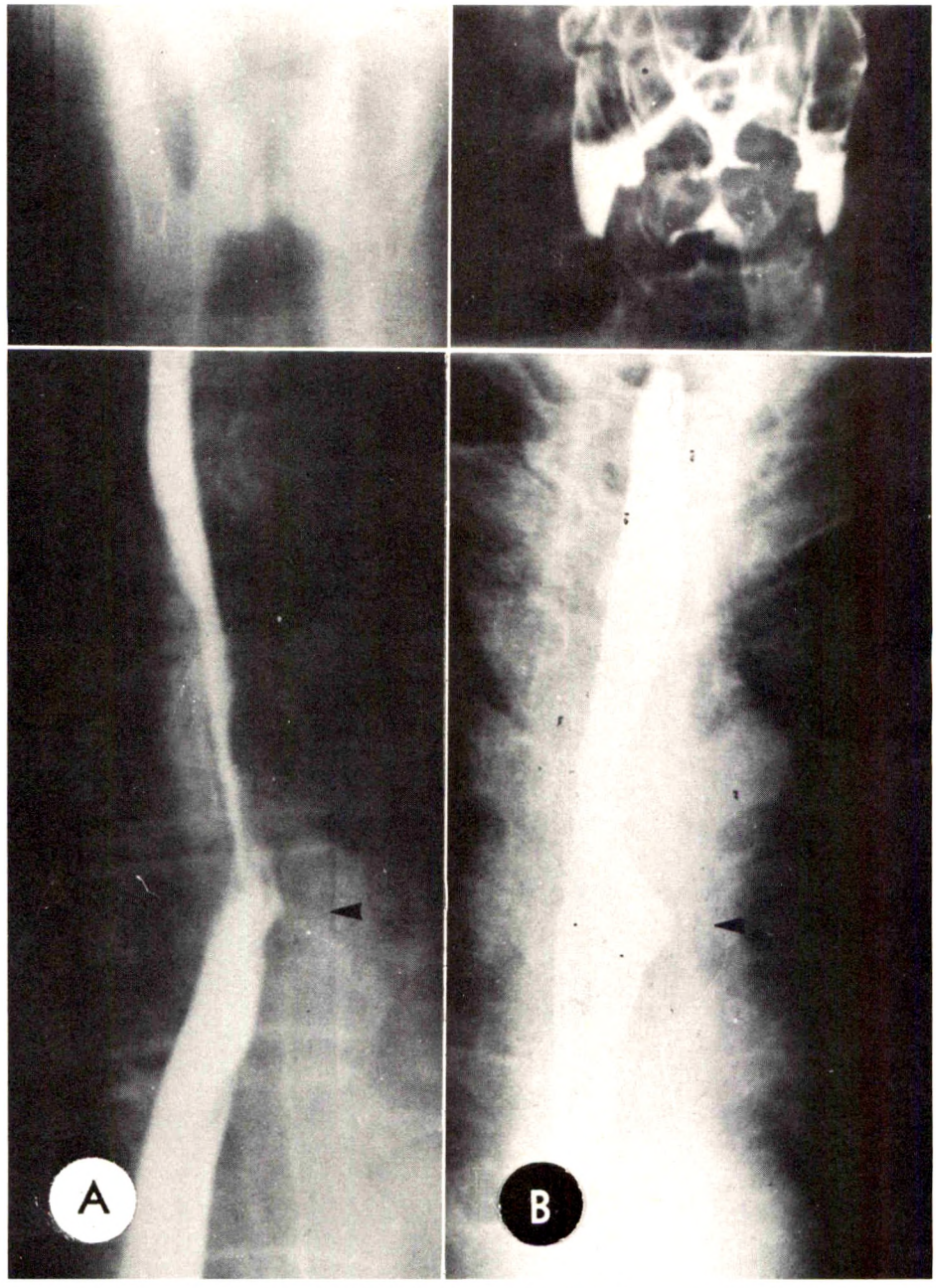

FIG. 5. (*A*) 1970; left cord paresis and slight esophageal traction by aorta. (*B*) 1974; same cord paresis (laryngography) and esophageal diverticulum at aortic arch level.

proximity to both the esophagus and the origin of the left recurrent laryngeal nerve was pointed out by Fleischner *et al.*[1] in connection with the esophageal deformity due to metastatic lymph nodes from carcinoma of the bronchus and often announced by left cord paresis.

Left cord paresis may cause only mild hoarseness or may even be asymptomatic because the cricothyroid muscle, innervated

by the superior laryngeal nerve, can compensate for intrinsic adductor weakness. The opposite cord also crosses the midline to permit phonation. The cord paresis in 2 of the patients in the diverticulum series was unsuspected; mild huskiness is a common feature of aging which is not usually investigated.

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REFERENCES

1. Fleischner, F. G., and Sachsse, E. Retrotracheal lymphadenopathy in bronchial carcinoma, revealed by barium filled esophagus. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 792–798.
2. Leech, T. R., Meckstroth, C. V., and Klasse, K. P. Exploratory thoracotomy in chronic lymphadenitis of mediastinum. *A.M.A. Arch. Surg.*, 1955, *71*, 383–391.
3. Shanks, S. C., and Kerley, P. A Textbook of X-Ray Diagnosis. Third edition. Volume 3. W. B. Saunders Company, Philadelphia.

ROENTGENOGRAPHIC FINDINGS IN TYPHOID FEVER*

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ABSTRACT:

Barium meal examinations have been performed on 20 patients with proved or clinically suspected typhoid fever. There were distinct abnormalities in the terminal ileum early in the febrile stage of the disease. The abnormality was different in a single case of relapse of typhoid. The study is easily and safely performed and we feel that it is a valuable adjunct in the diagnosis of typhoid fever, particularly in those cases where a positive blood culture is not available to confirm the clinical impression. The result of the roentgenographic examination is obtainable immediately and treatment can be initiated early.

TYPHOID fever is a bacterial disease caused by *Salmonella typhi* and *paratyphi* A, B, C. It is a multisystem disease with a wide spectrum of clinical presentations. The portal of entry and the most severely involved system is the gastrointestinal tract.[8] Bacilli multiply in the solitary lymph follicles and the aggregate lymph follicles known as Peyer's patches which are concentrated in the terminal ileum. The lymph follicles become hyperplastic and swollen and form button-like protrusions into the lumen. The Peyer's patches are enlarged, raised, oval, sharply delineated, and have their longitudinal axis parallel to the long axis of the bowel. They may be up to 6 cm. long and 0.4 cm. above the adjacent mucosa. Their luminal surfaces are convoluted. There is also edema of the mucosa and submucosa. The mesenteric lymph nodes are enlarged. At a later stage, toward the end of the second week after the onset of fever, necrosis occurs in these lymph follicles and Peyer's patches. This is followed by the development of oval flat ulcerations, rarely extending into the muscularis. Healing is usually complete without scarring.[1,4,10,14]

Despite modern effective treatment of typhoid fever, the complications of the disease continue to be potentially dangerous. Therefore, every effort should be made to establish an early diagnosis. Although the course of the illness is typical enough in the majority of cases to reach a presumptive clinical diagnosis, variations from the classical pattern are not uncommon.[2,5] Furthermore, the clinical picture may change with inadequate treatment or when the patient has been immunized. The definitive diagnosis of typhoid fever depends on the isolation of the causative organism, usually from the blood. This is successful during the first week of fever in 50–90 per cent of untreated cases and decreases significantly thereafter.[2,9,15] Blood cultures require an incubation period of at least 48 hours. The chances of isolating the organism decrease further in cases treated with effective antibiotics. Although the Widal test at times reveals a high titer early in the course of the disease, it is neither very reliable nor specific because the results can be altered by treatment, immunization and other *Salmonella* infections. Besides, it is the rise in titer and not any one absolute value which is helpful. A low titer does not exclude typhoid fever.[9,12,13,15]

Because of the importance of an early diagnosis, a barium meal examination of the small intestine was carried out in suspected cases. Our paper deals with the results of these examinations in 20 cases of proved or suspected typhoid fever treated

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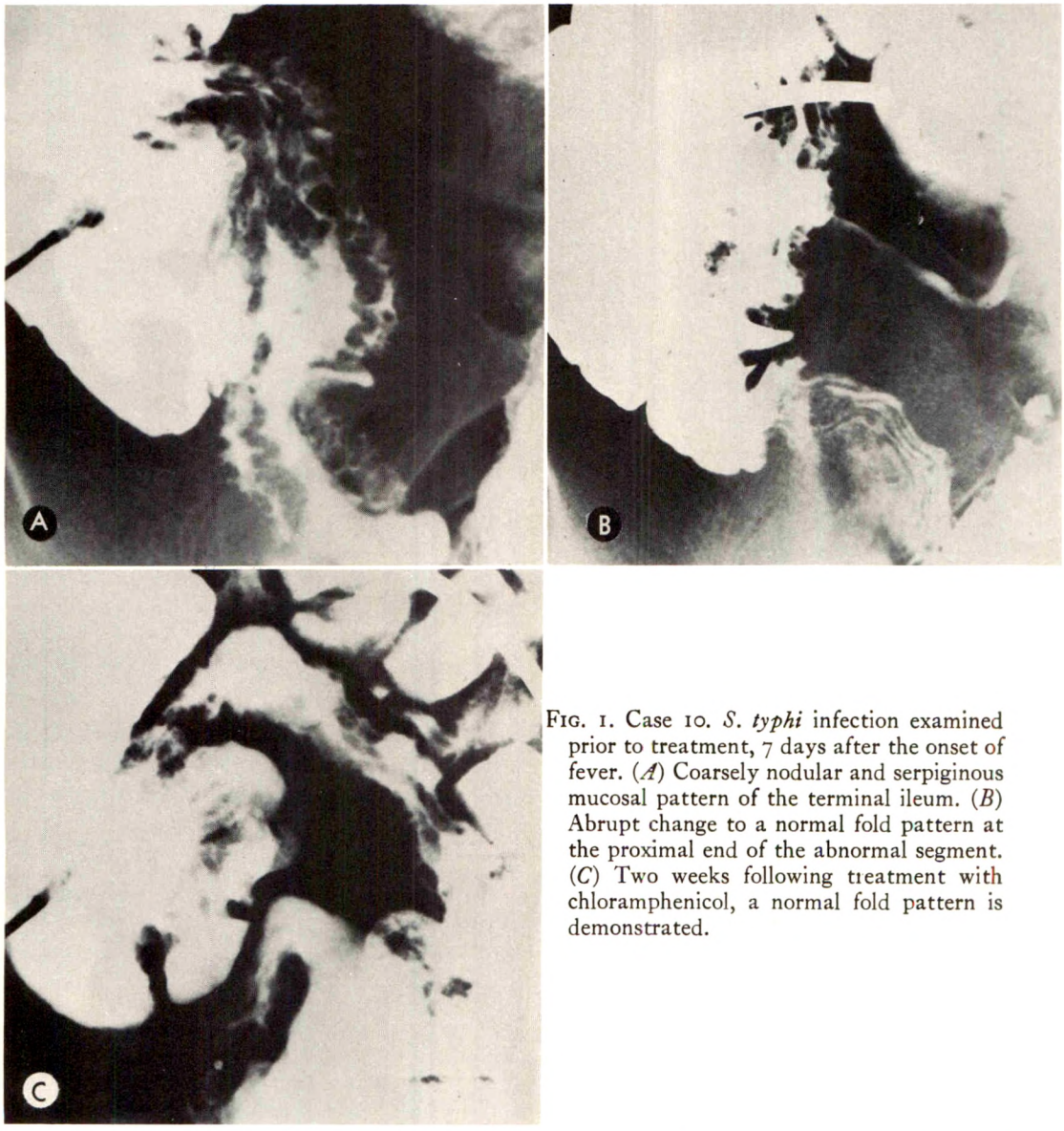

Fig. 1. Case 10. *S. typhi* infection examined prior to treatment, 7 days after the onset of fever. (*A*) Coarsely nodular and serpiginous mucosal pattern of the terminal ileum. (*B*) Abrupt change to a normal fold pattern at the proximal end of the abnormal segment. (*C*) Two weeks following treatment with chloramphenicol, a normal fold pattern is demonstrated.

at the American University of Beirut Hospital.

METHOD

Following ingestion of 550 cc. of a non-flocculable barium (Mixobar, Astra), 20 mg. of metoclopramide were administered intravenously to decrease the transit time through the intestine and to alleviate the severe nausea that some of the toxic patients experienced.[6] The patient was then placed on the right side to promote gastric emptying. Multiple exposures of the abdomen in the prone position were made at intervals.

ROENTGENOGRAPHIC FINDINGS

Barium reached the colon in 1 to 2½ hours. All of the small intestine was visualized using a pneumatic compression paddle to separate the crowded loops when necessary.

About half of the cases demonstrated very slight uniform dilatation of the ileum to 1½ times the normal caliber. There was no jejunal dilatation. Contrary to other re-

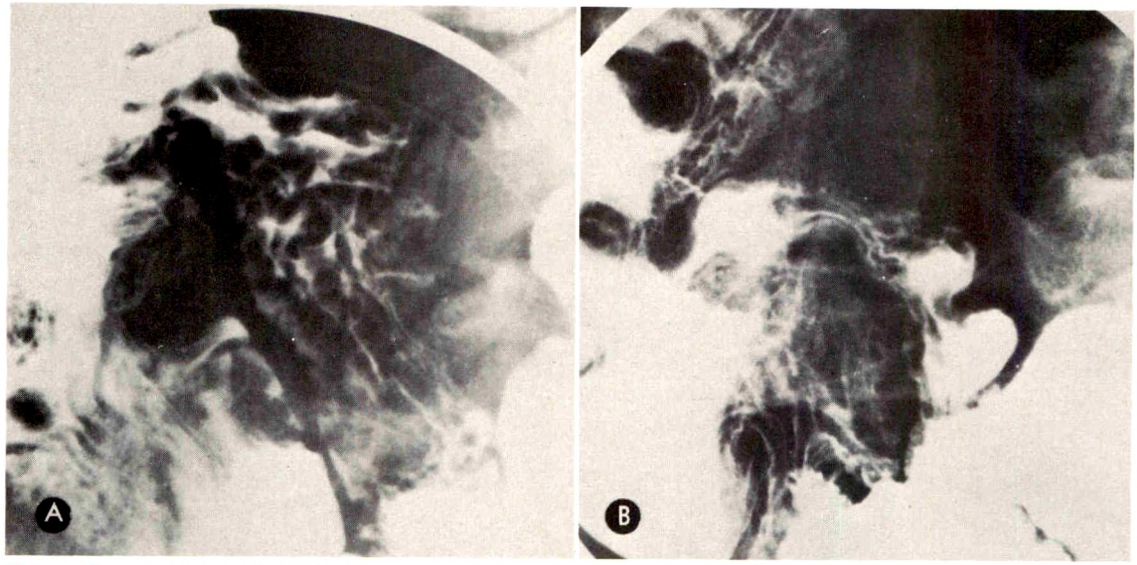

FIG. 2. Case 4. *S. typhi* infection examined prior to treatment, 9 days after the onset of fever. (*A*) Coarsely nodular and serpiginous pattern of the terminal ileum. (*B*) Four months following treatment with chloramphenicol a normal fold pattern is demonstrated.

ports, no localized dilated segment, excessive secretion, prolonged spasm, or hyperperistalsis was demonstrated.[3] Except for 1 case, which will be described, the small intestinal abnormality was confined to the terminal 10 to 15 cm. of the ileum. There was an abrupt change to a normal fold pattern proximally (Fig. 1*B*). The abnormal segment exhibited a coarsely nodular and serpiginous mucosal pattern, assuming mostly a longitudinal arrangement. This represented a variable combination of

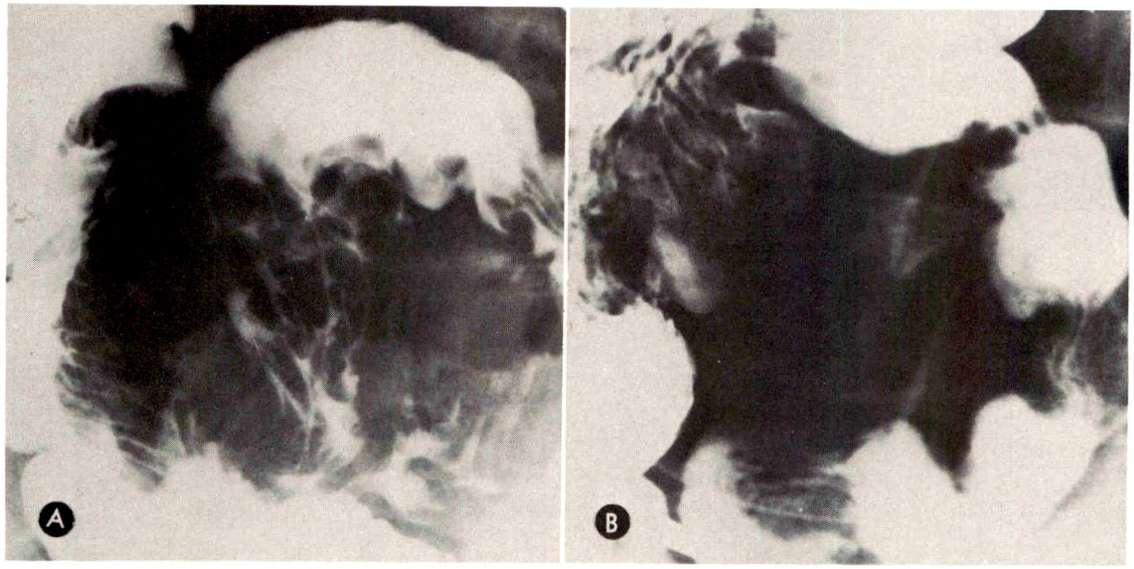

FIG. 3. Case 9. Negative blood cultures. Patient examined 6 days after the onset of fever. (*A*) Longitudinally arranged, ovoid, coalescing filling defects due to raised Peyer's patches and lymph follicles in the terminal ileum. Abrupt change to a normal fold pattern proximally. The cecum is edematous. (*B*) Four months following treatment with chloramphenicol a normal fold pattern is demonstrated.

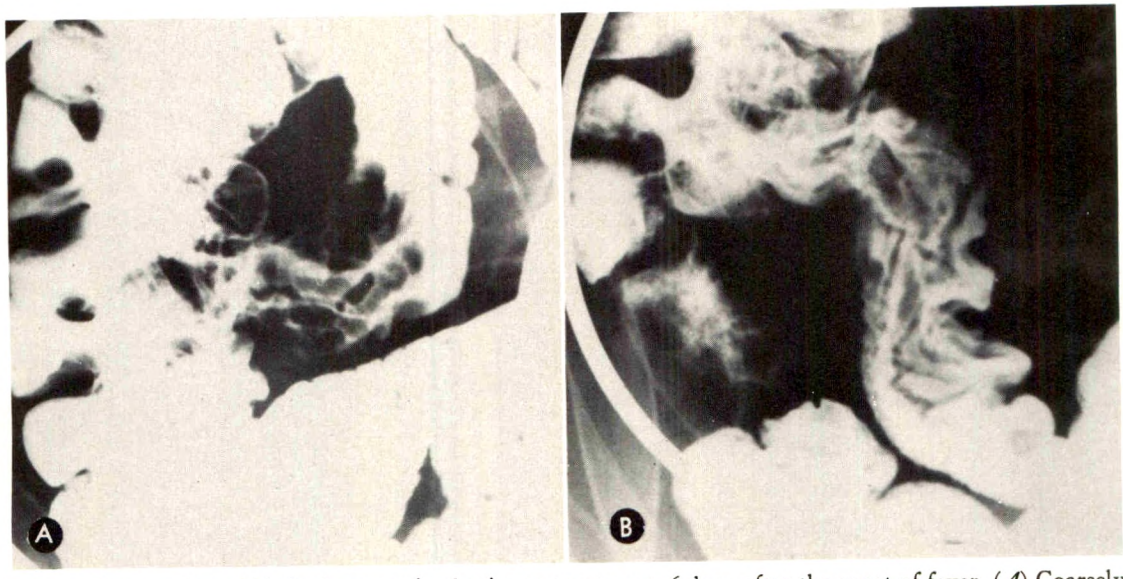

Fig. 4. Case 20. *S. typhi* infection examined prior to treatment, 6 days after the onset of fever. (*A*) Coarsely nodular pattern of the terminal ileum. (*B*) Two weeks following treatment with chloramphenicol a normal fold pattern is demonstrated.

the swollen lymph follicles, the raised Peyer's patches, and the edematous mucosa (Fig. 1*A*–10*A*). Occasionally, a solitary Peyer's patch was seen rising discretely above the adjacent mucosa (Fig. 5*A*; 8*A*; 9*A*; and 10*A*). There was no luminal narrowing or mural rigidity. The edema occasionally involved the cecum on either side of the ileocecal valve (Fig. 3*A*; 6*A*; and 8*A*). In most cases there was no separation of the ileal loops. However, the terminal ileum was easier to demonstrate

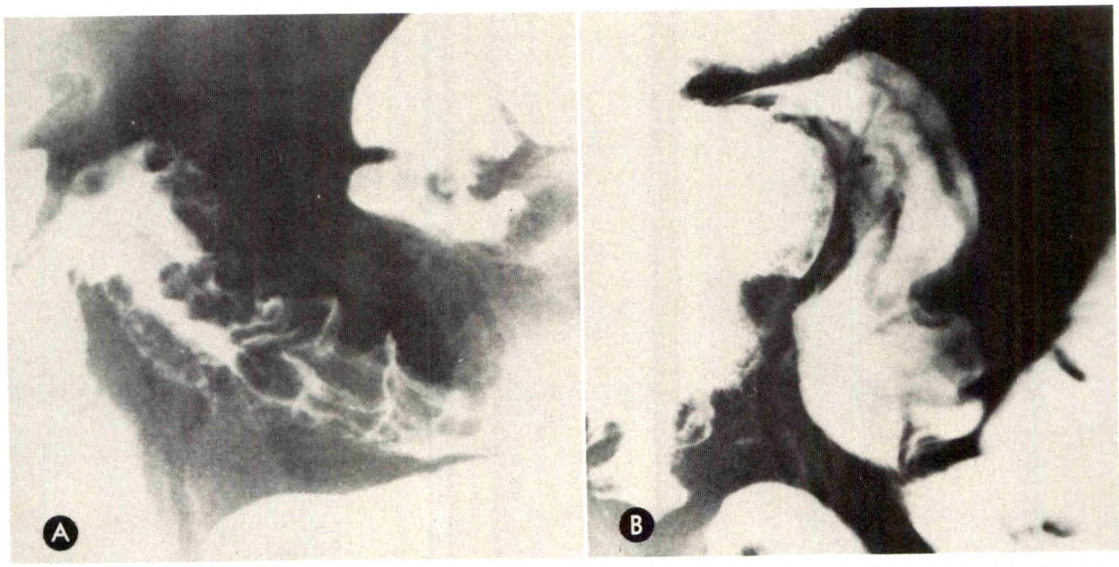

Fig. 5. Case 12. *S. typhi* infection examined 2 days after the start of adequate treatment with chloramphenicol, and 6 days after the onset of fever. (*A*) Longitudinally arranged, ovoid, coalescing filling defects due to raised Peyer's patches and lymph follicles in the terminal ileum. Abrupt change to a normal fold pattern proximally. (*B*) One week following treatment with chloramphenicol, a normal fold pattern is demonstrated.

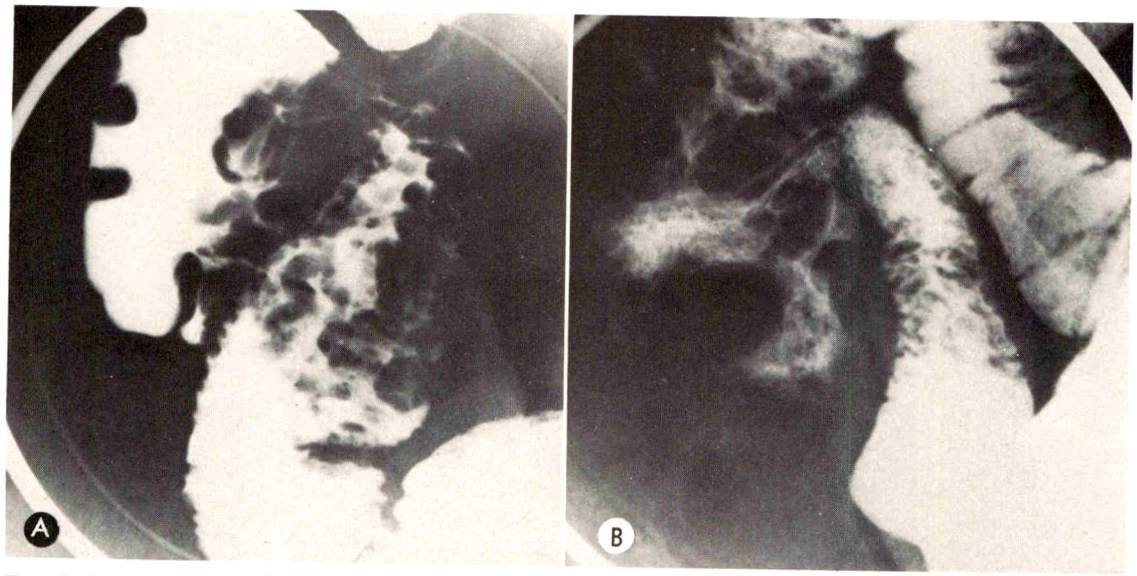

FIG. 6. Case 15. *S. para.* B infection examined prior to treatment, 15 days after onset of fever. (*A*) Coarsely nodular and serpiginous pattern of the terminal ileum and edema of the cecum adjacent to the ileocecal valve. (*B*) Two weeks following treatment with chloramphenicol a normal fold pattern is demonstrated.

by the pneumatic compression paddle before treatment as compared with the same cases re-examined after treatment. This is most likely due to thickening of the intestinal wall and enlargement of the mesenteric lymph nodes, resulting in slight separation of the loops. Only in a small number of cases, slight separation of the terminal ileum from the adjacent loops was obvious without the use of a compression paddle. In the cases where the colon was opacified, no abnormality was demonstrated except in the cecum adjacent to the ileocecal valve. This was a gross observa-

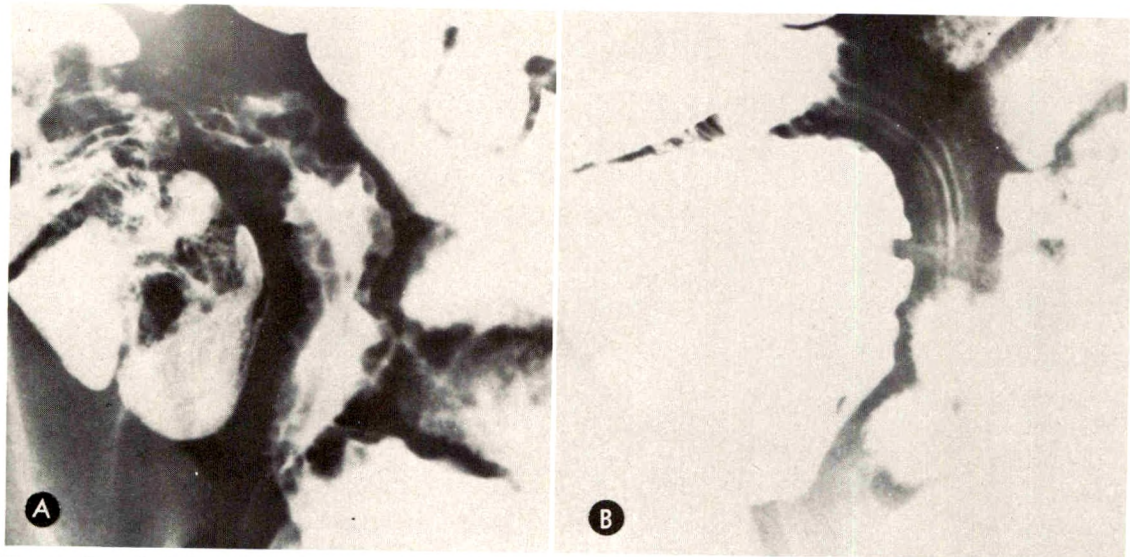

FIG. 7. Case 6. *S. typhi* infection examined 2 days after the start of adequate treatment with chloramphenicol and 5 days after the onset of fever. (*A*) Serrated margins and edematous folds of the terminal ileum. (*B*) Four months following treatment with chloramphenicol a normal fold pattern in demonstrated.

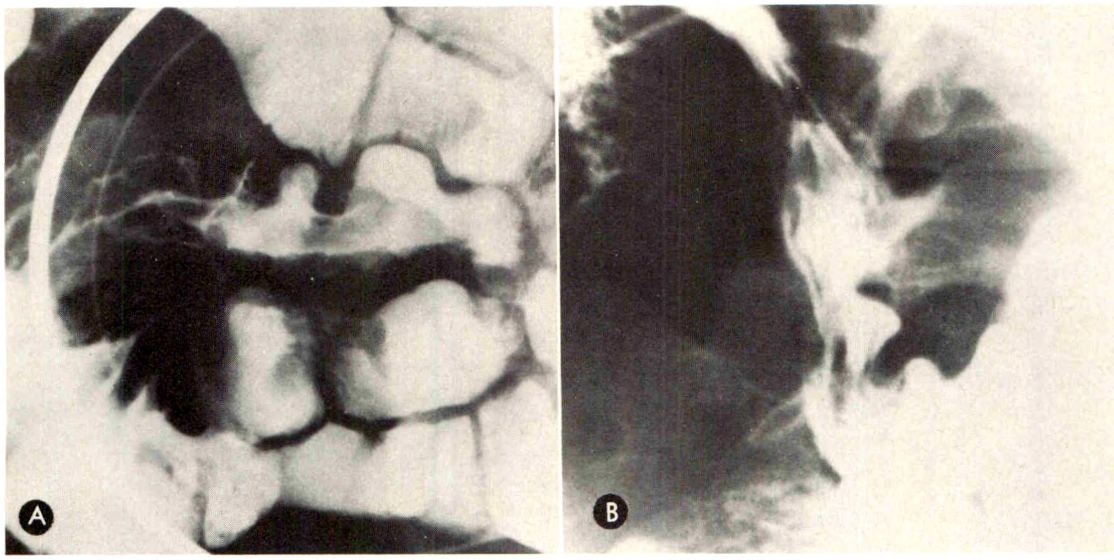

Fig. 8. Case 17. Negative blood cultures. Examined 11 days after the onset of fever. (*A*) Longitudinally arranged, ovoid, coalescing filling defects due to raised Peyer's patches in the terminal ileum. The cecum is edematous on either side of the ileocecal valve. (*B*) Four months following treatment with chloramphenicol a normal fold pattern is demonstrated.

tion since the colon was full of residue. The only patient who was studied during the relapse of a *Salmonella paratyphi* infection exhibited a different roentgenographic finding, with involvement of the terminal 50 cm. of the ileum (Fig. 11*A*). This represented a significantly longer segment when compared with the other cases. There was marked diffuse edema suggesting a stacked plate appearance, and there was mural rigidity.[7,11] The loops were moderately separated due to thickening of the wall and mesenteric lymph node enlargement. There was no significant alteration in the transit

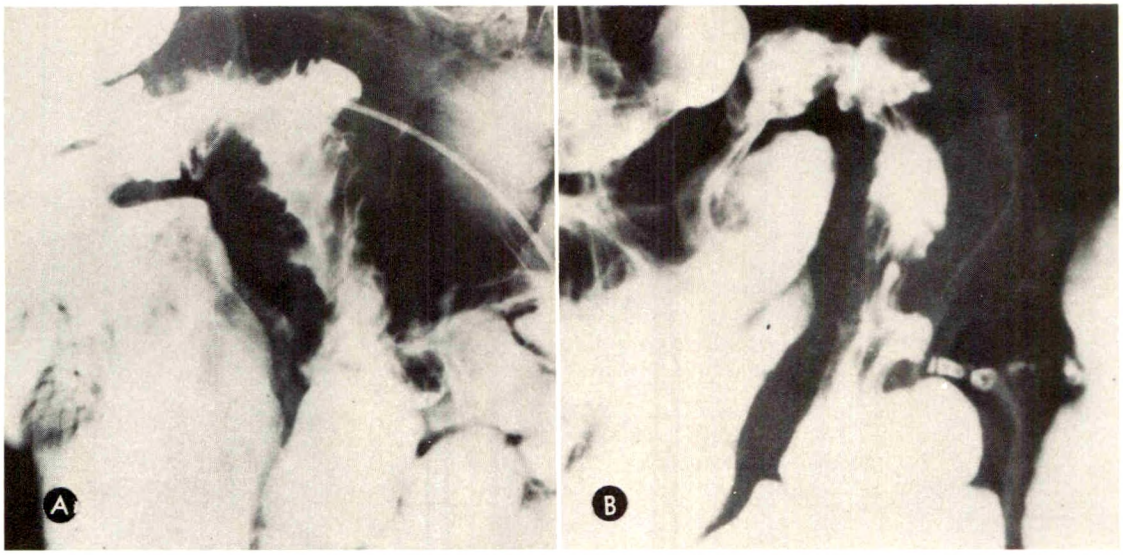

Fig. 9. Case 11. *S. para.* B infection examined prior to treatment, 6 days after the onset of fever. (*A*) Raised Peyer's patches with convoluted surfaces are seen tangentially in the terminal ileum. (*B*) Eight days following treatment with chloramphenicol, a normal fold pattern is demonstrated.

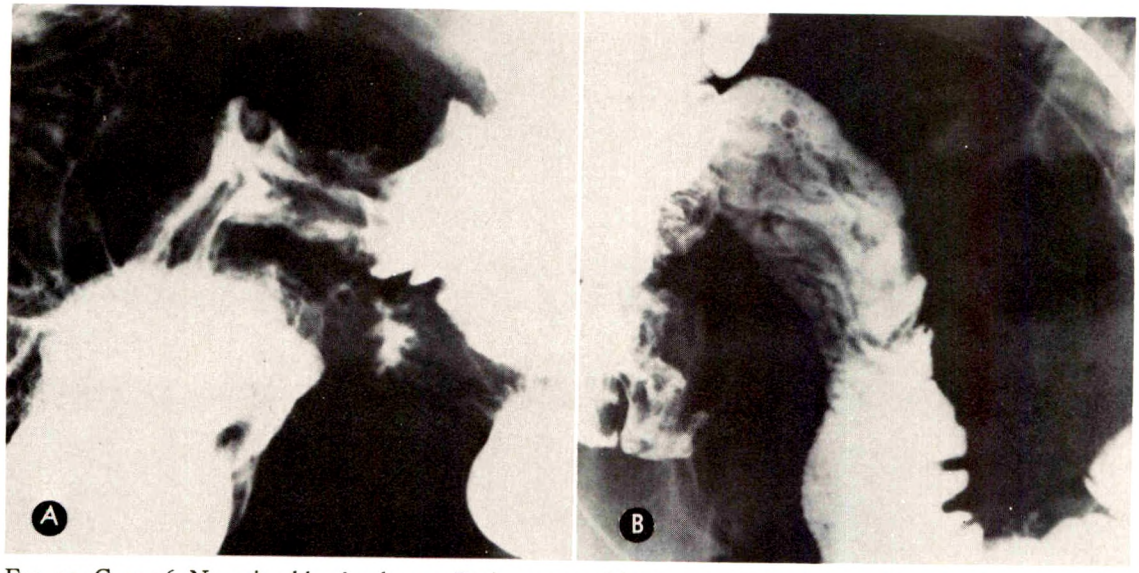

FIG. 10. Case 16. Negative blood cultures. Patient treated inadequately for 2 days. Examined 12 days after onset of fever. (*A*) Edematous terminal ileum. A large, raised Peyer's patch is seen tangentially at the upper surface of the terminal ileum. (*B*) Five days following treatment with chloramphenicol a normal fold pattern is demonstrated.

time of barium. In spite of an adequate clinical response, the roentgenographic appearance did not change after 1 week of treatment. It was only at the end of 3 weeks that marked improvement was noted (Fig. 11*B*).

RESULTS

The clinical, laboratory, and roentgenographic data are presented in Table 1.

Initially only bacteriologically proved cases were referred for roentgenographic study. Later in the study, cases with a

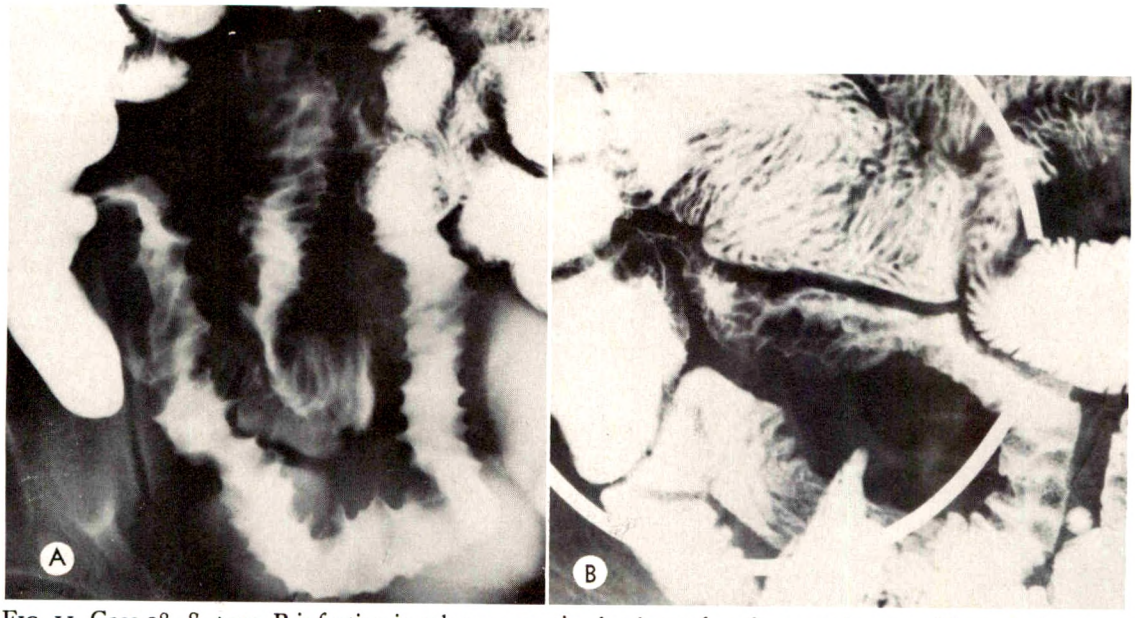

FIG. 11. Case 18. *S. para.* B infection in relapse, examined 7 days after the second onset of fever. (*A*) A long segment of the terminal ileum exhibits a stacked plate configuration, due to extensive edema. The involved segments are moderately separated due to the edema of the wall and mesenteric lymph node enlargement. (*B*) Three weeks following treatment there is marked regression of the edema.

TABLE I

CLINICAL DATA ON PATIENTS WITH TYPHOID FEVER

Case No.	Blood Culture	Widal at Time of Blood Culture	Duration of Treatment Before Roentgenographic Study (days)	Duration of Fever Before Roentgenographic Study (days)	Initial Roentgenographic Findings	Interval Between Initial and Follow-up Study	Roentgenographic Findings After Treatment
1	S. para. B	High	3	9	−		
2	S. typhi	Not done	4	7	−		
3	S. typhi	Not done	5	12	−		
4	S. typhi	High	0	9	+	4 months	−
5	S. para. B	Low	5	8	+	2 weeks	−
6	S. typhi	Low	2	5	+	4 months	−
7	S. typhi	High	0	5	+	3 weeks	−
8	S. typhi	High	5	10	+	4 weeks	−
9	Negative	Low	0	6	+	4 months	−
10	S. typhi	Low	0	7	+	2 weeks	−
11	S. para. B	Not done	0	6	+	8 days	−
12	S. typhi	Not done	2	6	+	7 days	−
13	S. typhi	Low	1	6	+	5 weeks	−
14	S. typhi	Low	1	5	+	2 weeks	−
15	S. para. B	Low	0	15	+	2 weeks	−
16	Negative	High	2 (Inadequate)	12	+	5 days	−
17	Negative	Low	0	11	+	4 months	−
18	S. para. B	Not done	0	7	+	3 weeks	Marked improvement
19	Negative	High	7 (Inadequate)	8	+	4 weeks	−
20	S. typhi	High	0	6	+	2 weeks	−

clinical suspicion of typhoid fever were included before the blood culture result confirmed the diagnosis. Of the 20 adult patients studied, 16 had a positive blood culture. In 13 of the latter patients roentgenographic changes were demonstrated, while in the remaining 3 of the 16 the study was negative. All 3 had already been under treatment by chloramphenicol, 3 gm. daily for 3, 4, and 5 days, respectively (Cases 1, 2 and 3). In the remaining 4 cases, although a positive culture was not obtained, roentgenographically all 4 were positive (Cases 9, 16, 17 and 19). Two of these had received inadequate treatment (Cases 16 and 19). Thus, of the total of 20 patients, 17 had positive roentgenographic findings. This number included all the patients who had received no treatment before the examination. There was no untreated patient with a bacteriologically proved diagnosis in whom the roentgenographic study was negative. In all patients with positive findings, there was clearing of the roentgenographic abnormality following adequate treatment with chloramphenicol. We could not detect any difference in the abnormality in patients suffering from *Salmonella typhi* or *paratyphi* infection. In 7 additional patients with negative culture and Widal test, roentgenographic examination was performed because of a clinical suspicion of typhoid fever. The examination was negative in all 7. Eventually, the clinical course of these patients excluded typhoid fever. Their fever subsided spontaneously without chloramphenicol treatment.

DISCUSSION

Our study has demonstrated definite roentgenographic changes in the intestine associated with typhoid fever. These changes are present early in the course of the disease. During a relapse of typhoid, a significantly longer segment of terminal ileum with a different pattern is demon-

strated as compared with the findings during a primary infection. Our earliest examination was performed on the fifth day of fever in 3 cases; all had positive findings (Cases 6, 7 and 14). In our experience, a short period of adequate treatment of up to 5 days may, but does not necessarily, revert the roentgenographic findings to normal (Cases 1, 2, 3 and 16). The examination is of particular value in those patients who have received inadequate treatment, thereby greatly reducing the chances of obtaining a positive blood culture (Cases 16 and 19). The roentgenographic study may be the only way of confirming the diagnosis when the clinical picture is suggestive but the blood culture is negative (Cases 9 and 17). We feel that the findings are typical enough to justify the initiation of treatment in such cases. In our experience, it is fairly safe to say that a negative roentgenographic study early in the course of a febrile illness rules out typhoid fever.

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REFERENCES

1. ANDERSON, W. A. D. Pathology. C. V. Mosby Company, St. Louis, 1971.
2. CHALMERS, I. M. Typhoid fever in endemic area: great imitator. *South African M. J.*, 1971, *45*, 470–472.
3. CHERIGIE, E., LAPORTE, A., and VERSPYCK. R. Aspect radiologique du grele chez les typhiques. *J. de radiol., d'électrol. et de méd. nucléaire*, 1953, *34*, 522–524.
4. GAINES, S., SPRINZ, H., TULLY, J. G., and TIGERTT, W. D. Studies on infection and immunity in experimental typhoid fever. *J. Infect. Dis.*, 1968, *118*, 293–305.
5. HATHOUT, S. E. D., and WAHAB, F. A. Unusual presentation of enteric infections. *J. Egyptian M. A.*, 1970, *45*, 56–71.
6. HOWARTH, F. H., COCKEL, R., ROPER, B. W., and HAWKINS, C. F. Effect of metoclopramide upon gastric motility and its value in barium progress meals. *Clin. Radiol.*, 1969, *20*, 294–300.
7. MARGULIS, A. R., and BURHENNE, H. J. Alimentary Tract Roentgenology. C. V. Mosby Company, St. Louis, 1973.
8. OSLER, W. Principles and Practice of Medicine. Appleton-Century-Crofts, New York, 1912.
9. REYNOLDS, D. W., CARPENTER, R. L., and SIMON, W. H. Diagnostic specificity of Widal's reaction for typhoid fever. *J.A.M.A.*, 1970, *214*, 2192–2193.
10. ROBBINS, S. L. Pathologic Basis of Disease. W. B. Saunders Company, Philadelphia, 1974.
11. SCHINZ, H. R., BAENSCH, W. E., FRIEDL, E., and UEHLINGER, E. Roentgen Diagnostics. Volume 4. Grune & Stratton, Inc., New York, 1954.
12. SCHROEDER, S. A. Interpretation of serologic tests for typhoid fever. *J.A.M.A.*, 1968, *206*, 839–840.
13. SEN, R., and SAXENA, S. N. Critical assessment of conventional Widal test in diagnosis of typhoid and paratyphoid fevers. *Indian J. M. Res.*, 1969, *57*, 1813–1819.
14. SMITH, J. H. Pathology of salmonelloses: review. *J. Egyptian M. A.*, 1970, *45*, 28–38.
15. WAHAB, M. F. A. Widal and blood picture in diagnosis of typhoid fever. *J. Egyptian M. A.*, 1970, *45*, 85–107.

CURRENT PERSPECTIVES ON GIARDIASIS*

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BALTIMORE, MARYLAND

ABSTRACT:

Giardia lamblia infestation can cause severe diarrhea and malabsorption, and the diagnosis is usually made by identification of cysts in the feces, but small intestinal biopsy or smears may be required. A wide spectrum of roentgen changes may be seen. In patients with a normal immune status, the small bowel is normal or shows an inflammatory bowel disease pattern. Eradication of the parasite reverses these changes. In some patients with IgA deficiency, nodular lymphoid hyperplasia occurs, and this is usually not reversible. Other patients with hypogammaglobulinemia or dysgammaglobulinemia and giardiasis may show a sprue pattern. This pattern most often persists after eradication of the parasite. Although the triad of giardiasis, IgA deficiency, and nodular lymphoid hyperplasia has a particularly high association, these, together with diarrhea, malabsorption, and various altered immune states may occur in any combination.

GIARDIA LAMBLIA, a multiflagellate protozoan, was once thought to be a harmless commensal organism in the gastrointestinal tract of man. It is now well established that this organism may be associated with severe diarrheal disease and malabsorption.[28,46] In recent years, *Giardia lamblia* infestation has been reported in a bewildering array of "altered immune states" in which its relationship to diarrheal disease has often been uncertain. It is the purpose of this report to review the current knowledge of giardiasis, to present illustrative cases, and to propose a scheme for understanding the inter-relationships of giardiasis, diarrheal disease, nodular lymphoid hyperplasia, and "altered immune states."

EPIDEMIOLOGY

Giardia lamblia was first identified in human feces by van Leeuenhoek and characterized by Lambl.[33] More recently, transmission and scanning electron microscopy have been used to demonstrate the anatomic and ultrastructural features.[30] The organism tends to inhabit the duodenum and jejunum in its human host and attaches to the mucosa by a ventral suction disk.[29] In the intestine *Giardia lamblia* exists as a motile trophozoite, but the cyst form is most commonly identified in the feces. Trophozoites die within hours of leaving the body, but the cysts can survive for many days or weeks outside the host.[28] The mode of transmission is poorly documented, but is probably most often by ingestion of food or water contaminated by human feces containing cysts. In an epidemic which occurred in Aspen, Colorado, sewage contamination of the water supply was the suspected cause.[28] In experimental studies in man,[35] ingestion of less than 100 organisms often produced infestation, and ingestion of greater numbers uniformly produced infection. The infestation is often self-limited, but a chronic infestation lasting many months is sometimes produced.

PATHOLOGY

In symptomatic cases, biopsy of the small intestine usually shows varying combinations and degrees of blunting of villi, acute and chronic inflammation of the

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.

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lamina propria, increased crypt epithelial mitoses and luminal bacterial overgrowth.[29,41,46,47] Ament and Rubin[2] studied patients with multiple biopsies at many levels of the small bowel. They emphasized the patchy distribution of the lesions in the mucosa which may be almost normal in 1 specimen, yet show severe changes at adjacent sites. This lack of uniformity in distribution may account for the poor correlation observed between single biopsies and the clinical and roentgenologic status. The organism is usually identified only in the mucus in the intervillous spaces,[30,41] although some investigators have demonstrated tissue invasion by differential staining and careful examination of multiple sections.[6,29] Histologic abnormalities usually disappear after treatment in normal individuals, and variable improvement occurs in patients who also have immunoglobulin deficiencies.[2]

Absence or marked reduction in the number of plasma cells in the lamina propria indicates a hypogammaglobulinemic state[2,13,14,15,18,19] and is sometimes associated with a marked increase in the number and size of lymphoid follicles and lymphocytic infiltration of the lamina propria—nodular lymphoid hyperplasia.[2,19,20] In selective IgA (immunoglobulin A) deficiency, plasma cells may be present in near normal numbers in the lamina propria, but there is a marked decrease in the proportion of cells which show immunofluorescent staining specific for IgA.[12]

DIAGNOSIS AND TREATMENT

The diagnosis of *Giardia lamblia* infestation is most often accomplished by identification of the characteristic cysts in the stool. It is important to recognize, however, that cysts may be absent from the stool even in patients with severe disease.[2,23,31,46] In these patients the organism may be found only in smears of the jejunal mucus or by careful examination of the intervillous spaces on stained sections of a small intestinal biopsy. The clinical significance of the infestation can be judged only after eradication of the organism.[17] This is usually accomplished easily by a course of metranidazole (Flagyl) or quinacrine (Atabrine), although relapses are not uncommon and require additional medication.[2,28,46] Disappearance of cysts from the stool after therapy may occur in some patients who have persistent symptoms, with organisms remaining in duodenal aspirates.[46]

CLINICAL AND ROENTGENOGRAPHIC FEATURES

To understand the clinical and roentgenographic spectrum of giardiasis, it is necessary to know the relationships between giardiasis, diarrheal disease, nodular lymphoid hyperplasia and "altered immune states." Figure 1, *A* and *B* shows a simple diagram which demonstrates these relationships and which will serve as a basis for the discussion below.

ASYMPTOMATIC GIARDIA INFESTATION

(Fig. 1*A*-a)

Of various populations studied in the United States, 1.5 to 20 per cent have been found to be infected with *Giardia lamblia*.[28] The vast majority of these individuals are asymptomatic[6] and have normal small bowel roentgenograms.[26]

SYMPTOMATIC GIARDIASIS

(Fig. 1*A*-b)

Children,[21,46] postgastrectomy patients,[43] and travelers to endemic areas[21] (*e.g.*, Leningrad,[39] India,[3] Aspen, Colorado[28]) are believed to be prone to develop clinically significant infestations. The acute form may range from a mild gastrointestinal upset to a severe, protracted illness with profuse diarrhea, intestinal cramping, bloating, malabsorption, and weight loss.[28] A chronic malabsorption syndrome frequently occurs.[46] In children, chronic giardiasis has been confused with celiac disease because they may have failure to thrive and show flattened villi on mucosal biopsy, but the response to a gluten free diet is poor.[6,11]

The roentgenographic findings consist

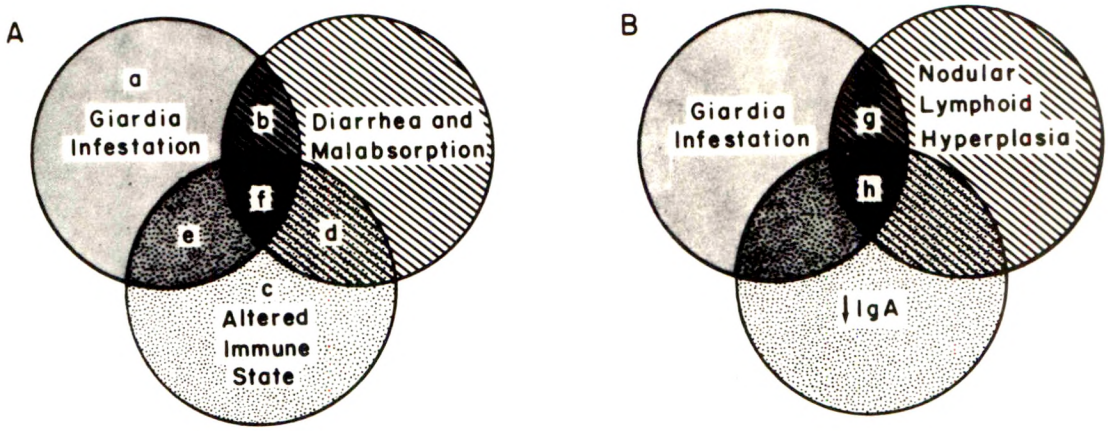

FIG. 1. (*A* and *B*) This mnemonic device summarizes the associations reported in the literature among *Giardia lamblia* infestation, diarrhea and malabsorption, nodular lymphoid hyperplasia, and a variety of "altered immune states," several of which have in common a decreased level of serum immunoglobulin A (↓ IgA). The device is useful to emphasize that each of the entities may occur independently or in variable combination with the others. A detailed description of the relationships is given in the text.

of thickening of the valvulae conniventes, increased secretions, and disordered motility (Fig. 2–6) which are a reflection of inflammatory changes in the intestinal mucosa.[26,33,45] Roentgenologic abnormalities are most apparent in the duodenum and jejunum. The single most important finding is thickening of the mucosal folds (Fig. 2, *A–C*) which is attributable to diffuse mucosal inflammation including proliferative changes in the mucosal glands, edema of the lamina propria and widespread infiltration by inflammatory cells. In severe cases the folds are irregularly tortuous and present a distinctly nodular appearance when seen on end (Fig. 3, *A* and *B*). Increased secretions result in barium dilution and flocculation. There is a finely granular appearance of the barium, which is often less pronounced when the newer colloidal barium suspensions are used. Disordered motility results in spasm and segmentation of the barium column and rapid transit of barium to the cecum (Fig. 2*B*). In 1 of our patients the combination of increased motility and diffuse thickening of folds resulted in episodes of transient jejunal intussusception (Fig. 6, *A–D*). The roentgenologic findings are not specific; similar changes may be seen in other parasitic and inflammatory diseases of the bowel.[25,26,32,36]

ALTERED IMMUNE STATES

There have been no prospective, controlled studies to prove an increased susceptibility to giardiasis in patients with any type of altered immunity. Nevertheless, many investigators suspect an increased incidence of the disease in these patients, particularly when there is a deficiency of IgA.[5,46] IgA is synthesized and released by plasma cells in the lamina propria of the intestine. Some of the IgA passes into the blood, where its concentration is most conveniently measured (serum IgA). Much IgA is taken up by the epithelial cells of the intestine. The epithelial cells produce a nonimmunoglobulin glycoprotein "secretory component" which is bound to IgA and the resultant molecule (secretory IgA) is secreted into the intestinal lumen. Secretory IgA is the principal immunoglobulin in saliva, tears, enteric fluid,[13,34] and other external secretions in which it is generally assumed to play an important role in the body's defense.[42] This role is best documented in defense against

FIG. 2. *Giardiasis: normal immune status.* A 36 year old white female had a sudden onset of severe diarrhea. Stool examination showed cysts of *Giardia lamblia.* Serum immunoglobulins were normal. Response to Flagyl was excellent. (*A*) The valvulae conniventes of the duodenum are thickened and tortuous. (*B*) Twenty minute roentgenogram. The mucosal folds in the proximal jejunum are also thickened and tortuous. Rapid transit time is noted. (*C*) Sixty minute roentgenogram. There are moderate segmentation and minimal evidence of increased secretions.

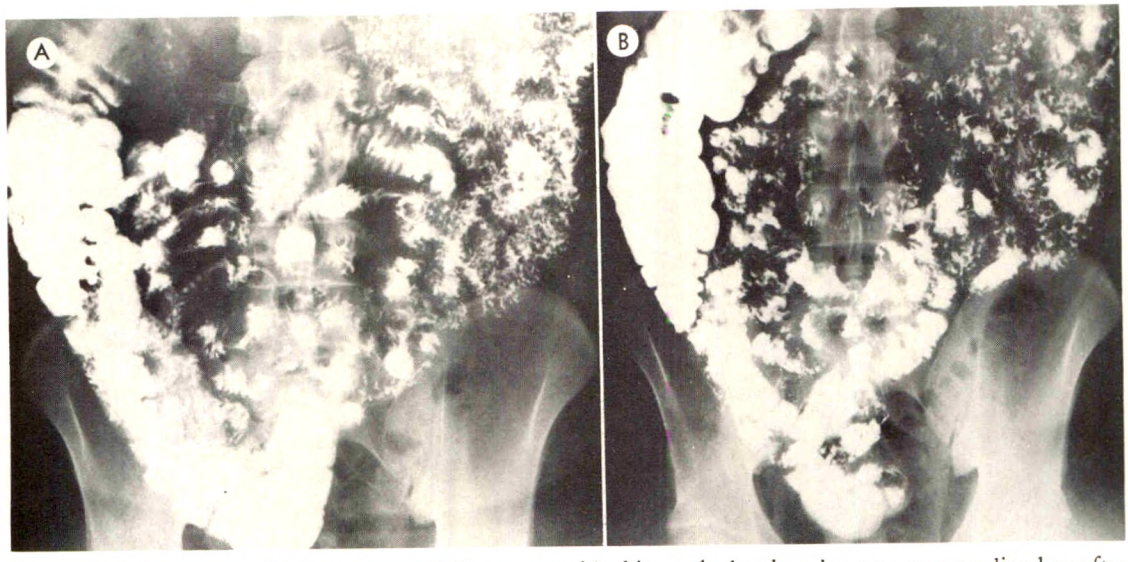

Fig. 3. *Giardiasis: normal immune status.* This 21 year old white male developed severe crampy diarrhea after a trip to India. *Giardia lamblia* cysts were identified in the stool. Serum carotene and D-xylose excretion was low and returned to normal after successful therapy with Flagyl. (*A*) Forty minute roentgenogram. Multiple areas of spasm are evident. The mucosal folds of the jejunum are thickened and have a nodular appearance when seen on end. (*B*) Sixty minute roentgenogram. Increased secretions are present with barium dilution and flocculation. The thickened nodular mucosal folds are again seen.

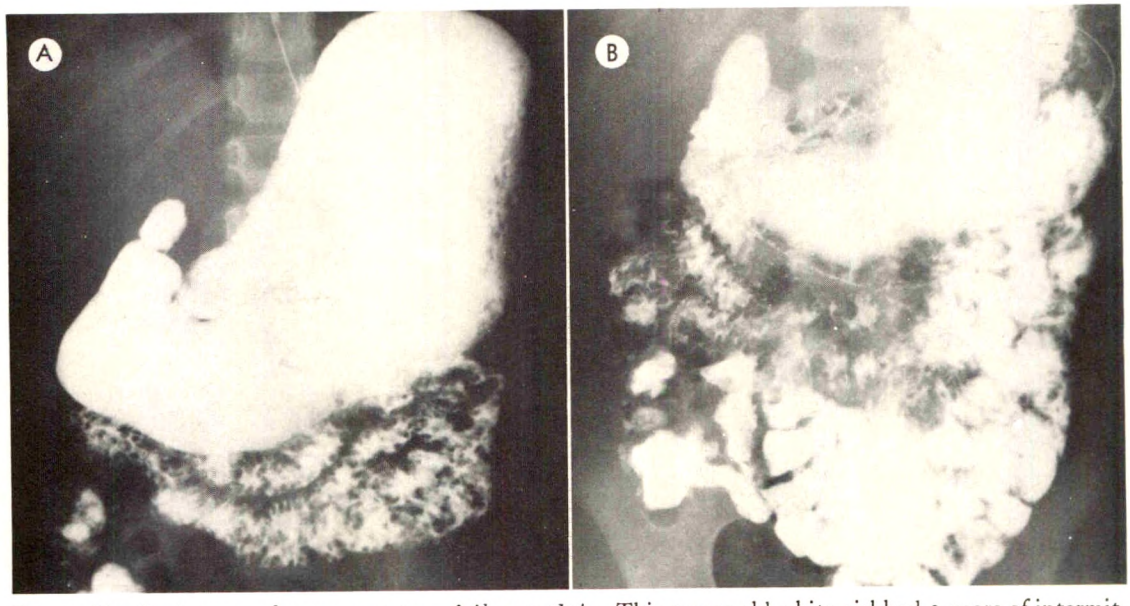

Fig. 4. *Giardiasis: normal immune status—failure to thrive.* This 5 year old white girl had 2 years of intermittently diarrheal stools containing up to 12 gm. of fat per day. Her weight was below the third percentile and her bone age was retarded. She was initially thought to have celiac disease but failed to respond to a gluten free diet. *Giardia lamblia* cysts were finally identified in the stools and her response to Flagyl was dramatic with complete disappearance of diarrhea and restoration of normal growth and development. (*A*) The mucosal folds are thickened and have a nodular appearance when seen on end. (*B*) Twenty minute roentgenogram. Hypersecretion with barium dilution, segmentation and flocculation are present.

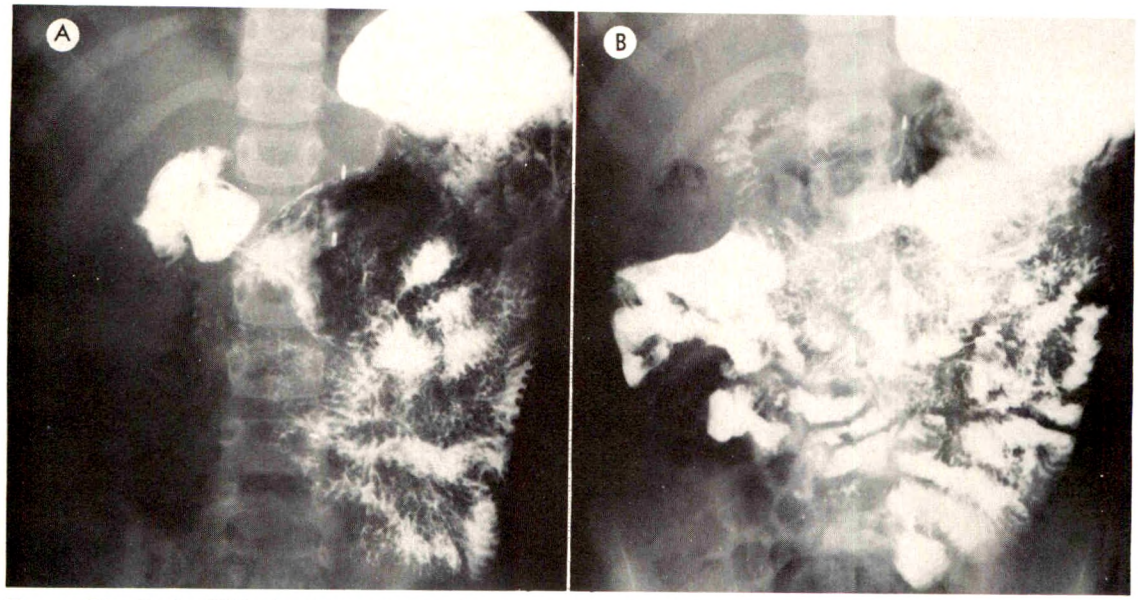

FIG. 5. *Giardiasis: Wilms' tumor patient on immunosuppressive therapy.* This 6 year old white female developed severe diarrhea while on therapy with Vincristine and Actinomycin D. *Giardia lamblia* cysts were present in the stool and immunoglobulin levels were normal. Response to eradication of the parasite was excellent. (*A*) The valvulae conniventes are thickened. (*B*) Hypersecretion is evident with dilution of barium, flocculation, and slight segmentation.

viral agents, but IgA very likely also plays some part in defense against bacteria, protozoan parasites, food allergens,[7] and other offending agents in the intestine.[16,24] A number of recent, excellent reviews provide more detailed information on the immunology of the intestine.[16,18,24,37,40,42]

Although giardiasis has been most often reported in association with primary immunoglobulin deficiencies, it may also occur in any other altered immune state. One of our patients with a Wilms' tumor developed severe giardiasis while on therapy with Vincristine and Actinomycin D (Fig. 5, *A* and *B*). Another patient with cartilage-hair hypoplasia (metaphyseal chondrodysplasia, Type McKusick), normal immunoglobulins, and evidence of defective cellular immunity, had severe lifelong malabsorption with giardiasis as a contributing factor. Still another patient with Addison's disease, hypoparathyroidism, and chronic mucocutaneous candidiasis had malabsorption with giardiasis.[10] We have also observed giardiasis in alcoholism and other chronic diseases in which altered immunity may play a role. Whenever *Giardia lamblia* is discovered in a patient with immunodeficiency and diarrheal disease, every effort should be made to eradicate the organism. Eradication may not be followed by improvement,[17,22] but often improvement is dramatic, and the contribution of *Giardia lamblia* is only then appreciated.[2,21,31]

Until the immunology of the intestine and giardiasis are more completely understood, it is helpful to think in the general terms and relationships depicted in Figure 1, *A* and *B*. Since giardiasis has been most often reported with various immunoglobulin deficiency states, these will be described below. We will omit descriptions of other immunodeficiency states which have been associated with malabsorption and diarrhea without giardiasis.[1,5,37]

SELECTIVE DEFICIENCY OF IGA

(Fig. 1*A*-c). Selective deficiency of IgA is relatively common and occurs in 0.14 to 0.9 per cent of the population.[7] In this condition there is a marked decrease in the number of IgA producing plasma cells in

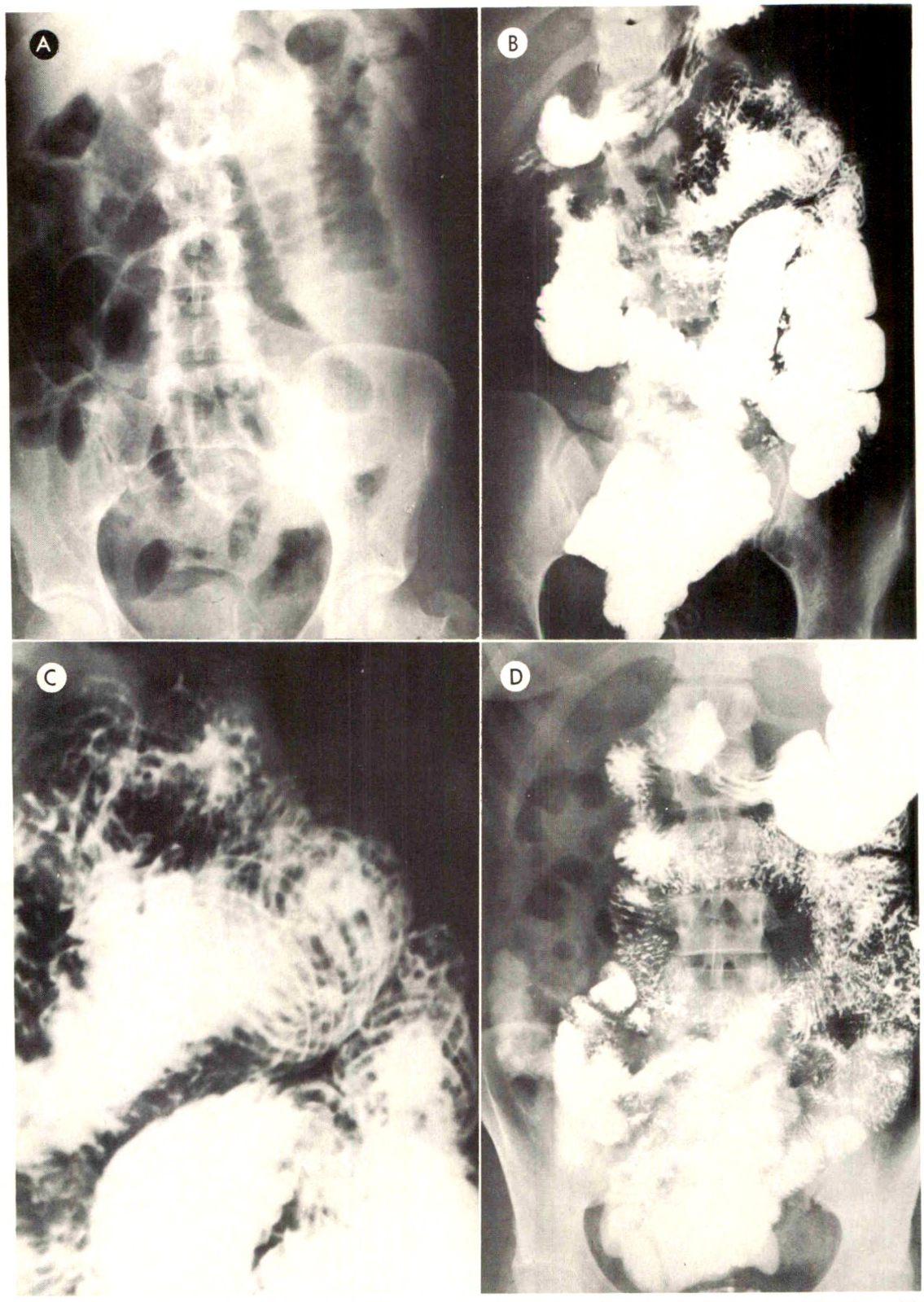

FIG. 6. *Giardiasis: selective IgA deficiency*. This 24 year old white female had a sudden onset of diarrhea with up to 20 stools per day containing 15 gm. of fat and cysts of *Giardia lamblia*. IgA was absent from the serum and duodenal fluid but other immunoglobulins were normal. At one point in the course she developed transient intestinal obstruction due to jejunojejunal intussusception. The patient suffered relapse after a single course of Flagyl, but her symptoms completely resolved after a second course. (*A*) Plain abdominal roentgenogram shows a pattern of incomplete small bowel mechanical obstruction. (*B*) An area of intussusception is present in the jejunum. (*C*) Close-up of *B*. A jejunojejunal intussusception is seen. (*D*) Subsequent gastrointestinal examination. The mucosal folds in the duodenum and jejunum are thickened, tortuous, and distorted.

the intestine and little or no IgA can be detected in the serum. IgG and IgM are present in normal concentrations and most of these individuals have no symptoms related to the intestine.[15,37,38]

(Fig. 1*A*-d). Crabbé and Heremans[12] first reported a patient with selective deficiency of IgA, steatorrhea, and mucosal biopsy abnormalities who responded to a gluten free diet. Symptoms recurred when gluten was reintroduced. No *Giardia lamblia* infestation was recognized in this patient or in similar reported cases[18,19,37] which have been summarized by Gelzayd *et al.*[15] These authors suggest the term IgA deficient sprue for this entity.

(Fig. 1*A*-e, f). Giardiasis may be solely responsible for diarrheal disease in some patients with selective IgA deficiency.[27] We have encountered such a patient who presented with transient small bowel obstruction due to intussusception (Fig. 6, *A–D*). Other cases have been reported in the literature, but we are aware of none where intussusception was documented. When *Giardia lamblia* is the sole cause of symptoms, eradication of the organism is curative.

IgA deficiency with nodular lymphoid hyperplasia and giardiasis

(Fig. 1 *B*-h). A few patients with IgA deficiency,[17,27] whether selective or as a part of primary acquired hypogammaglobulinemia or dysgammaglobulinemia[2,18,19,22,37] also have nodular lymphoid hyperplasia. Many, but not all, of these patients have giardiasis.[2,17,18,19,21,22,23,27,44] The relationships among these 3 conditions are depicted in Figure 1*B*. The simultaneous occurrence of 3 relatively rare conditions strongly suggests a causal relationship, but each may occur independently of the others,[4,8,9,14] and their true relationship is obscure. Hermans *et al.*[18,19] reported the clinical findings in 10 patients with this triad. Their patients all had low serum concentrations of IgA and IgG and usually of IgM. Subsequently, there have been reports with selective IgA deficiency[17] and borderline low IgA.[27] Clinical response to eradication of *Giardia lamblia* is unpredictable, but vigorous treatment is indicated since marked improvement may occur.[2,18,19,21,23]

Hodgson *et al.*[20] described the roentgenographic findings in the 10 original patients, and other descriptions are scattered in the literature.[2,17,22,23,26,44] Innumerable tiny nodules varying in size from 1 to 3 mm. are seen as polypoid mucosal masses. The nodules are generally most numerous in the jejunum, but the entire small bowel and colon[20,27,44] may be involved. Umbilication of the nodules has not been reported. This nodular pattern must be distinguished from scattered coarse nodular thickening of folds viewed on end which may be seen in any patient with giardiasis. Poor mixing of barium and secretions may give rise to a finely granular appearance which must also be distinguished. When giardiasis is present, the roentgenographic changes of inflammatory bowel disease and malabsorption may also be seen. Inflammatory changes disappear with therapy, but the nodules will persist.[2,20,27]

Infantile X-linked agammaglobulinemia

This condition is usually not associated with diarrheal disease[2,37] (Fig. 1*A*-c), although Ochs *et al.*[31] have studied 1 patient in whom giardiasis was clearly demonstrated to be the cause of severe malabsorption and diarrhea which responded to eradication of the organism (Fig. 1*A*-f). The small bowel series in Ochs' patient showed thickened folds in the duodenum and jejunum with marked segmentation and returned to normal following treatment.

Primary acquired hypogammaglobulinemia and dysgammaglobulinemia

Most of these patients, particularly early in the course of their disease and with only mild depression of serum immunoglobulin levels, have no gastrointestinal disturbance[19] (Fig. 1*A*-c). An estimated 20 per cent of patients have chronic diarrhea

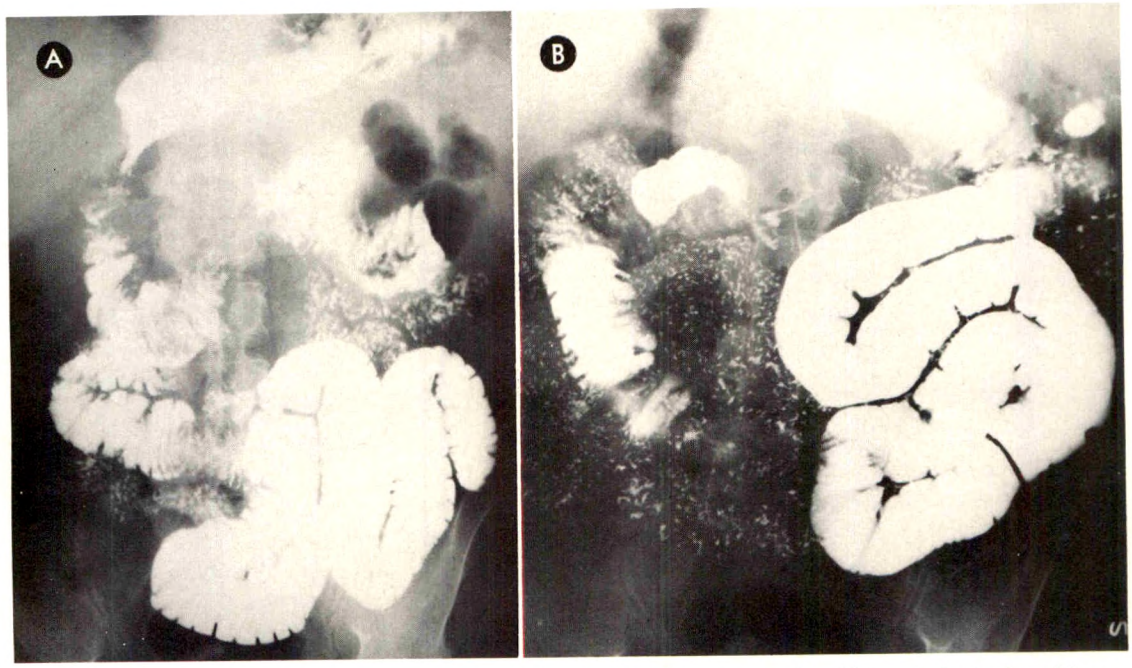

Fig. 7. *Giardiasis: primary acquired hypogammaglobulinemia.* This 54 year old white male had a long history of multiple pulmonary infections. He developed severe diarrhea and malabsorption with a 40 pound weight loss. *Giardia lamblia* trophozoites were identified by small bowel biopsy. IgA was absent from the serum; IgG and IgM were markedly depressed. Plasma cells were absent from the lamina propria of the intestine. Improvement was definite but incomplete after eradication of the parasite, and a sprue pattern persisted roentgenographically. (*A* and *B*) Small bowel studies show a sprue pattern with dilatation, flocculation and segmentation.

and malabsorption, a "sprue-like syndrome"[19,37] (*cf.* selective IgA deficiency and infantile x-linked agammaglobulinemia). The cause of malabsorption is usually not clearly determined, but it sometimes improves after some combination of antibiotics, gamma globulin injections, fresh frozen plasma infusions, corticosteroids, or a gluten free diet. *Giardia lamblia* often is either absent from the intestine (Fig. 1*A*-d) or eradication produces little or no improvement in symptoms[5,15,18,19,22,43] (Fig. 1*A*-e). In some of these patients, however, giardiasis is apparently the dominant cause of symptoms, and marked improvement occurs with treatment[2] (Fig. 1*A*-f). The small bowel roentgen findings in primary acquired hypogammaglobulinemia have been reviewed by Vermess *et al.*[44] In such patients with chronic malabsorption, a sprue pattern is present with atonic, dilated small bowel loops, increased secretions and prolonged transit (Fig. 7, *A* and *B*).

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REFERENCES

1. Ament, M. E., and Ochs, H. D. Gastrointestinal manifestations of chronic granulomatous disease. *New England J. Med.*, 1973, *288*, 382–387.
2. Ament, M. E., and Rubin, C. E. Relation of giardiasis to abnormal intestinal structure and function in gastrointestinal immunodeficiency syndromes. *Gastroenterology*, 1972, *62*, 216–226.
3. Antia, F. P., Desai, H. G., Jeejeebhoy, K. N., Kane, M. P., and Borkar, A. V. Giardiasis in adults: incidence, symptomatology and absorption studies. *Indian J. M. Sc.*, 1966, *20*, 471–477.
4. Bahk, Y. W., Ahn, J. S., and Choi, H. J.

Lymphoid hyperplasia of stomach presenting as umbilicated polypoid lesions. *Radiology*, 1971, *100*, 277–280.

5. BINDER, H. J., and REYNOLDS, R. D. Control of diarrhea in secondary hypogammaglobulinemia by fresh plasma infusions. *New England J. Med.*, 1967, *277*, 802–803.
6. BRANDBORG, L. L., TANKERSLEY, C. B., GOTTLIEB, S., BARANCIK, M., and SARTOR, V. E. Histological demonstration of mucosal invasion by Giardia lamblia in man. *Gastroenterology*, 1967, *52*, 143–150.
7. BUCKLEY, R. H., and DEES, S. C. Correlation of milk precipitins with IgA deficiency. *New England J. Med.*, 1969, *281*, 465–469.
8. CAPITANIO, M. A., and KIRKPATRICK, J. A. Lymphoid hyperplasia of colon in children. *Radiology*, 1970, *94*, 323–327.
9. CHARLESWORTH, D., FOX, H., and MAINWARING, A. R. Benign lymphoid hyperplasia of terminal ileum. *Am. J. Gastroenterol.*, 1970, *53*, 579–584.
10. CHILGREN, R. A., MEUWISSEN, H. J., QUIE, P. G., and HONG, R. Chronic mucocutaneous candidiasis, deficiency of delayed hypersensitivity, and selective local antibody defect. *Lancet*, 1967, *2*, 688–693.
11. COITNER, J. A. Giardiasis: cause of celiac syndrome. *A.M.A. Am. J. Dis. Child.*, 1959, *98*, 311–316.
12. CRABBÉ, P. A., and HEREMANS, J. F. Selective IgA deficiency with steatorrhea: new syndrome. *Am. J. Med.*, 1967, *42*, 319–326.
13. EIDELMAN, S., DAVIS, S. D., LAGUNOFF, D., and RUBIN, C. E. Relationship between intestinal plasma cells and serum immunoglobulin A (IgA) in man. (Abst.). *J. Clin. Invest.*, 1966, *45*, 1003–1004.
14. FRANKEN, E. A. Lymphoid hyperplasia of colon. *Radiology*, 1970, *94*, 329–334.
15. GELZAYD, E. A., MCCLEERY, J. L., MELNYK, C. S., and KRAFT, S. C. Intestinal malabsorption and immunoglobulin deficiency. *A.M.A. Arch. Int. Med.*, 1971, *127*, 141–147.
16. GINSBERG, A. L. Alterations in immunologic mechanisms in diseases of gastrointestinal tract. *Am. J. Digest. Dis.*, 1971, *16*, 61–80.
17. GRYBOSKI, J. D., SELF, T. W., CLEMETT, A., and HERSKOVIC, T. Selective immunoglobulin A deficiency and intestinal nodular lymphoid hyperplasia: correction of diarrhea with antibiotics and plasma. *Pediatrics*, 1967, *42*, 833–837.
18. HERMANS, P. E. Nodular lymphoid hyperplasia of small intestine and hypogammaglobulinemia: theoretical and practical considerations. *Fed. Proc.*, 1967, *26*, 1606–1611.
19. HERMANS, P. E., HUIZENGA, K. A., HOFFMAN, H. N., BROWN, A. L., JR., and MARKOWITZ, H. Dysgammaglobulinemia associated with nodular lymphoid hyperplasia of small intestine. *Am. J. Med.*, 1966, *40*, 78–89.
20. HODGSON, J. R., HOFFMAN, H. N., and HUIZENGA, K. A. Roentgenologic features of lymphoid hyperplasia of small intestine associated with dysgammaglobulinemia. *Radiology*, 1967, *88*, 883–888.
21. HOSKINS, L. C., WINAWER, S. J., BROITMAN, S. A., GOTTLIEB, L. S., and ZAMCHECK, N. Clinical giardiasis and intestinal malabsorption. *Gastroenterology*, 1967, *53*, 265–279.
22. HUGHES, W. S., CERDA, J. J., HOLTZAPPLE, P., and BROOKS, F. P. Primary hypogammaglobulinemia and malabsorption. *Ann. Int. Med.*, 1971, *74*, 903–910.
23. JOHNSON, B. L., GOLDBERG, L. S., POPS, M. A., and WEINER, M. Clinical and immunological studies in case of nodular lymphoid hyperplasia of small bowel. *Gastroenterology*, 1971, *61*, 369–374.
24. KRAFT, S. C., and KIRSNER, J. B. Immunological apparatus of gut and inflammatory bowel disease. *Gastroenterology*, 1971, *60*, 922–951.
25. LOUISY, C. L., and BARTON, C. J. Radiological diagnosis of strongyloides stercoralis enteritis. *Radiology*, 1971, *98*, 535–541.
26. MARSHAK, R. H., RUOFF, M., and LINDNER, A. E. Roentgen manifestations of giardiasis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *104*, 557–560.
27. MILANO, A. M., LAWRENCE, L. R., and HOROWITZ, L. Nodular lymphoid hyperplasia of small intestine and colon with giardiasis: case with borderline serum IgA levels. *Am. J. Digest. Dis.*, 1971, *16*, 735–737.
28. MOORE, G. T., CROSS, M., MCGUIRE, D., MOLLOHAN, C. S., GLEASON, N. N., HEALY, G. R., and NEWTON, L. H. Epidemic giardiasis at ski resort. *New England J. Med.*, 1969, *281*, 402–407.
29. MORECKI, R., and PARKER, J. G. Ultrastructural studies of human Giardia lamblia and subjacent jejunal mucosa in subject with steatorrhea. *Gastroenterology*, 1967, *52*, 151–164.
30. MUELLER, J. C., JONES, A. L., and BRANDBORG, L. L. Scanning electron microscope observation in human giardiasis. Scanning electron microscopy/1973, (Part III). In: Proceedings of the Workshop on Scanning Electron Microscopy in Pathology. IIT Research Institute, Chicago, Ill., April, 1973, pp. 557–564.
31. OCHS, H. D., AMENT, M. E., and DAVIS, S. D. Giardiasis with malabsorption in X-linked agammaglobulinemia. *New England J. Med.*, 1972, *287*, 341–342.
32. PAULINO, G. B., JR., and WITTENBERG, J. Intestinal capillariasis: new cause of malabsorption pattern. AM. J. ROENTGENOL., RAD.

Therapy & Nuclear Med., 1973, *117*, 340–345.

33. Peterson, G. M. Intestinal changes in Giardia lamblia infestation. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *77*, 670–677.
34. Plaut, A. G., and Prayoonsri, K. Immunoglobulins in human small intestinal fluid. *Gastroenterology*, 1969, *56*, 522–530.
35. Rendtorff, R. C. Experimental transmission of human intestinal protozoan parasites. II. Giardia lamblia cysts given in capsules. *Am. J. Hyg.*, 1954, *59*, 209–220.
36. Rivera, E., Maldonado, N., Vélez-Garcia, E., Grillo, A. J., and Malaret, G. Hyperinfection syndrome with strongyloides stercoralis. *Ann. Int. Med.*, 1970, *72*, 199–204.
37. Roy, C. C., and Dubois, R. S. Human gut and immune homeostasis: brief survey of current concepts. *Clin. Pediat.*, 1971, *10*, 275–281.
38. Schwartz, D. P., and Buckley, R. H. Serum IgE concentrations and skin reactivity to anti-IgE antibody in IgA-deficient patients. *New England J. Med.*, 1971, *284*, 513–517.
39. Shultz, M. G., Brodsky, R. E., and Spencer, H. C. Letter to Editor. *New England J. Med.*, 1974, *290*, 862.
40. Smith, R. T. Gamma-A immunoglobulins and concept of local immunity. *Pediatrics*, 1969, *43*, 317–321.
41. Takano, J., and Yardley, J. H. Jejunal lesions in patients with giardiasis and malabsorption: electron microscopic study. *Bull. Johns Hopkings Hosp.*, 1965, *116*, 413–429.
42. Tomasi, T. B., Jr. Secretory immunoglobulins. *New England J. Med.*, 1972, *287*, 500–506.
43. Twomey, J. J., Jordan, P. H., Jarrold, T., and Trubowitz, S. Syndrome of immunoglobulin deficiency and pernicious anemia. *Am. J. Med.*, 1969, *47*, 340–353.
44. Vermess, M., Waldmann, T. A., and Pearson, K. D. Radiographic manifestations of primary acquired hypogammaglobulinemia. *Radiology*, 1973, *107*, 63–69.
45. Welch, P. B. Giardiasis with unusual findings. *Gastroenterology*, 1944, *3*, 98–102.
46. Yardley, J. H., and Bayless, T. M. Giardiasis. *Gastroenterology*, 1967, *52*, 301–304.
47. Yardley, J. H., Takano, J., and Hendrix, T. R. Epithelial and other mucosal lesions of jejunum in giardiasis: jejunal biopsy studies. *Bull. Johns Hopkins Hosp.*, 1964, *115*, 389–406.

ESOPHAGEAL MONILIASIS*

A REVIEW OF COMMON AND LESS FREQUENT CHARACTERISTICS

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ABSTRACT:

Invasion of the esophagus by *Candida albicans* probably occurs more frequently than the reported cases suggest. The disease usually occurs following immunosuppression but may occur after antibiotic therapy. It is heralded by the sudden onset of severe pain and dysphagia, and recovery or death from dissemination may occur. Roentgenographically, impaired motility is much more prominent than disordered motility or spasm. Narrowing, a cobblestone epithelium, and later, erosions and ulcerations are seen. Antifungal agents are adequate therapy.

SINCE the original description of roentgenographic changes resulting from esophageal moniliasis or candidiasis by Andrén and Theander in 1956,[1] no more than 100 additional cases have been reported. Most of these have appeared as isolated case reports with few analyses of large series. Collectively these reports combined with our own cases form a clinical and roentgenographic spectrum of the disease which is the subject of this report.

INCIDENCE

The Cancer Institute of Jutland found esophageal moniliasis in 5 per cent of 694 consecutively admitted patients.[13] At autopsy an even higher prevalence has been demonstrated, particularly in patients dying of some of the predisposing diseases. In a postmortem series of leukemic patients, 10 per cent had esophageal and 16 per cent gastrointestinal invasion by *Candida albicans*.[16] These findings indicate that the disease occurs more frequently than the isolated case reports suggest.

PATHOGENESIS

Candida albicans is found in the mouth, throat, sputum, and feces of normal children and adults. In almost all patients esophageal invasion by these fungi is an autonomous infection resulting when an immunologic imbalance develops between the host and the normal commensal flora. This happens most frequently in patients with leukemia or lymphoma and in those requiring corticosteroid therapy. Esophageal invasion has also been reported in patients with generalized debilitation from diabetes,[18] systemic lupus erythematosis,[9] multiple myeloma,[18] ulcerative colitis,[11] achalasia of the esophagus,[11] hemoglobin sickle-cell disease,[17] renal failure,[6] Paget's disease,[7] and other diseases.

Candidiasis of the esophagus has been found in otherwise healthy adults after a course of antibiotics for a sore throat or upper respiratory infection.[4] Tetracyclines, in particular, have been implicated in esophageal invasion by *Candida albicans*. It has been postulated that the fungi overgrow because they do not have to compete for nutritive substances with the normal bacterial flora which has been removed by the antibiotics.[23] Occasionally esophageal moniliasis may occur even in healthy adults where exposure to such contributing causes cannot be traced.[12]

CLINICAL SYMPTOMS

Characteristically esophageal moniliasis manifests itself by dysphagia associated with intense pain localized to the upper retrosternal area. The pain may radiate

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into the back following the distribution of pain originating from the esophagus.[12] Chest pain may be very severe and may precede dysphagia so that myocardial infarction has been initially suspected in some patients.[18] Pain and dysphagia are of sudden onset, progress rapidly if untreated, and are not relieved by the administration of alkali.[19]

In some patients the fungi invade the oral cavity before spreading to the esophagus. Oral thrush may be mild, however, and can occur concomitantly with esophageal disease. It may also be entirely absent.[10,12]

The course of esophageal moniliasis varies widely. Complete recovery may be rapid or systemic invasion may occur as mycelia enter the systemic circulation through ruptured esophageal vessels. Dissemination of moniliasis to other organs is invariably associated with extensive invasion of the gastrointestinal tract.[5] Systemic candidiasis frequently leads to death.

Recovery from symptoms may occur after 2 days of treatment.[13] After 1 week of therapy, the esophagus has been shown to be normal by esophagoscopy.[17] Recovery usually takes place after institution of antifungal therapy with nystatin or amphotericin B. It may also follow simple withdrawal of contributing agents such as corticosteroids or an improvement in the patient's general clinical status (Fig. 7, *A* and *B*; and 8, *A* and *B*). After recovery from esophageal moniliasis, patients are still susceptible to recurrent attacks.[6]

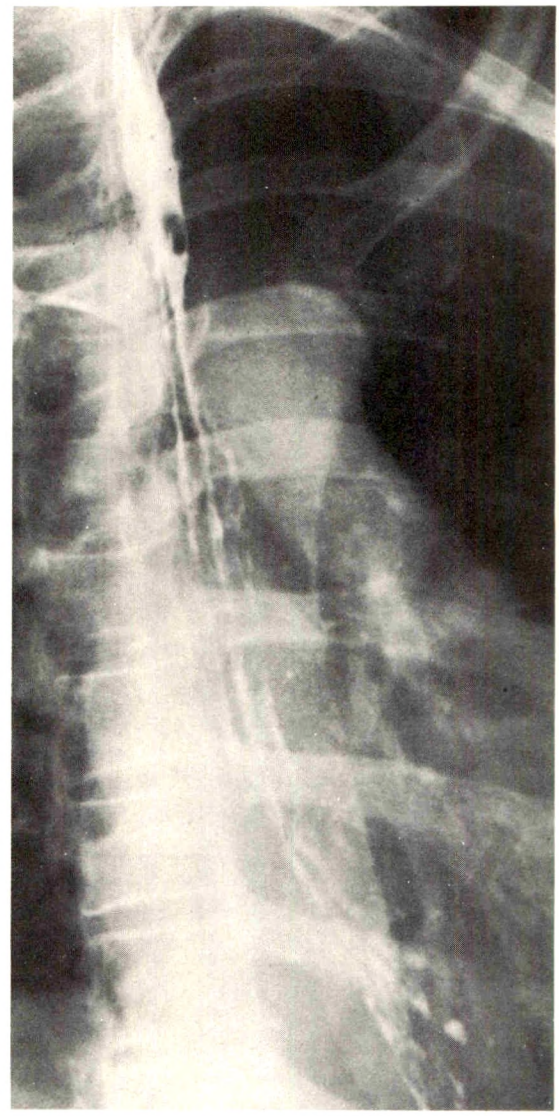

Fig. 1. Esophageal moniliasis in a 14 year old girl with poorly controlled diabetes. The esophageal folds are irregularly widened and resemble those in patients with varices.

NONROENTGENOGRAPHIC DIAGNOSTIC STUDIES

Because *Candida albicans* can be found in the mouth and feces of healthy individuals, smears and cultures from these sites usually do not establish clinical disease conclusively. When the fungi are found in the mycelial form rather than exclusively as yeasts, however, invasion can be assumed. Esophagoscopy with or without biopsy can be particularly helpful when the esophagus is normal roentgenographically and no thrush can be seen in the oral cavity. A high risk of perforation from endoscopy in patients with monilial esophagitis is emphasized in published reports. We could find only 1 reported case where an esophagocutaneous fistula developed shortly after esophagoscopy.[22] Jensen *et al.*,[13] in particular, have shown endoscopy to be more sensitive in detecting early disease than the roentgenogram.

The presence of thrush in association with a characteristic esophagogram is con-

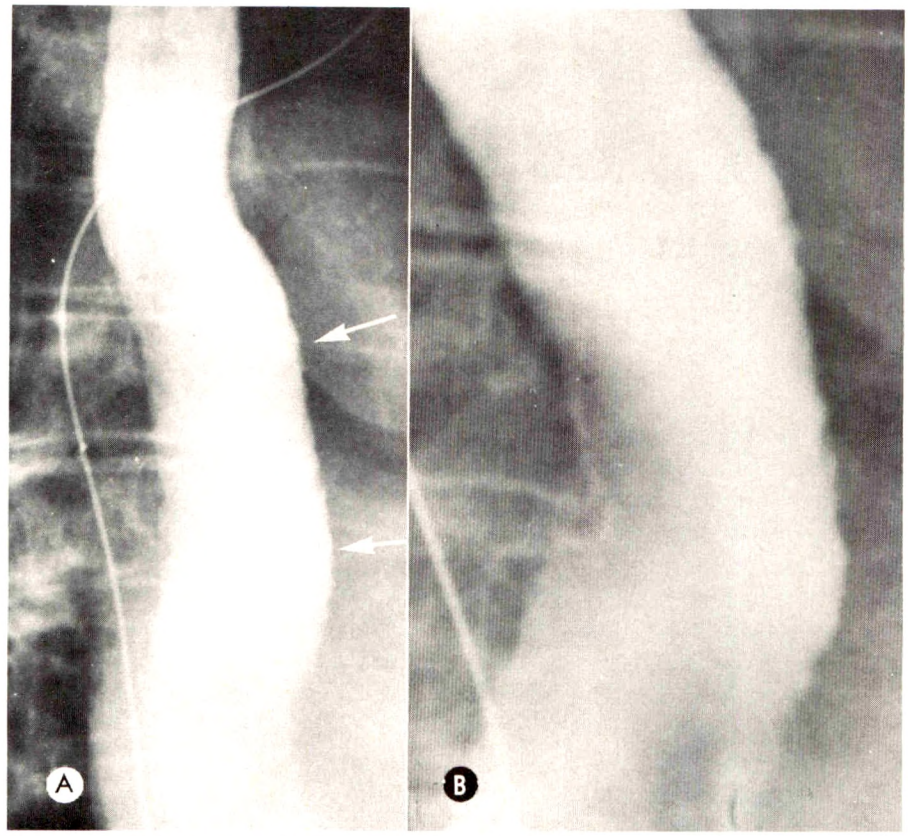

FIG. 2. (*A*) The esophageal lumen is finely nodular particularly along its left lateral side (arrows) at the carinal and subcarinal level. (*B*) On close inspection (on the magnified view) variations in the size of these nodular defects are visible.

sidered conclusive for esophageal invasion in most patients.[13] In such circumstances it does not seem prudent to delay institution of therapy because the organism cannot be isolated. Such an empirical approach seems justified because of the high morbidity and mortality that systemic moniliasis carries.

ROENTGENOGRAPHIC FINDINGS

Motility Disorders. The esophagus becomes atonic and peristalsis is impaired when *Candida albicans* invades the esophagus. Primary and secondary peristaltic waves are diminished in frequency and magnitude.[9,17,19] The degree of impaired motility directly reflects the severity of esophageal involvement and is usually worse in the distal portion of the esophagus. Spasm and irritability were frequently observed in reported cases.[14] Our own experience does not support this finding although many of our patients were examined with cinematography in addition to standard fluoroscopy and filming. One may wonder whether in many described cases the esophagus was actually incompletely distended from an inability to swallow an adequate bolus, thus appearing fortuitously spastic. Spasm can occur in conjunction with diminished peristalsis as a transient event, however. This has been well demonstrated with a cinematography sequence by Sanders *et al.*[17]

Barium was intermittently aspirated into the larynx and trachea in 1 of the 2 originally reported cases.[1] Such a swallowing dysfunction is actually rare and is not reported in other adult cases including those

from our own group. In children with thrush, painful swallowing frequently leads to aspiration and bronchopneumonia.[1,4]

Localization. Esophageal moniliasis probably results from spread of *Candida albicans*

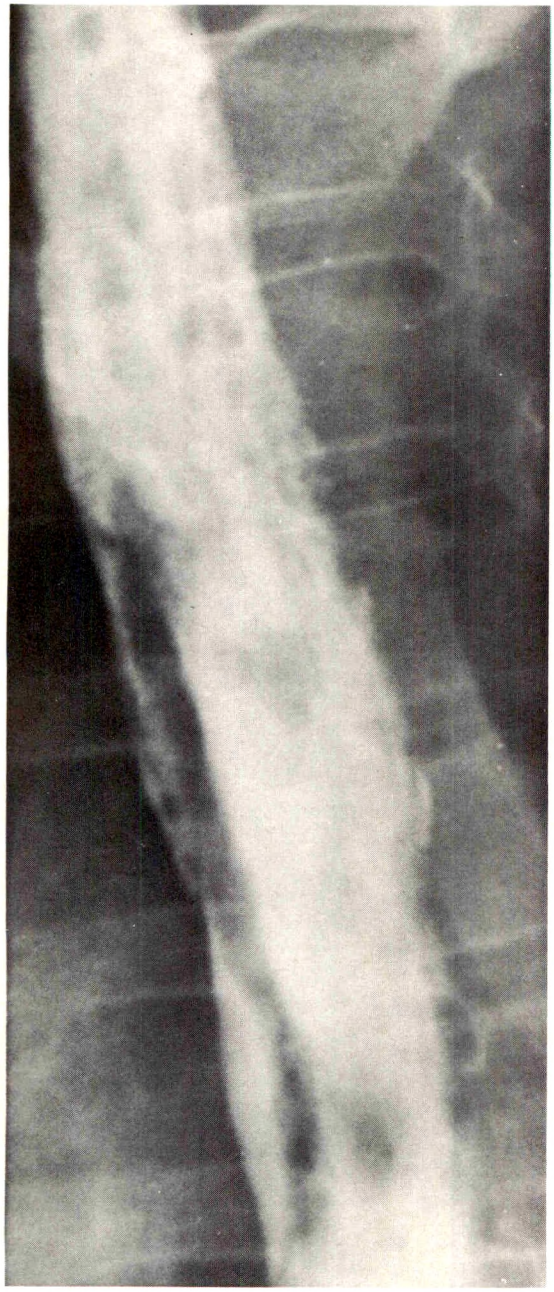

Fig. 3. Detail view of the esophagus at the thoracic inlet in a leukemic patient with esophageal moniliasis. Defects of various sizes and shapes produce an irregular cobblestone pattern.

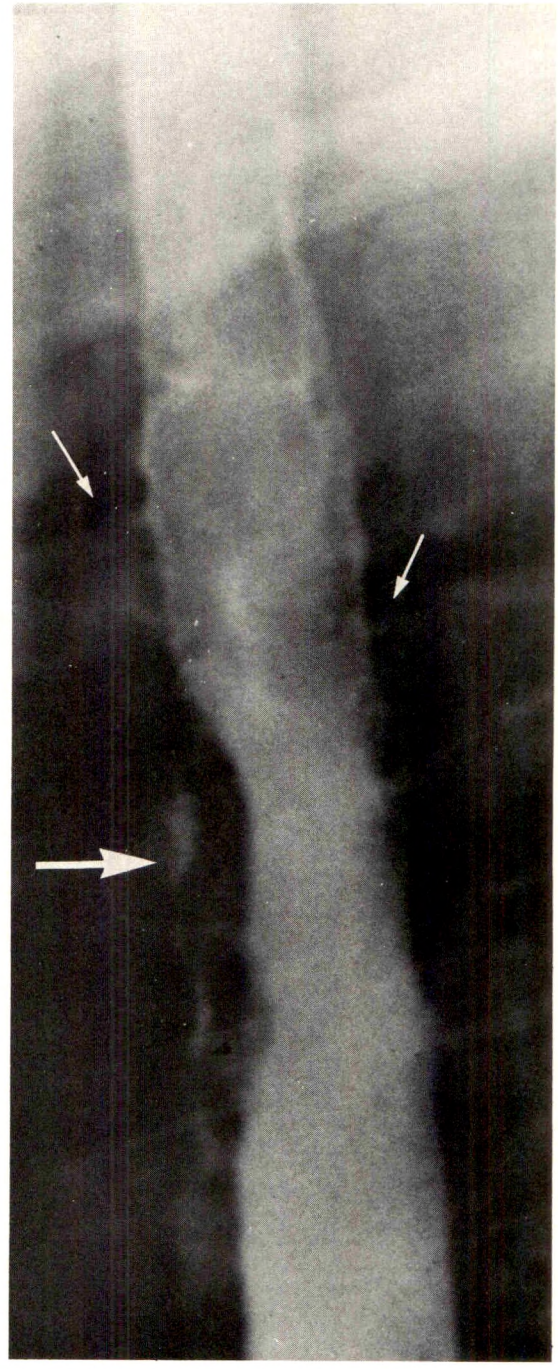

Fig. 4. Same patient as in Figure 1, examined at a later date with more severe disease. Detail view of middle third of intrathoracic esophagus. Barium has penetrated under a detached pseudomembrane forming a double tract which parallels the esophageal lumen (coarse arrow). In other areas the esophagus is ulcerated with hair-like fissures (fine arrows).

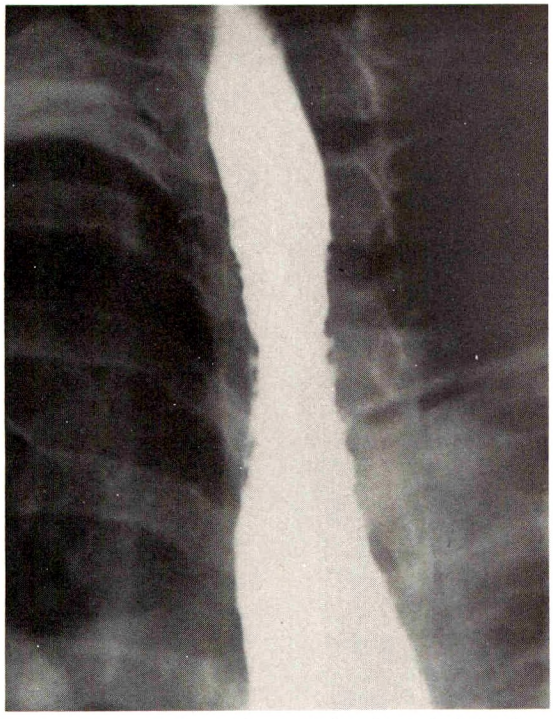

FIG. 5. Coarser ulcerations at the thoracic inlet in a patient with diffuse monilial invasion of the esophagus. The esophageal lumen is slightly narrowed at this point. More distally, the esophagus was actually wide and atonic. Such localized ulcerations above the aortic arch may resemble intramural pseudodiverticula.

from the oral cavity into the esophagus.[19] The cervical esophagus has been shown to be involved from the level of the cricopharyngeus by esophagoscopy,[17] yet involvement at the cervical level or in the hypopharynx has not been described roentgenographically.[4] The intrathoracic esophagus is invaded by the fungi segmentally or diffusely. Surprisingly, with segmental disease there is a predilection for involvement of the distal and middle sections (Fig. 6).[5]

Esophageal Lumen. Although narrowing of the esophageal lumen has been reported early in the course of the disease, the lumen may appear fortuitously diminished because of incomplete distention by an inadequate bolus. As the disease progresses, compromise of luminal diameter may occur from both edema of the mucosa and the presence of an overlying pseudomembrane. With spread of the disease through the esophageal wall, a long esophageal segment may become constricted. Although the lumen usually tapers into the stricture, occasionally the diameter change may be abrupt, resembling an esophageal neoplasm.

Esophageal Morphology. Early in the

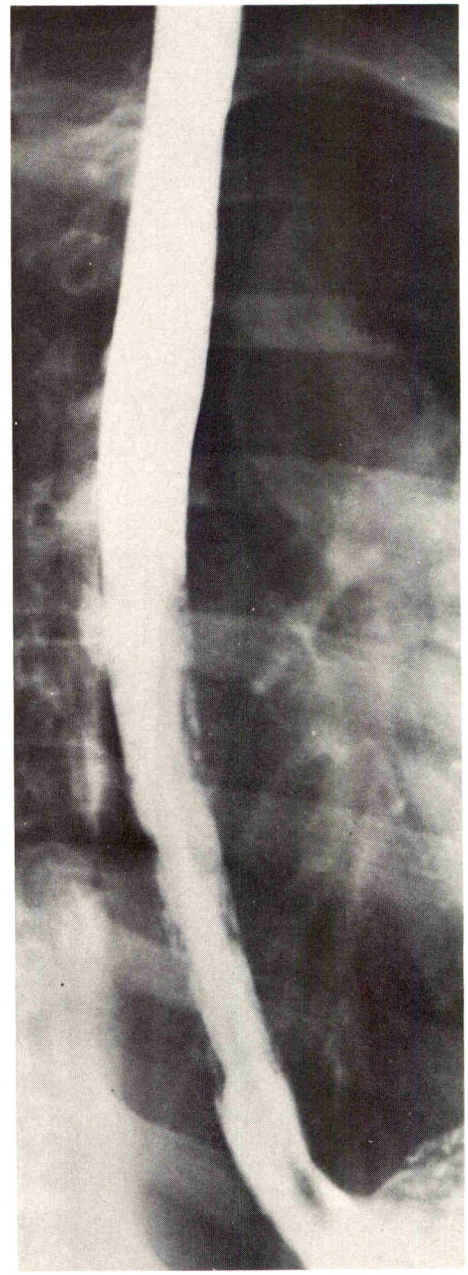

FIG. 6. A characteristic distribution of esophageal moniliasis with involvement of the distal two-thirds. The nodular defects narrow the lumen slightly.

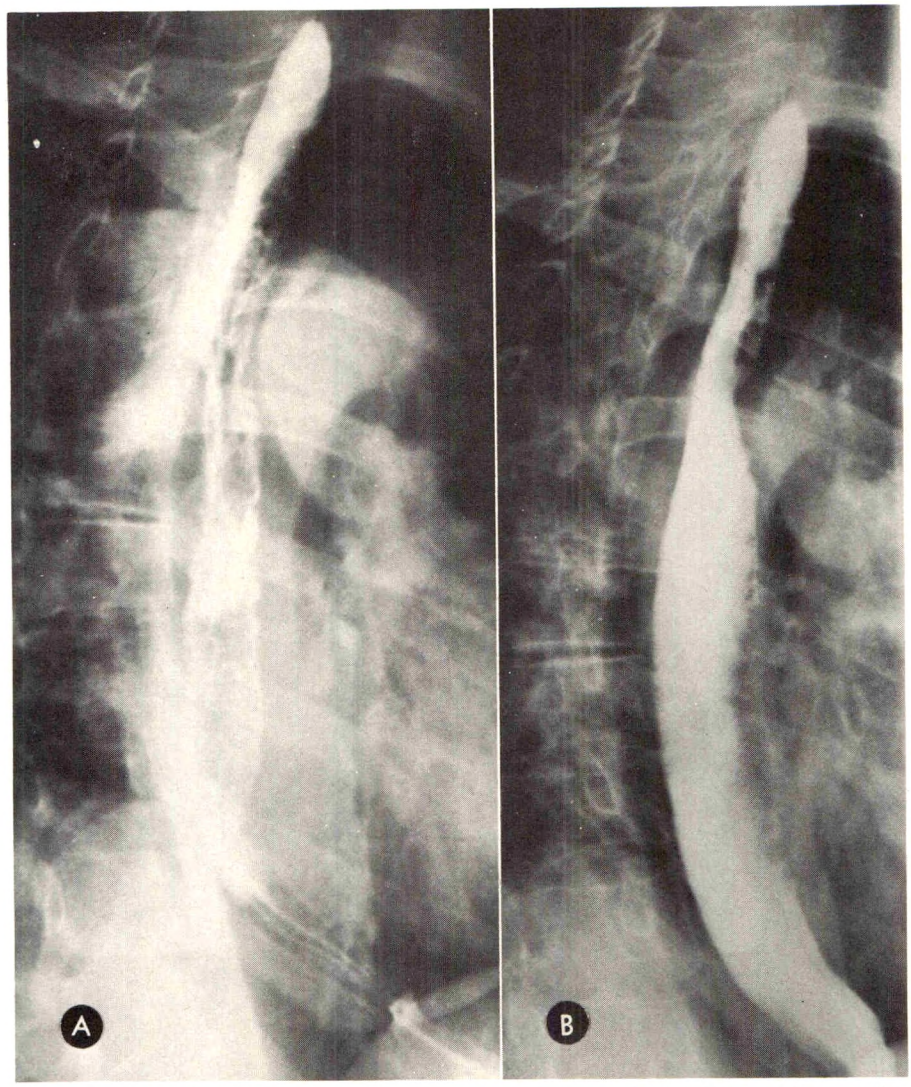

Fig. 7. (*A*) Extensive monilial involvement of the esophagus in an adult patient with acute leukemia. (*B*) A repeat esophagogram 1 month later after chemotherapeutically induced remission of leukemia and a course of nystatin. Although the patient had no symptoms of esophageal disease at this time, a short esophageal segment just above the aortic arch remains abnormal.

course of the disease, an esophagogram may show only minor irregularities of the mucosal folds (Fig. 1; and 2, *A* and *B*).[18] This progresses to a frank cobblestone pattern of the esophagus. Grossly and histologically, cobblestoning reflects a granular mucosa caused by submucosal edema and associated inflammatory changes (Fig. 3).[9,14] The esophageal surface may become granular or nodular also from direct seeding of *Candida albicans* colonies on the mucosa.[11] Soon erosions and ulcerations of the esophageal mucosa develop, and the esophageal lumen becomes shaggy roentgenographically (Fig. 5). Ulcerations are multiple and of varying size. The intervening esophageal contour is nodular with irregular, round and oval defects of various sizes. At this point the ulcerated esophageal mucosa is covered by a whitish-yellow membrane composed of fungi and debris. Microscopically, growth of the organisms can be seen in the mucosal and submucosal layers; and mucosal erosions and ulceration, intramural

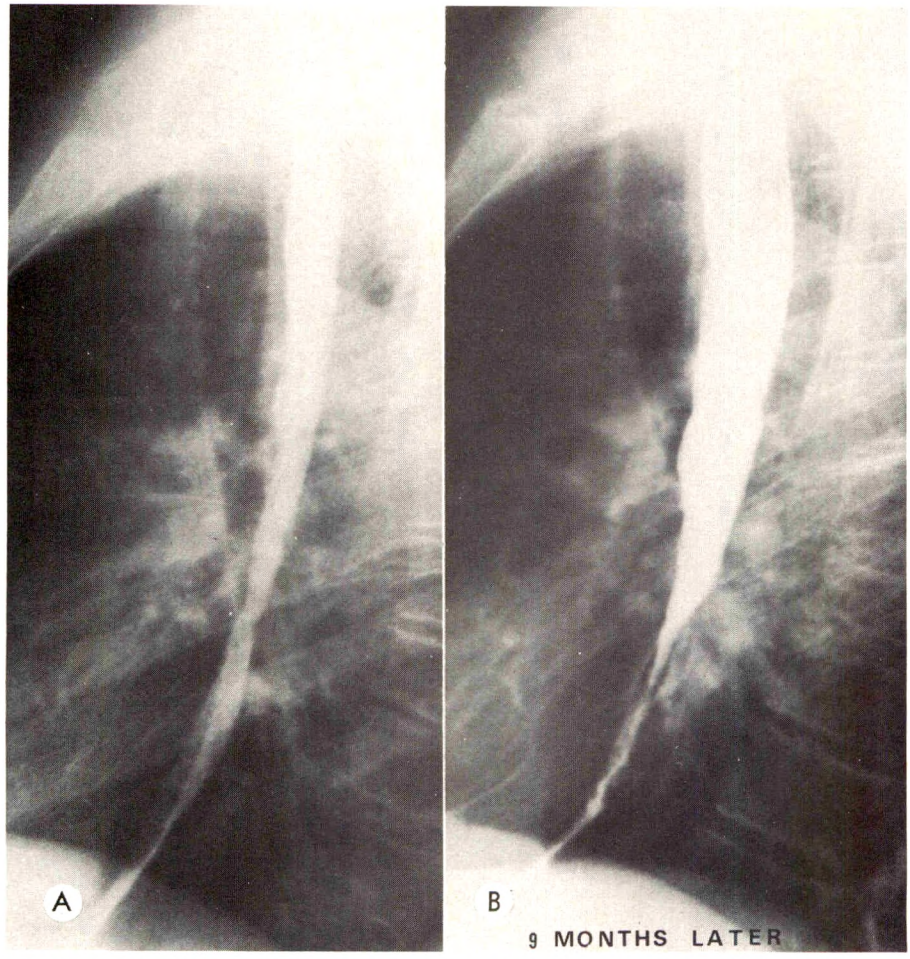

FIG. 8. (*A*) Diffuse monilial esophagitis with more severe involvement of the distal two-thirds. (*B*) A permanent stricture of the distal esophagus, an infrequent complication of esophageal moniliasis, developed 9 months later.

hemorrhage, and mural necrosis are present. Inflammatory cells infiltrate the esophageal wall.[5]

Occasionally the pseudomembrane may partially separate from the esophageal wall permitting contrast material to penetrate under it resulting in a double-contour appearance (Fig. 4).[6,10] Contrast material adheres to the esophageal mucosa for prolonged periods because of dryness of the surface[10] and inefficient esophageal emptying because of impaired peristalsis.

DIFFERENTIAL DIAGNOSIS

Round and oval nodular defects of esophageal moniliasis may simulate varices of the esophagus. Unlike varices, however, the defects do not change size or appearance with variations in intrathoracic pressure.[20] Occasionally an asymmetrical, abrupt stenosis due to monilial disease may simulate an infiltrating neoplasm.[7] The most important differential point for distinguishing varices and carcinoma from moniliasis is the striking variability of roentgen findings over a short time period.[20] There is usually no difficulty in distinguishing peptic esophagitis where ulcerations are single or few in number, unlike those in moniliasis. Corrosive esophagitis can be excluded by the clinical history.

One must remember that moniliasis may occur in patients affected by other esophageal disease such as achalasia, carcinoma of

the esophagus, and lupus erythematosis. Thus the initial abnormalities of esophageal motility and morphology can be in part or predominently due to the antecedent disease. This can be best evaluated by serial esophagograms or by comparison with prior baseline study. While microscopic infiltration of the esophagus by leukemia is not rare, lesions of such size that they can be recognized grossly are infrequent.[8] Lymphomatous invasion of the esophagus is invariably due to a direct extension of neoplasm from the mediastinum.[8]

When ulcerations are deep and undermined, they may resemble esophageal diverticula. More recently these projections have been shown to be dilated esophageal glands, located intramurally.[2,3,15] Although monilia were cultured from esophagi in patients with such pseudodiverticulosis, it is unlikely that the fungus was a pathogen as assumed in an earlier report.[21] As with varices and carcinoma, serial esophagograms will help to distinguish diverticula from ulcers.

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I am grateful to Joseph Ferrucci, M.D. of Massachusetts General Hospital, Boston, Massachusetts for making available Figure 8, *A* and *B*.

REFERENCES

1. Andrén, L., and Theander, G. Roentgenographic appearances of esophageal moniliasis. *Acta radiol.*, 1956, *46*, 571–574.
2. Beauchamp, J. M., Nice, C. M., Belanger, M. A., and Nietzschman, H. R. Esophageal intramural pseudodiverticulosis. *Radiology*, 1974, *113*, 273–277.
3. Boyd, R. M., Bogoch, A., Greig, J. H., and Trites, A. E. W. Esophageal intramural pseudodiverticulosis. *Radiology*, 1974, *113*, 267–271.
4. Brown, J. W., and McKee, W. M. Acute monilial esophagitis occurring without underlying disease in young male. *Digest. Dis.*, 1972, *17*, 85–88.
5. Eras, P., Goldstein, M. J., and Sherlock, P. Candida infection of gastrointestinal tract. *Medicine*, 1972, *51*, 367–369.
6. Friedmann, G., and Tismer, R. Candida oesophagitis. *German M. Monthly*, 1968, *13*, 418–420.
7. Gibson, M. J., and Harris, M. Unusual case of monilial oesophagitis. *Brit. J. Radiol.*, 1967, *40*, 391–392.
8. Givler, R. L. Esophageal lesions in leukemia and lymphoma. *Digest. Dis.*, 1970, *15*, 31–36.
9. Goldberg, H. I., and Dodds, W. J. Cobblestone esophagus due to monilial infection. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 608–612.
10. Grieve, N. W. T. Monilial oesophagitis. *Brit. J. Radiol.*, 1964, *37*, 551–554.
11. Guyer, P. B., Brunton, F. J., and Rooke, H. W. P. Candidiasis of oesophagus. *Brit. J. Radiol.*, 1971, *44*, 131–136.
12. Holt, J. M. Candida infection of oesophagus. *Gut*, 1968, *9*, 227–231.
13. Jensen, K. B., Stenderup, A., Thomsen, J. B., and Bichel, J. Oesophageal moniliasis in malignant neoplastic disease. *Acta med. scandinav.*, 1964, *175*, 455–459.
14. Kaufman, S. A., Scheff, S., and Levene, G. Esophageal moniliasis. *Radiology*, 1960, *75*, 726–732.
15. Lupovitch, A., and Tippins, R. Esophageal intramural pseudodiverticulosis: disease of adnexal glands. *Radiology*, 1974, *113*, 271–273.
16. Prolla, J. C., and Kirsner, J. B. Gastroin testinal lesions and complications of leukemias. *Ann. Int. Med.*, 1964, *61*, 1084–1103.
17. Sanders, E., Levinthal, C., and Donner, M. W. Monilial esophagitis in patient with hemoglobin SC disease. *Ann. Int. Med.*, 1962, *57*, 650–654.
18. Sheft, D. J., and Shrago, G. Esophageal moniliasis: spectrum of disease. *J.A.M.A.*, 1970, *213*, 1859–1862.
19. Svoboda, V. M. Röntgenologische Symptomatologie der Candida-Ösophagitiden. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1964, *100*, 334–341.
20. Svoboda, M., Sochmann, J., and Urbanova, D. Tumoriodes Röntgenbild der Speiseröhre bei Moniliasis. *Radiol. Diag.*, 1962, *3*, 79–85.
21. Troupin, R. H. Intramural esophageal diverticulosis and moniliasis: possible association. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 613–616.
22. Weiss, J., and Epstein, B. S. Esophageal moniliasis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1962, *88*, 718–720.
23. Woods, J. W., Manning, I. H., Jr., and Patterson, C. N. Monilial infections complicating therapeutic uses of antibiotics. *J.A.M.A.* 1951, *145*, 207–211.

PERFORATED VISCUS PRESENTING WITH GAS IN THE SOFT TISSUES (SUBCUTANEOUS EMPHYSEMA)*

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ABSTRACT:

We have reviewed the spectrum of gaseous densities in the soft tissues secondary to a perforated viscus. All patients presented late and most were elderly. The most common surgical procedure was diversion of the fecal stream proximal to the perforation. In our series 4 of 7 patients died in the immediate postoperative period. Knowledge of the mechanism and differential diagnosis of this entity will prevent overlooking this possibility, as occurs too frequently, particularly with subcutaneous emphysema of the leg.

IN 1853, 42 years before Roentgen's discovery, the first case of subcutaneous emphysema secondary to a perforated hollow viscus was reported.[1] Review of the literature 100 years later revealed only 38 cases of this unusual presentation.[17] Roentgenographic findings of this entity have been recorded infrequently since.

In the past 11 years we have collected 7 cases which illustrate the spectrum of gas in the soft tissues following a perforation within the gastrointestinal tract. These include 2 cases each of perforation secondary to colonic carcinoma and diverticulitis. A single case each of perforation of the ileum, the appendix, and a duodenal diverticulum is also included. We believe this to be the first reported case of subcutaneous emphysema secondary to a ruptured duodenal diverticulum.

REPORT OF CASES

CASE I. This 71 year old white female was admitted to S.M.H. for evaluation of a pelvic mass. A laparotomy revealed adhesions involving the omentum and small bowel. A lysis of adhesions was performed. However, abdominal distention began on the first postoperative day and continued to the fifth postoperative. Roentgenograms at this time revealed a pattern of small bowel obstruction and perforation with subcutaneous emphysema (Fig. 1). Repeat laparotomy showed that the peritoneal sutures had given over a length of 6 inches. Intestinal contents were found in the peritoneal cavity. Two perforations of the distal small bowel and adhesions were found. The patient was discharged on the nineteenth hospital day in satisfactory condition.

CASE II. This 47 year old white female was

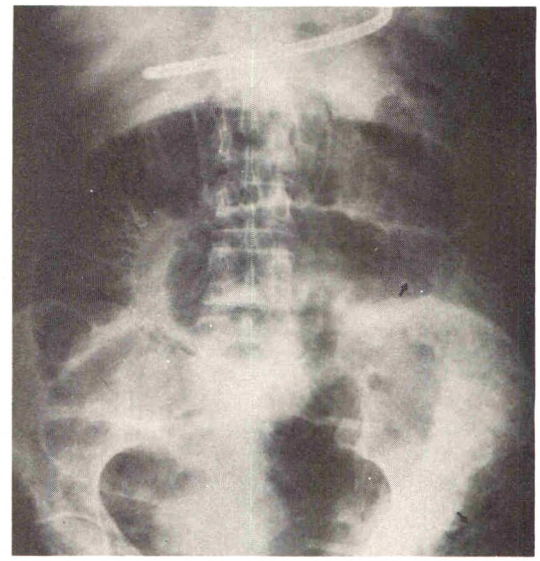

FIG. 1. Case 1. Linear streaking (arrows) represents subcutaneous emphysema in the anterior abdominal wall. There was a perforation of the small bowel secondary to obstruction. The peritoneal sutures had given away.

* Presented as a Scientific Exhibit at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.

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admitted to S.M.H. with a chief complaint of bloody diarrhea for 3 weeks and pain in the right buttock for 5 days. She had been seen by her private physician the previous day. He had palpated a large rectal mass and had biopsied it. During the night she became quite ill and noted that her right leg began to swell. On admission, she complained of increasing difficulty in using her right leg which was "now totally numb."

Physical examination on admission revealed the right thigh to be tense and twice the size of the left. Purple discoloration was present from the groin to the knee. The thigh was covered with blisters containing amber colored exudate. Her temperature was 104°, the white blood cell count was 13,900 with a shift to the left. Roentgenograms showed interstitial and subcutaneous emphysema in the right leg and presacral space (Fig. 2, *A–C*). A sigmoid colostomy and fasciotomy of the right anterior thigh were performed as emergency procedures but the patient expired 8 hours after operation.

Autopsy revealed a large tumor of the rectum which had extended into the obturator foramen and had perforated. A culture of the content of one of the blisters grew *Clostridium perfringens*.

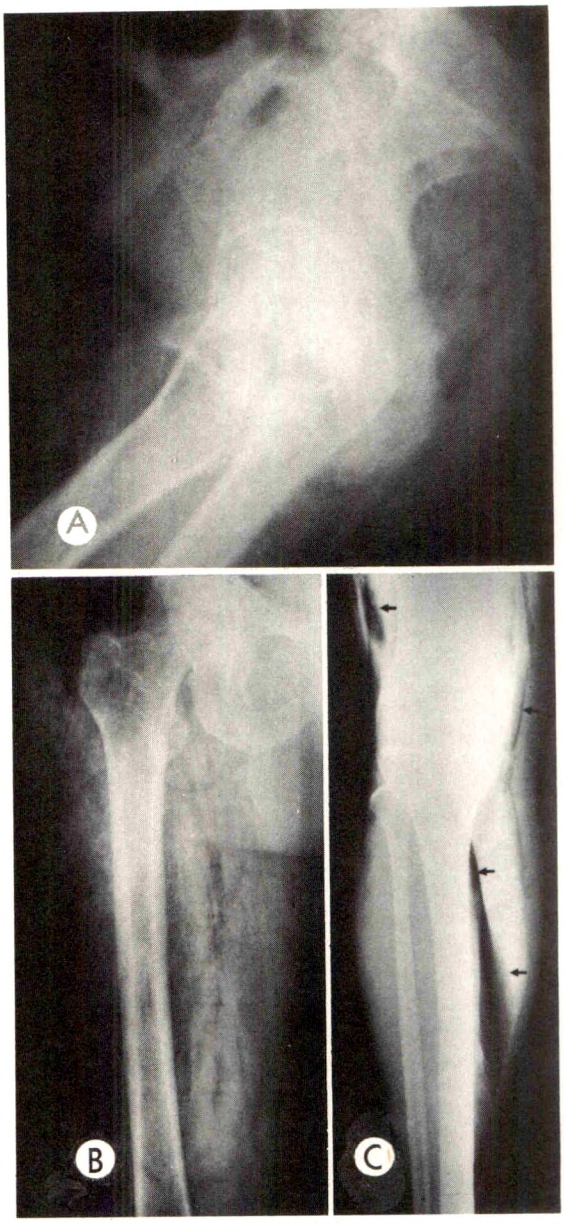

Fig. 2. Case II. (*A*) Massive subcutaneous emphysema was present in the presacral space. (*B* and *C*) Descent inferiorly is demonstrated (arrows). Autopsy revealed carcinoma of the rectum extending into the obturator foramen.

Case III. This 92 year old white female had a 2 week history of nausea and abdominal distention. On the day of admission to S.M.H. the patient noted "swelling" of the right side of her body from the neck to the hip.

Physical examination revealed extensive subcutaneous emphysema on the right side of her body. The rectal temperature was 101°. Abdominal roentgenograms revealed air in the retroperitoneal tissues and along the right lateral abdominal wall (Fig. 3, *A* and *B*). Chest roentgenograms revealed subcutaneous emphysema along the right chest wall. A barium enema examination revealed an obstructing lesion of the splenic flexure. Operation revealed an adenocarcinoma of the splenic flexure and a perforation at the hepatic flexure.

Case IV. This 64 year old Black female was admitted to G.U.H. with a 3 day history of mild right lower quadrant pain. The patient recalled mid and lower abdominal pain 2 weeks prior to admission which had subsided spontaneously.

Physical examination revealed a right lower quadrant mass which was tender and posteriorly located. The white blood cell count was 7,500 with a normal differential. Abdominal roentgenograms were thought to be compatible with a perforated viscus on the right with subcutaneous gas (Fig. 4). The diagnosis of an appendiceal abscess was made. At laparotomy, a large, partially organized, retroperitoneal abscess was found with myositis and gangrene•

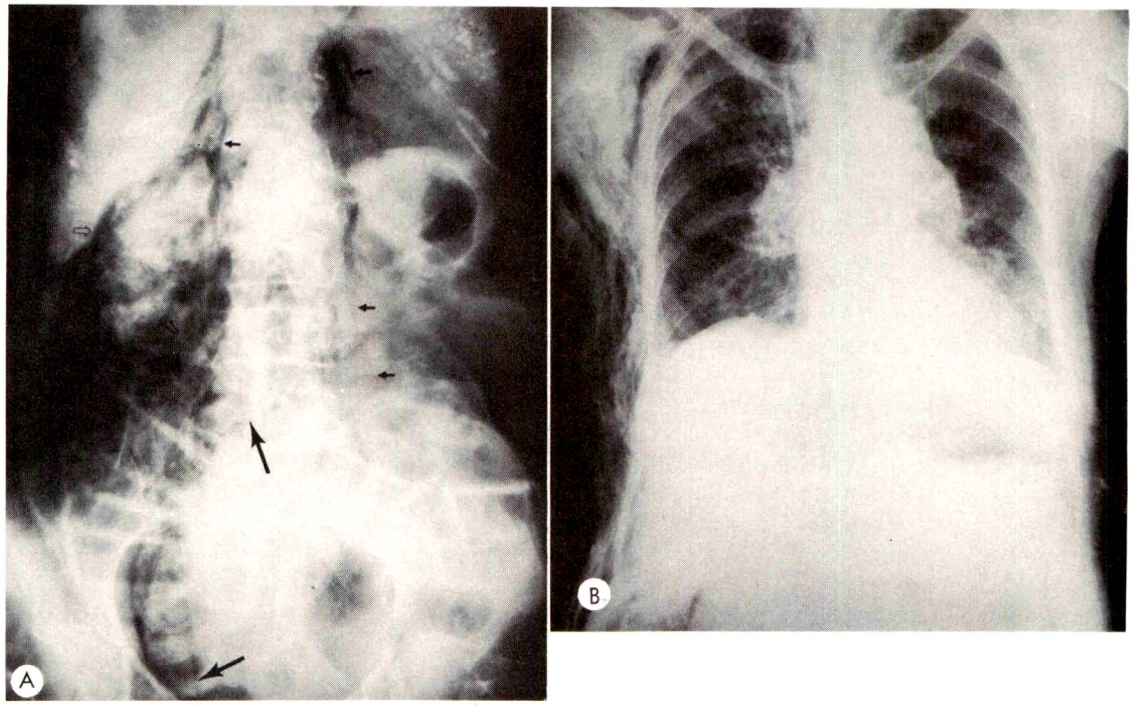

FIG. 3. Case III. There has been a perforation of the colon at the hepatic flexure secondary to an obstructing carcinoma at the splenic flexure. (*A*) Gas outlines the right kidney (open arrows) and right adrenal gland. Gaseous streaking above the psoas muscles is also seen (small closed arrows). Intra-mural gas is present in the colon (large arrows). (*B*) Subcutaneous gas in the abdominal wall dissects upwards.

of the right rectus abdominis muscle and necrosis of the subcutaneous fat.

The patient had a stormy postoperative course and expired on the sixteenth postoperative day. The autopsy showed right-sided peritonitis with adjacent panniculitis. This was thought to be secondary to a ruptured appendix. An organizing subdiaphragmatic abscess was also found. The immediate cause of death was a massive pulmonary embolus.

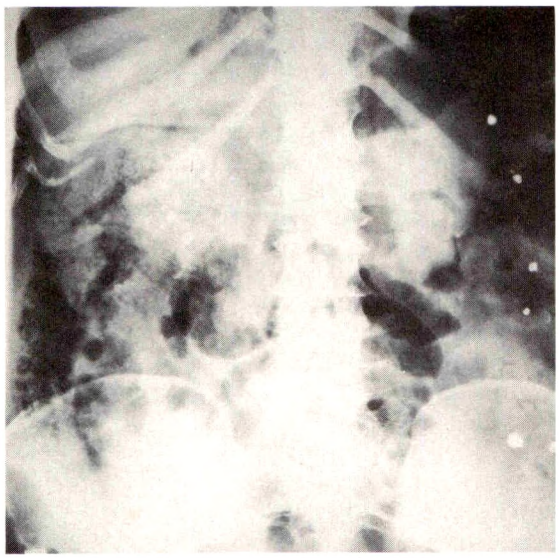

FIG. 4. Case IV. An intraperitoneal appendiceal abscess was present. This process extended anteriorly causing necrosis of the subcutaneous fat and right rectus abdominis muscle.

CASE V. This 77 year old Black female was admitted to G.U.H. with a 3 week history of intermittent abdominal pain. Physical examination revealed a left midabdominal mass. The patient was afebrile but the white blood cell count was 18,700 with a shift to the left. Abdominal roentgenograms revealed an abscess in the left abdominal wall (Fig. 5, *A* and *B*). A barium enema examination revealed evidence of diverticulitis. A diverting loop colostomy of the transverse colon was performed and the abscess in the anterior abdominal wall was drained. There was a perforation of the transversalis fascia. The patient did well postoperatively and was discharged on the fifteenth postoperative day to be readmitted at a later date for definitive surgery.

CASE VI. This 60 year old Black female was

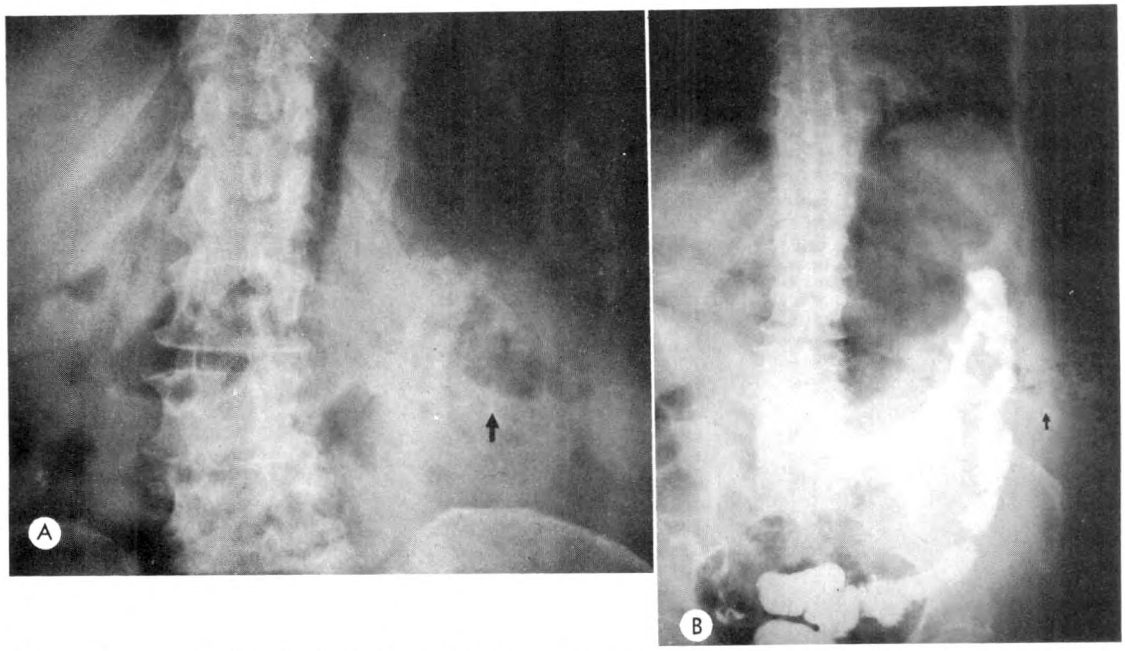

FIG. 5. Case V. (*A*) There is air in the subcutaneous tissues (arrow) representing an abscess secondary to a perforation caused by diverticulitis. (*B*) A surgically created fistula is present 10 days after surgery (arrow).

admitted to G.U.H. with a chief complaint of draining pus from the lower abdominal wall. Two weeks prior to admission she had developed left lower quadrant pain and had been treated for pyelonephritis. However, the pain continued and she noticed a "lump" in the left lower quadrant below a large umbilical hernia.

Physical examination revealed a 3×4 cm. mass in the left lower quadrant which was surrounded by a large region of erythema and edema. A large umbilical hernia was present but this was not thought to be of clinical concern. An abdominal roentgenogram showed subcutaneous air in the anterior abdominal wall (Fig. 6). The white blood cell count was 20,000 with a shift to the left.

Incision and drainage of the abscess in the abdominal wall with debridement of necrotic subcutaneous tissue was performed followed by a loop colostomy of the ascending colon.

She expired on the tenth postoperative day. At autopsy a fistula from the distal transverse colon to the anterior abdominal wall was found, thought to be secondary to diverticulitis.

CASE VII. This 89 year old white male was admitted to S.M.H. with a chief complaint of progressive abdominal distention. Physical examination revealed the abdomen to be distended and diffusely tender to rebound. The bowel sounds were absent. An incarcerated right femoral hernia was thought to be present. The white blood cell count was 8,000 with a shift to the left. Abdominal roentgenograms revealed free peritoneal air as well as extra peritoneal air (Fig. 7*A*). A pneumomediastinum with air in the left supraclavicular area and in both sides of the neck was present (Fig. 7*B*).

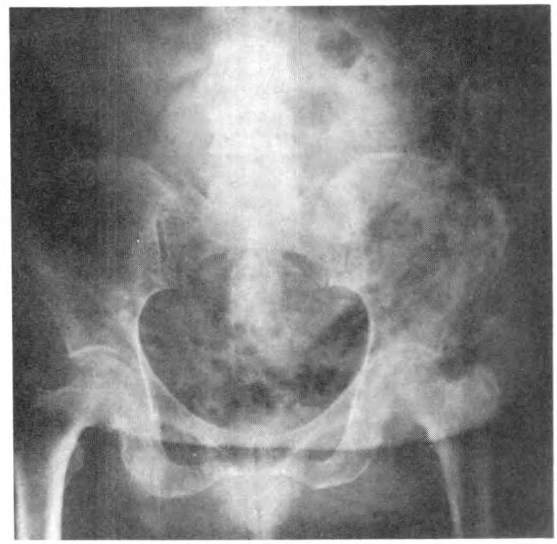

FIG. 6. Case VI. Mottled densities in the anterior abdominal wall reflect abscess formation; an umbilical hernia is also present.

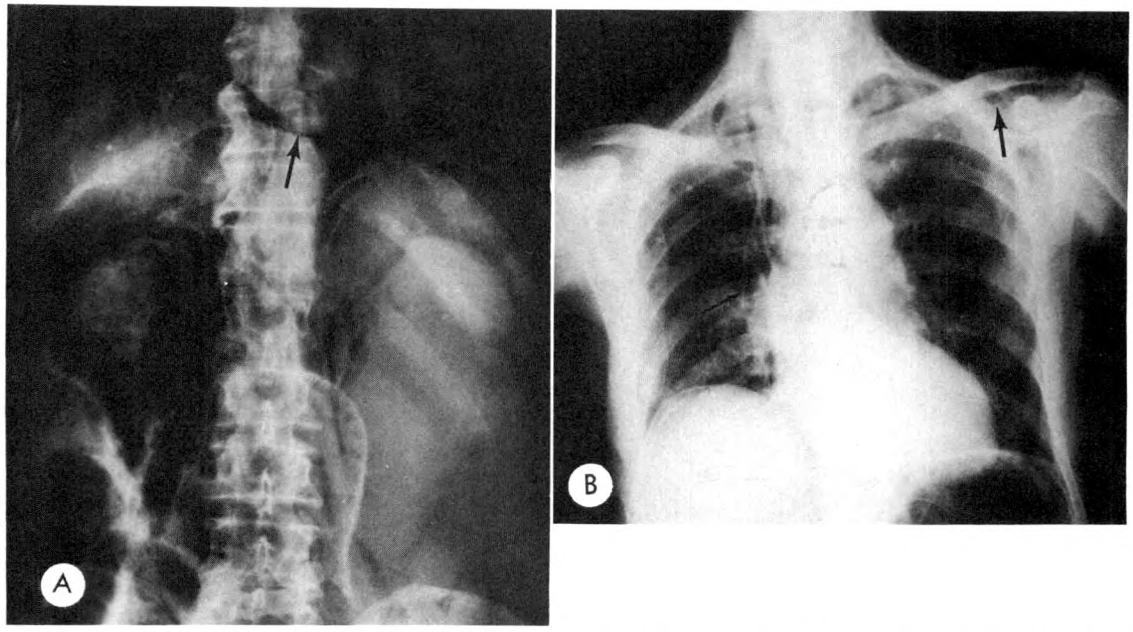

FIG. 7. Case VII. (*A*) The abdominal roentgenogram reveals free peritoneal air as well as air in the retroperitoneal structures. Gas has also dissected into the falciform ligament (arrow). (*B*) A pneumomediastinum (small arrow) is present. Air is present in both sides of the neck and the left supraclavicular region (large arrow). Free air beneath the diaphragm is also seen. Autopsy revealed a perforated duodenal diverticulum.

The patient expired shortly after admission. Autopsy revealed a right femoral hernia with obstruction of the small bowel and perforation of a gangrenous duodenal diverticulum. This diverticulum was 2.5 cm. in diameter and arose from the upper lateral aspect of the descending duodenum.

DISCUSSION

A ruptured viscus may: (1) give rise to a localized abscess or peritoneal gas and fluid; (2) cause necrosis of adjacent tissue (Case IV); (3) cause fistula formation (Cases V and VI) which may act as a safety valve mechanism (Case V); or (4) give rise to dissection of gas through soft tissue planes (Cases I, III and VII). This dissection may be intramural within the bowel, in the immediate subcutaneous tissues, or through the interstitium of muscle. Dissection of gas is frequently accompanied by retroperitoneal abscess.

Subcutaneous emphysema may occur secondary to perforation of any part of the gastrointestinal tract. The perforation may be either intraperitoneal or extraperitoneal. Rupture of an extraperitoneal structure is the more readily explained. These structures include portions of the duodenum and rectum, the posterior surfaces of the ascending and descending colon and, at times, the appendix. In cadavers, air has been injected into the posterior peritoneum and presacral space.[20] The former resulted in diffusion of the air into the anterior peritoneal area; the latter resulted in subcutaneous emphysema of the lower abdominal wall, thighs, scrotum, and buttocks. Common routes were: (1) along neurovascular bundles which penetrate the muscle and fascia of the abdominal wall; (2) through naturally occurring defects in the abdominal wall which occur at the inguinal rings; (3) inferiorly along the femoral vessels into the thigh and then upward along vessels over the inguinal ligaments; and (4) inferiorly through the pelvic floor, along the rectum into the subcutaneous tissues of the buttocks and thighs, and thence upward over the abdomen.

Gas may dissect cephalad through the

diaphragmatic hiatus producing pneumomediastinum and cervical subcutaneous emphysema.[14]

Dissection of gas secondary to intraperitoneal structures may occur through a surgical (Fig. 1) or necrotic rent in the peritoneum or through dissection into mesenteric attachments. In cases of perforation of the sigmoid or ascending colon, entrance of gas into the lower extremities is thought to occur through the iliacus and psoas muscles. The iliacus muscle has its origin from the iliac crest in the greater portion of the iliac fossa. The psoas muscles have their origin from the last thoracic and all 5 lumbar vertebrae. The iliacus and psoas muscles unite in a common tendon which passes beneath the inguinal (Poupart's) ligament and inserts on the lesser trochanter of the femur. An abscess frequently overlies the ilio-psoas muscle in cases of subcutaneous emphysema of the leg.[5,6]

It is believed that gas can dissect along mesenteric attachments. This has been postulated in cases of retroperitoneal air secondary to perforation of the sigmoid colon.[9] A more striking example of this concept has been provided by Padula[18] in explaining subcutaneous emphysema in gastric or duodenal perforation. Hydrogen peroxide was injected into the subserosal tissues of the stomach and duodenum. There was subserosal diffusion of gas from the cardia *via* the phrenico-esophageal and aortic hiatus to the posterior mediastinum and soft tissues of the neck.

There is considerable discussion in the literature concerning whether the gas in the subcutaneous tissues has its origin directly from the perforated organ or results from secondary infection. Citing pressure gradients between the intestinal lumen and peritoneal cavity, Oetting *et al.*[17] postulated that the site of perforation and the pressure gradient were of major importance with infection only an "additional factor." Certainly the speed and extent to which an organ or a side of the body swells favors a peristaltic pump of gastrointestinal origin in many cases. This is particularly true in cases of gastric and duodenal perforation.[23] However, because of the large number of gas forming organisms in the colon, infection probably contributes significantly to the spread of gas through the soft tissues in cases where this organ is perforated. In our series clinical signs of infection were present in most cases. Abscesses were confirmed by surgery in many cases. The mottled densities and linear streakings are typical of retroperitoneal abscesses and were demonstrated in Cases III and IV. Abscesses were proved present in Cases V and VI. Finally, infection by gas forming organisms in the retroperitoneal space can give rise to a full blown picture of subcutaneous emphysema without a perforated viscus.[8]

Only rough correlation can be drawn between the site of perforation and the site of first appearance of subcutaneous emphysema. Emphysema of the abdominal wall tends to be secondary to a perforation of the large or small bowel. Supraclavicular emphysema is frequent in gastroduodenal perforation. Including Case II, there have been 20 reported cases of subcutaneous emphysema following perforation in the literature.[4,12] Three cases involve the appendix, 16 cases the colon, and 1 case was secondary to a toothpick perforating the terminal ileum.

This unusual presentation appears to occur predominantly in the elderly. The average age in our series is 70 years. More subtle symptomatology in this age group apparently accounts for this insidious presentation with far advanced disease at the time of presentation. This has been particularly well documented in appendicitis.[2,7,24] In individuals over 60 years perforation of the appendix with localized or generalized peritonitis was found in 70 per cent as compared to 20 per cent in all age groups.[22]

DIFFERENTIAL DIAGNOSIS

Gastrointestinal perforation: Pneumomediastinum and dissection of air into the soft tissues of the neck are well known as roent-

genographic signs of perforation of the esophagus. Excluding this entity, Oetting and co-worker's[17] survey of the world literature revealed the most common cause of subcutaneous emphysema to be the result of perforation of the colon or appendix (55 per cent). Perforated gastric ulcer was second most common(20 per cent).

Thoracic: Subcutaneous emphysema of the chest wall is frequently seen with insertion of a chest tube. It has been reported as a frequent complication of tracheostomy.[21] Deep cervical emphysema frequently presents as an early sign of bronchial or tracheal rupture.[10]

Retroperitoneal abscess: The divisions of the retroperitoneal space have been extensively reviewed.[14] Communication between the divisions exists. Any retroperitoneal abscess containing gas forming organisms is potentially capable of causing subcutaneous emphysema. Frequent gas producing organisms include *Escherichia coli*, *Aerobacter aerogenes*, *Klebsiella pneumoniae*, *Bacteroides* and anaerobic *Streptococci*. Causes of retroperitoneal abscesses have been reviewed elsewhere.[2,16] Two cases of renal abscesses in diabetic patients have been reported to give subcutaneous emphysema in the tissues of the chest and abdominal wall.[8]

Soft tissue infection: Air in the soft tissues of the leg immediately suggests infection by *Clostridium perfringens* (gas gangrene). Infection by this organism usually occurs in a traumatized extremity with necrosis of soft tissue acting as a seat for infection. If this clinical history is lacking, other causes which we have mentioned should be reviewed. Infection in soft tissues of the leg by other gas forming organisms can cause such subcutaneous emphysema[3,11] and this is frequent in diabetics.

Postsurgical: With a rent in the peritoneum, subcutaneous emphysema can occur postsurgically without a perforated viscus.[15,19] In 1 case this was attributed to increased peritoneal pressure secondary to hiccups. This entity usually resolves spontaneously in several days.

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REFERENCES

1. Abeille, M. Perforations intestinales consecutive a une phlegmaise, peritonite generale ultime. *Gas. d. hôp*, 1853, *105*, 422–423 from Oetting.[17]
2. Altemeier, W. A., and Alexander, J. W. Retroperitoneal abscess. *A.M.A. Arch. Surg.*, 1961, *83*, 512–524.
3. Altemeier, W. A., and Culbertson, W. R. Acute non-clostridial crepitant cellulitis. *Surg., Gynec. & Obst.*, 1948, *87*, 206–212.
4. Appendiceal abscess: Clinicopathologic Conference. Edited by P. Cryer and J. Kissane. *Am. J. Med.*, 1974, *56*, 92–102.
5. Beaconsfield, P. Carcinoma of sigmoid presenting as emphysematous cellulitis of leg. *Am. Surg.*, 1953, *38*, 919–921.
6. Case records of Massachusetts General Hospital. *New England J. Med.*, 1965, *273*, 387–393.
7. Celis, R., and Cigtay, O. Massive subcutaneous emphysema from perforated appendix. *South M. J.*, 1974, *67*, 118–120.
8. Clifford, N. J., and Katz, I., Subcutaneous emphysema complicating renal infection by gas-forming coliform bacteria: report of two cases in diabetic patients. *New England J. Med.*, 1962, *266*, 437–439.
9. Culver, G., and Concannon, J. P. Retroperitoneal emphysema from perforation of intraperitoneal viscus. *Radiology*, 1950, *55*, 86–88.
10. Eijgelaar, A., and Homan, Van Der Heide, J. N. Reliable early symptom of bronchial or tracheal rupture. *Thorax*, 1970, *25*, 120–125.
11. Gillies, C. L. Interstitial emphysema in diabetes mellitus due to colon bacillus infection. *J.A.M.A.*, 1941, *117*, 2240–2242.
12. Korsten, J., Mattey, W. E., Bastidas, J., and Sicat, L. C. Subcutaneous emphysema of thigh secondary to ruptured diverticulum of ascending colon. *Radiology*, 1973, *106*, 555–556.
13. Maw, A. R. Perforation of sigmoid colon resulting in subcutaneous emphysema of head and neck and associated pneumoperitoneum. *Brit. J. Surg.*, 1968, *55*, 712–714.
14. Meyers, M. A. Radiological features of spread and localization of extraperitoneal gas and their relationship to its source. *Radiology*, 1974, *110*, 17–26.

15. Nelson, H. Free gas in peritoneum and subsequent subcutaneous emphysema. *Brit. M. J.*, 1920, *2*, 280.
16. Neuhof, H., and Arnheim, E. E. Acute retroperitoneal abscess and phlegmon. *Ann. Surg.*, 1944, *119*, 741–758.
17. Oetting, H. K., Kramer, N. E., and Branch, W. E. Subcutaneous emphysema of gastrointestinal origin. *Am. J. Med.*, 1955, *19*, 872–886.
18. Padula, J. Zur Frage des subkutanen Emphysema bei perforierten gastroduodenalen Geschwüren. *Zentralbl. Chir.*, 1926, *53*, 2839–2841.
19. Shambaugh, P., and Burgert, P. H. Subcutaneous emphysema in cancer of colon. *A.M.A. Arch. Surg.*, 1962, *85*, 344–347.
20. Stahlgren, L. H., and Thabit, G. Subcutaneous emphysema: important sign of intra-abdominal abscess. *Ann. Surg.*, 1961, *153*, 126–133.
21. Stowe, D. G., Kenan, P. D., and Hudson, W. R. Complications of tracheostomy. *Am. Surg.*, 1970, *36*, 34–38.
22. Thorbjarnarson, B., and Loehr, W. J. Acute appendicitis in patients over age of sixty. *Surg., Gynec. & Obst.*, 1967, *125*, 1277.
23. von Heimburg, R. L., Alexander, S. T., Sauer, W. G., and Bernatz, P. E. Subcutaneous emphysema from perforated gastric ulcer: case report and review of literature. *Ann. Surg.*, 1963, *158*, 1–5.
24. Williams, J. S., and Hale, H. W. Acute appendicitis in elderly: review of 83 cases. *Ann. Surg.*, 1965, *162*, 208–212.

A FEASIBILITY STUDY FOR PERCUTANEOUS RETROPERITONEAL LYMPH NODE BIOPSY*

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ABSTRACT:

A percutaneous technique for the biopsy of retroperitoneal lymph nodes was studied in animals and cadavers. Access to the retroperitoneal space from the groin to the diaphragm has been achieved, and satisfactory biopsies of iliac and lumbar lymph nodes opacified by pedal lymphangiography have been obtained. Further investigations, however, are needed to improve the technique and to minimize the potential hazards associated with this procedure.

EVALUATION of the iliac and lumbar lymph nodes is essential in determining the extent of involvement of certain neoplastic diseases. Laparotomy and lymph node biopsy in conjunction with lymphangiography are employed in pretreatment staging.[1,4]

A percutaneous method for retroperitoneal lymph node biopsy has considerable potential as long as the technique is readily accomplished without undue morbidity. Percutaneous lymph node biopsies have been performed in the mediastinum by Nordenstrom[2,3] and in the pelvis by Ruttimann.[5] A percutaneous approach for biopsy of pelvic and para-aortic lymph nodes is presented.

MATERIAL AND METHOD

Bilateral pedal lymphangiography was performed in the usual manner in 15 anesthetized dogs and in 3 human cadavers. In dogs a few large lymph nodes are opacified at the level of the aortic bifurcation but few, if any, in the lumbar area.

Lymphangiography in the human cadavers was undertaken as soon after death as possible since clotting of the lymph would impede the progress of injection of the contrast material. The technique was that employed in living patients except for the use of a larger volume of contrast material. Intradermal injection in the dorsum of the feet of a particulate dye (Patent Blue V, Alphazurine 2G, etc.) allowed visualization of the subcutaneous lymphatics. Dissection and cannulation of the lymphatics and injection of approximately 20 cc. of Ethiodol into each lower extremity opacified the inguinal, iliac, and lumbar lymph nodes and the thoracic duct.

The retroperitoneal lymph nodes were still opacified in another cadaver with adenocarcinoma of the endocervix who had been examined by lymphangiography 1 month ante mortem and was found to have extensive retroperitoneal lymph node metastases.

TECHNIQUE FOR BIOPSY

(Fig. 1, *A–E*)

A small incision, 1 cm. in length, was made in the skin at the crease in the groin over the femoral artery. A tract was created with a tapered, slightly curved, Teflon sheathed, metallic introducer dissecting under the inguinal ligament along the femoral and iliac vessels into the pelvis. The position of the tract in the perivascular space and therefore in the retroperitoneal space was verified under fluoroscopic control by a side-to-side motion of the introducer which displaced the lymphatics and lymph nodes which accompany the iliac artery and vein.

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Supported in part by the George Alfred Cook Memorial Fund.

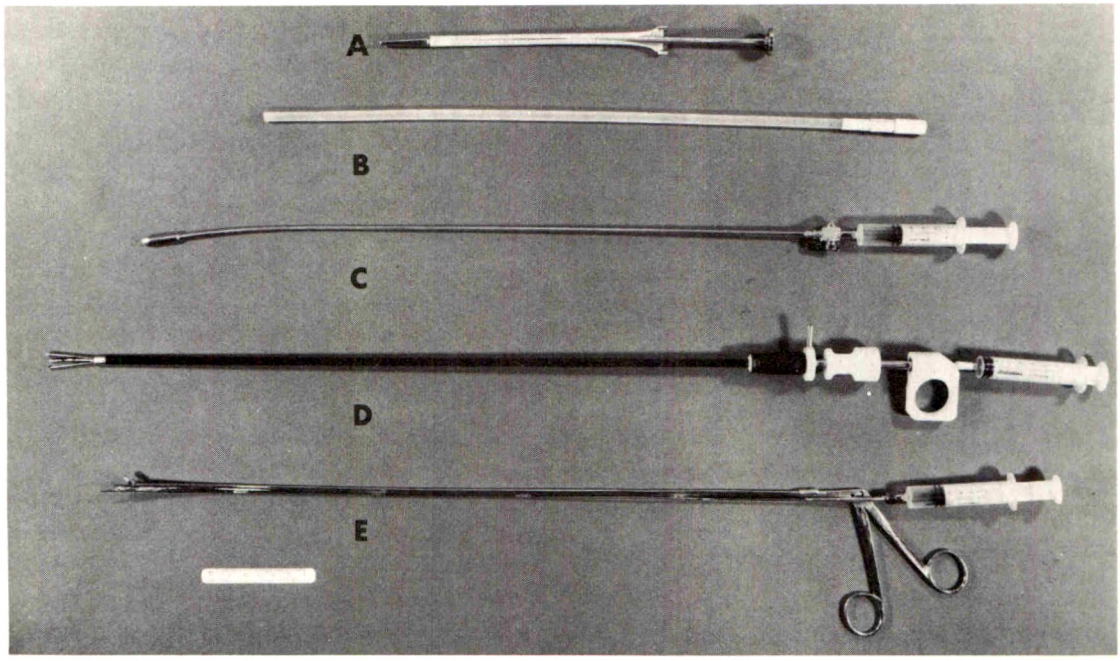

Fig. 1. Instrumentation for percutaneous retroperitoneal biopsies. (*A*) Teflon sheath metallic introducer. (*B*) Teflon tubing which is passed through the Teflon sheath once the introducer is removed. (*C*) Probe with bullet-shaped tip which fits into the Teflon tubing. This probe-tubing combination is passed through the Teflon sheath when the metallic introducer is removed. (*D*) Biopsy forceps (modified fallopian tube transection forceps). (*E*) Biopsy forceps (modified bronchoscopic forceps).

The metallic part of the introducer was removed, leaving the Teflon sheath in place to function as a tunnel into the retroperitoneal space. Through this tunnel a Teflon sheathed gently curved probe equipped with a bullet shaped tip and central lumen was introduced into the perivascular space. Once the proper position of the probe was confirmed, the retroperitoneal space was insufflated with gas (air, CO_2, or N_2O) injected through the lumen of the probe which dissected the tissues. Under fluoroscopic guidance, the probe was advanced gently through the ipsilateral retroperitoneal space up to the diaphragm.

The probe was then removed from the sheath and replaced with a biopsy forceps. A variety of forceps has been utilized including a gastroscopic biopsy forceps, a bronchoscopic biopsy forceps, and a forceps usually utilized for fallopian tube transection. The latter 2 biopsy forceps have been modified by the addition of a 19 gauge retractable needle for aspiration (Fig. 2, *A* and *B*). The needle, which was positioned below or between the jaws of the forceps, was advanced and used for aspiration immediately before the jaws were closed for

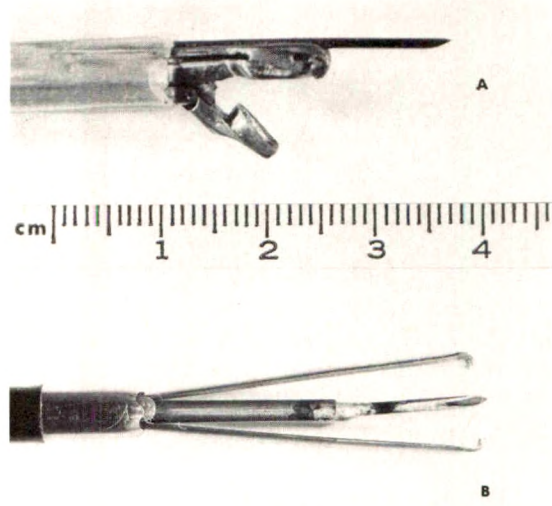

Fig. 2. (*A*) Biopsy tip of forceps (Fig. 1*E*) with aspiration needle. (*B*) Biopsy tip of forceps (Fig. 1*D*) with central aspiration needle.

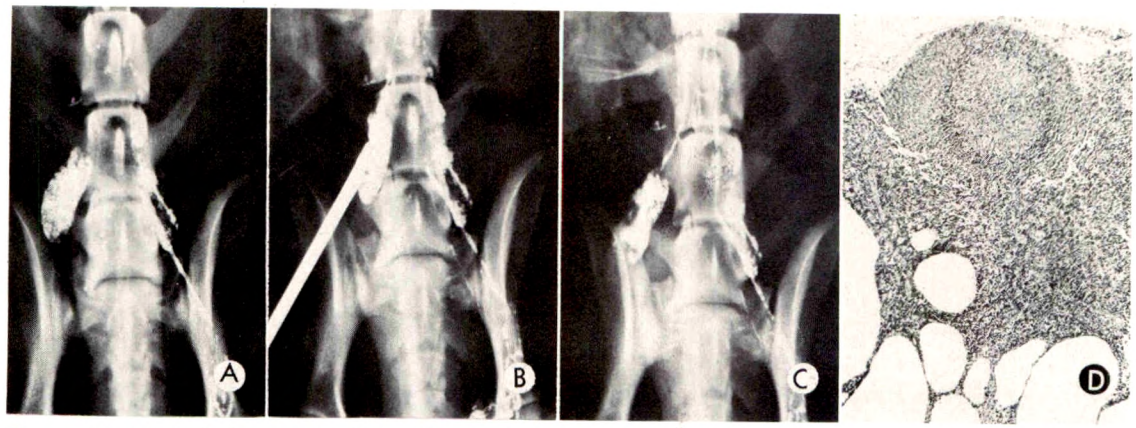

FIG. 3. Biopsy technique—experimental animal (dog). (*A*) Opacified lymph nodes, postlymphangiogram. (*B*) Forceps in the process of retroperitoneal lymph node biopsy. (*C*) Post-biopsy changes in lymph node. (*D*) Lymph node biopsy specimen. The spaces are created by lymphangiography (×50).

procurement of the biopsy. This permits a check for the presence of a vascular structure between the jaws thereby reducing the likelihood of trauma to vessels by the biopsy. Aspiration biopsy could also be accomplished with this needle arrangement. The bronchoscopic biopsy forceps was also modified for greater maneuverability by curving the distal end approximately 15 degrees.

Under fluoroscopic guidance the forceps was brought in contact with the opacified lymph node. The mouth of the forceps was opened and used as a dissecting instrument to free the lymph node of the surrounding tissue. The lymph node was moved in various directions to insure nodal contact. Lateral motion usually displaced the lymph node away from the adjacent blood vessels. The lymph node to be biopsied was then positioned between the jaws of the forceps and the needle advanced for aspiration. In the absence of aspirated blood the jaws were closed around the needle resulting in biopsy (Fig. 3, *A–D*). The instrument was then withdrawn to retrieve the tissue leaving the Teflon sheath in place. If additional biopsy material was required the forceps was reinserted.

The animals were sacrificed at varying intervals, from immediately after to 11 weeks following the biopsy. The biopsy specimens were evaluated histologically and the site of the biopsy was examined grossly for local trauma. The cadavers were autopsied after biopsy to determine the position of the probe and the extent of damage.

RESULTS

In the 8 animals sacrificed immediately after biopsy the probe or forceps inserted in the inguinal area was found in the retroperitoneal space. Perforation of the posterior peritoneum was seen in 6 animals. This perforation usually occurred at the level of the renal pedicle. Perforation of major pelvic vessels occurred in 3 animals, 2 involving arteries, 1 involving veins. Perforation of major vessels was immediately evident as blood appeared in the Teflon sheath. In the other animals small hematomas of 1 to 2 cc. in size were seen at the sites of biopsy.

In the 7 animals sacrificed at varying intervals from a few days to 11 weeks following biopsy, no significant abnormalities were found. Ecchymoses were noted at the sites of biopsy. No obvious retroperitoneal hemorrhage was seen.

Postmortem examinations of the 4 human cadavers revealed the probe and forceps in the retroperitoneal space. No significant injuries were seen which could be attributed to the biopsy procedure (Fig. 4, *A–C*; and 5, *A–C*).

The size of the biopsy specimens varied up to 5 mm. in diameter and was quite

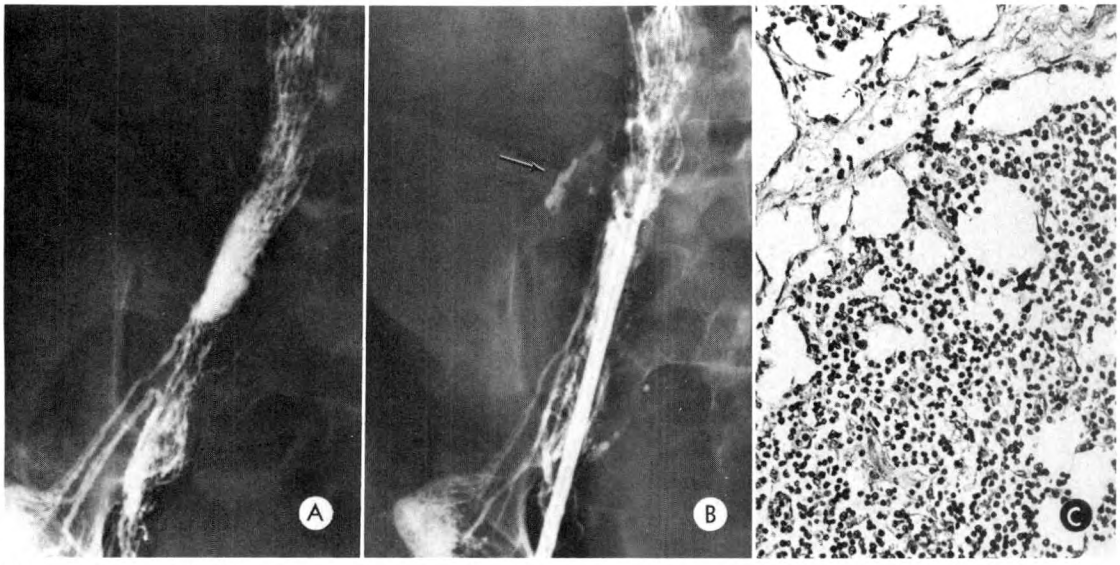

Fig. 4. Biopsy technique—postmortem lymphangiography. (*A*) Lymphatics and lymph nodes opacified by postmortem lymphangiography. (*B*) Lymph node biopsy of a common iliac lymph node. Arrow shows extravasation of the contrast material during biopsy. (*C*) Biopsy specimen depicting changes due to postmortem lymphangiography. Note the absence of lipogranulomas (×90).

adequate for histologic examinations (Fig. 3*D*; 4*C*; and 5*C*). One biopsy contained a portion of a nerve. Lymph node tissue was secured in all subjects.

DISCUSSION

A thorough assessment of the extent of neoplastic disease is prerequisite to the intelligent management of any patient with cancer. Any technique which contributes to the accuracy of this evaluation is justifiable as long as the results warrant the risks and expenses.

Selective lymphadenectomy in conjunction with lymphangiography for staging prior to therapy has found increasing application. At present at M. D. Anderson Hospital pretreatment lymphadenectomy is performed in the management of patients with Hodgkin's disease and with carcinoma of the cervix. Lymphangiography is also important in the pretreatment staging of patients with carcinomas of the testicle, of the bladder, and of the prostate. If a percutaneous approach to the retroperitoneal and pelvic lymph nodes could be accomplished with a minimum of complications it would enable, by a simpler technique, confirmation of the extent of tumor spread, thereby obviating the need for exploratory laparotomy. The described technique would also reduce the time from histologic diagnosis to the initiation of therapy. As in all other lymph node biopsy procedures, including exploratory laparotomy, not all lymph nodes will be within the grasp of the biopsy forceps. Of course, a negative biopsy does not exclude the presence of disease.

This percutaneous approach involves the passage of blunt instruments and a biopsy forceps through the retroperitoneal space. The potential complications include: (1) inadvertent biopsy of non-nodal tissue, *i.e.*, blood vessels, nerves, ureters and muscle; (2) penetration or perforation of viscera and the peritoneum; (3) infections; and (4) seeding of neoplastic cells. Trauma to blood vessels, nerves, and the ureter is by far the most serious of the possible complications. Of the more than 100 specimens obtained, 2 biopsies were taken of arteries and 1 of a vein. Subsequently the biopsy forceps was modified with a retractable needle for aspiration.

The viscera in the retroperitoneal region in all the animals and in 3 human cadavers

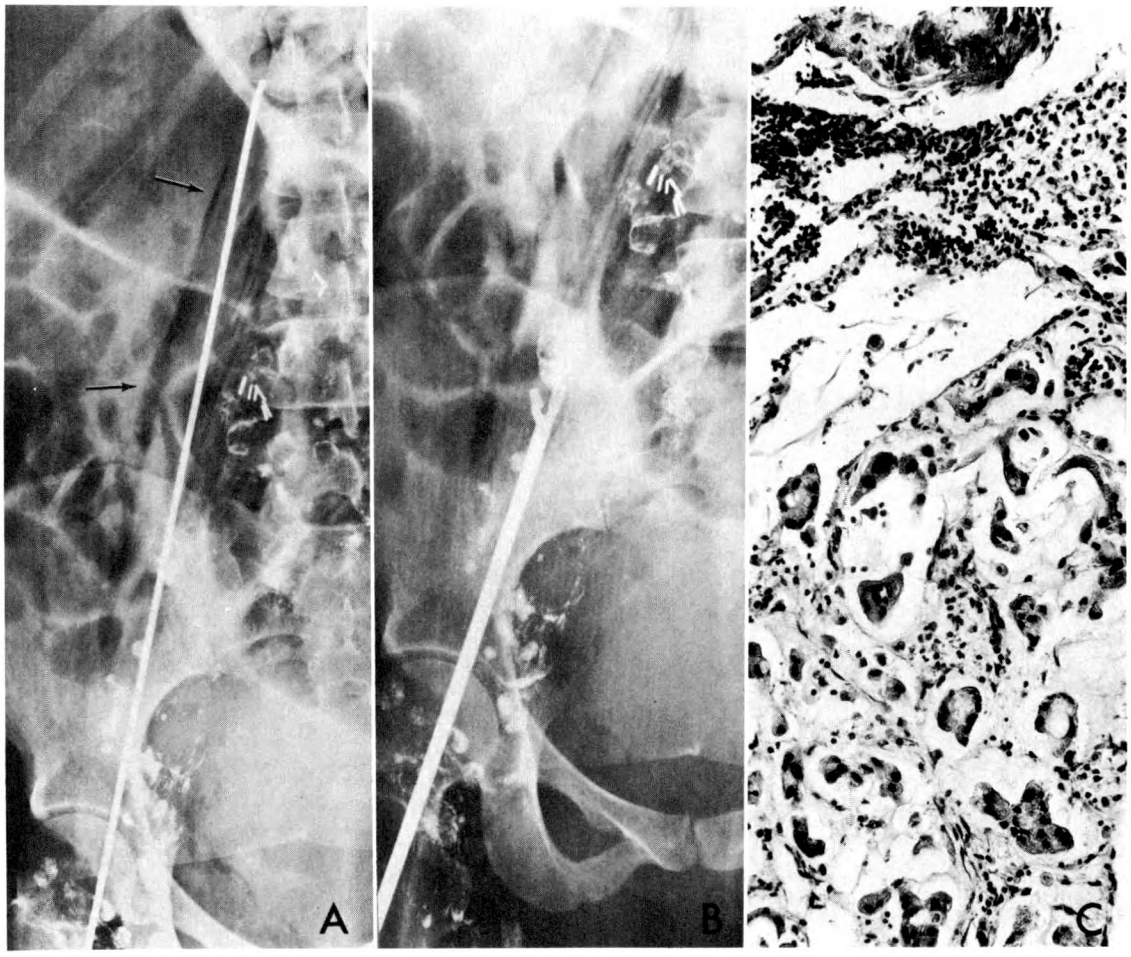

FIG. 5. Biopsy technique—postmortem in a patient with adenocarcinoma of the endocervix examined during life by lymphangiography. (*A*) A probe placed in retroperitoneal space. Note the air dissecting the retroperitoneal space (arrows). (*B*) Biopsy of retroperitoneal lymph node. (*C*) Biopsy specimen—metastatic adenocarcinoma (×90).

studied were freely movable and not bound down by infiltrating neoplasm or chronic inflammation; such fixation at times does occur. In the cadaver of the patient with adenocarcinoma of the endocervix, exploratory laparotomy and lymph node biopsy had been performed prior to the percutaneous procedure. At autopsy hydronephrosis was also present. No obvious damage was inflicted by the percutaneous approach. In an effort to minimize trauma to the urinary system, pyelography would be advisable at the time of biopsy.

Perforation of the peritoneum was seen in 6 animals but not in the cadavers. In humans the retroperitoneal space is potentially large and allows relatively easy access up to the diaphragm. Perforation of the peritoneum is of little significance, for the peritoneum is traversed in transabdominal retroperitoneal lymph node biopsy.

One other potential complication is the seeding of malignant cells along the biopsy tract. This risk is present with all percutaneous biopsy procedures.

It is possible that percutaneous retroperitoneal biopsy may be accomplished with preoperative analgesia and local anesthesia in the groin at the site of entry. The retroperitoneal space is supposedly free of neural supply. If necessary, general anesthesia may be employed.

Further refinement of this technique is obviously necessary. Biplane fluoroscopy would allow better localization of the opacified lymph nodes and more security in the contact of the lymph node with the biopsy forceps. Greater maneuverability of the probe and the biopsy forceps is desirable to gain easier access to more lymph nodes. Considerable improvement is needed in the design of the biopsy forceps. The aspiration needle projecting between the jaws of the forceps has added safety and is currently being used in the hope of avoiding vascular trauma. A more secure grasp of the lymph node to be biopsied would be of considerable assistance. In addition, retroperitoneal endoscopy during biopsy is being explored employing a fiberscope. Thus far we find that the retroperitoneum is readily visible but landmarks are difficult to identify.

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We wish to express our appreciation to Miss Irene Polanksy, R.T. (ARRT), Mrs. Raquel Collins, B.S., and Miss Cheryl Lawrence, B.S., for their technical assistance. We are also indebted to Mr. Eugene Szwarc, R.T. (ARRT), of the Medical Communications Department for his photographic assistance.

REFERENCES

1. Glatstein, E., Guernsey, J. M., Rosenberg, S. A., and Kaplan, H. S. Laparotomy and splenectomy in Hodgkin's disease. *Cancer*, 1969, *24*, 709–718.
2. Nordenstrom, B. Transjugular approach to mediastinum for mediastinography and mediastinal needle biopsy: preliminary report. *Invest. Radiol.*, 1967, *2*, 134–140.
3. Nordenstrom, B. Paraxiphoid approach to mediastinum for mediastinography and mediastinal needle biopsy: preliminary report. *Invest. Radiol.*, 1967, *2*, 141–146.
4. Piver, S. M., and Barlow, J. J. Paraaortic lymphadenectomy in staging patients with advanced local cervical cancer. *Obst. & Gynec.*, 1974, *43*, 544–548.
5. Ruttimann, A. Iliac lymph node aspiration biopsy through paravascular approach: preliminary report. *Radiology*, 1968, *90*, 150–151.

ELECTRONIC ANAL SPHINCTER STIMULATION IN BARIUM ENEMA EXAMINATION*

A PRELIMINARY COMMUNICATION

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ABSTRACT:

Electronic stimulation of the external anal sphincter muscle was successful in preventing premature evacuation of barium in 28 of 30 patients. All of them were unable on 1 or more occasions to retain the barium during previously attempted studies by conventional methods. None of the patients had an underlying neurologic problem affecting the anal sphincter.

No adverse reactions were noted.

IN prior and unrelated studies of patients who had fecal incontinence, a clearly visible contraction of the anal musculature was observed during electronic stimulation of the external anal sphincter muscle. We thought a similar technique might be effective in the prevention of barium incontinence during colon examination in those patients who had previously failed to retain the contrast material using conventional catheters with or without air-insufflated balloons.

MATERIAL AND METHOD

An hour-glass shaped plug was equipped with a pair of electrodes. A tubular channel through the plug was fitted at the external end with a nozzle permitting a convenient connection to the barium container (Fig. 1). A transverse bar prevented further penetration of the plug into the rectum as well as excessive movement of the plug during contraction of the musculature. Proper alignment of the plug within the anal canal was secured by 3 straps centered around the plug and attached to the body by means of adhesive: 1 in the sacral area and 1 in each groin. Thus a firm positioning of the plug in the anal canal was secured regardless of the patient's body movements or the maneuvers necessary to perform the barium enema examination. The electrodes were connected to a battery-operated stimulator generating a rectangular, bipolar current with an impulse duration of 1 millisecond at a constant frequency of 300 Hz.

The voltage may be regulated from 0 to 12.5 volts by means of a potentiometer. The patient is placed in the left decubitus position and the plug is inserted into the anal canal after applying a small amount of K-Y jelly.

After the plug is secured by means of straps, the contraction response is checked

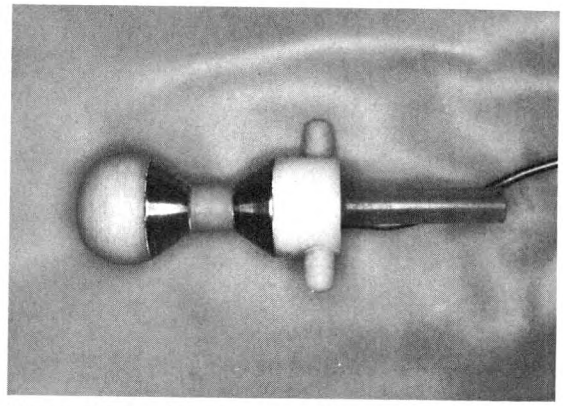

FIG. 1. Presently used plug with horizontal limiting bar at the distal end. Circular arrangement of electrodes.

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by slowly increasing the voltage. In the majority of cases 7 volts suffice to cause a strong and visible contraction of the external sphincter muscle without pain. At times voltage had to be raised to 12.5 before a contraction was visible.

Pain in the anal area was the only factor limiting the amount of voltage. A tingling sensation in the anal area was perceived by practically all patients. When the patient's maximal contraction threshold is established the plug is connected to the barium container and a routine examination of the colon is started.

Leaks occurring during the examination were recorded with reference to the amount of the barium injected as well as the time at which they became evident. Each patient had an anoscopic examination 24 hours after the procedure in order to observe any local reaction which could be attributed to the electronic stimulation.

Cardiac rate and blood pressure were checked during the procedure. Patients on cardiac pacemakers were monitored with electrocardiography during the entire procedure.

Inability to fill the entire colon or premature evacuation was regarded as a failure. Thirty patients were examined. Their ages ranged from 34 to 86 years with a median of 67.3 years. All patients had failed on previous attempts to retain the barium enema using a balloon-tipped catheter. None of the patients had a neurologic problem affecting the sphincteric system.

RESULTS

In 28 patients visualization of the entire colon was achieved. A diagnosis of a significant abnormality was made in 4 cases. Minor leaks occurred in 13 patients, otherwise successfully examined. These leaks, usually in the amount of 100–200 ml. of contrast material, did not impair the quality of the examination. Most of these leaks occurred at the end of the procedure. In 15 cases not a single drop of barium was lost during the entire examination. Two patients failed to retain the barium. The leak occurred after visualization of the splenic flexure and was caused by a spasm of the descending and sigmoid colon.

Follow-up anoscopic examination did not show any reaction which could be attributed to the electronic stimulation. None of the patients had any signs of irritation or burns of the mucous membrane lining the anal canal. Two patients had a cardiac pacemaker, 1 external and 1 internal. Continuous electrocardiographic monitoring during the procedure did not show any abnormalities in cardiac rhythm. None of the patients showed any significant change in cardiac rate or blood pressure.

DISCUSSION

One of the remarkable features of the external anal sphincter muscle is its contractibility when in a state of apparent flabbiness and lack of tonus. Full advantage of this phenomenon was taken by Caldwell[1] who was able to restore fecal continence by means of electronic stimulation applied by implanted electrodes; and later by Hopkinson[6] and Fischer and von der Mosel[4] who produced the stimulation by means of an inserted anal plug. The strong contraction observed during the anal stimulation led to the speculation that a similar technique might be used effectively in the barium enema examination of those patients who for various reasons were unable to retain the contrast material.

Vodovnik *et al.*[8] showed that currents with frequencies higher than 100 Hz were well tolerated. Pain was diminished to absent with a frequency higher than 500 Hz. Since pain in the anal canal was the only side effect when the voltage was increased above a certain limit (7 volts in this study), a frequency of 300 Hz with an impulse duration of 1 millisecond was selected. Harvey and Robertson[5] employed from 9 to 14 volts without untoward reaction, and Clark[2] used a fixed voltage of 10. In 15 patients examined in this study the maximal voltage did not exceed 7 volts since experience had shown that higher voltages caused pain. One possible explanation for this dis-

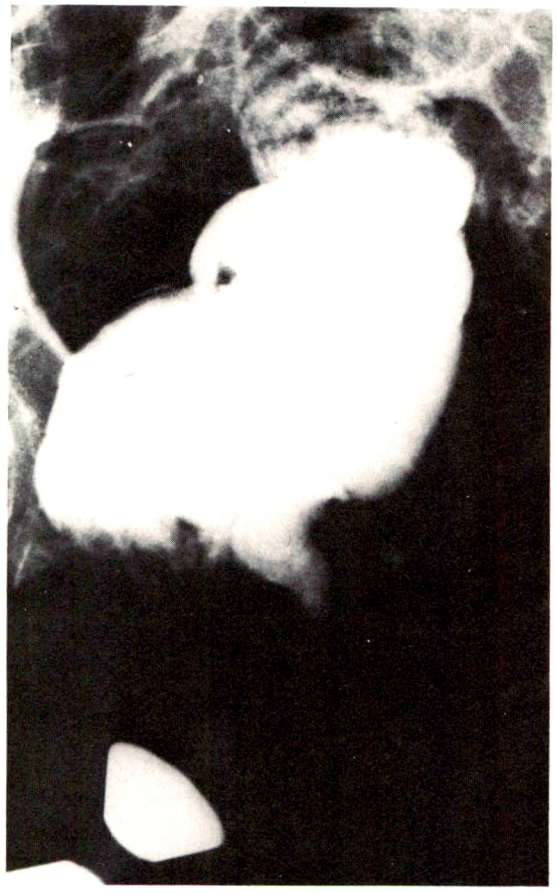

FIG. 2. Initial stage of barium enema examination. Marked contraction extends beyond the anal canal; none of the contrast medium is in the ano-rectal junction.

crepancy was the different frequency of the stimulating current—20 and 50 Hz used by Clark[2] and Harvey and Robertson,[5] respectively, compared to 300 Hz used in this study. Another possible explanation lies in the power output of the stimulators when under load. These authors do not report the voltage measured during actual stimulation, *i.e.*, when the circuit was closed and under load. Observations during our study clearly indicate that the stimulation does not involve the anal sphincter muscle alone: rather, the entire pelvic floor seemed to be affected, possibly including the pubo-rectal muscle. Only this could explain the tremendous holding power of the anal musculature, thus preventing a leak or an expulsion of the plug despite maximal distention of the rectum with barium.

Figure 2 illustrates the initial phase of barium enema injection. The degree of spasm is sufficient to produce the gap in the barium column at the ano-rectal junction. Figure 3 shows the late stage of barium enema examination when the rectum is fully distended, yet the electronic plug is firmly in place. No leak is visible.

In 13 patients minor leaks occurred at the end-stage of the examination, probably due to sphincter muscle fatigue. The external anal sphincter muscle consists chiefly of T-units with a frequency of 3–4 Herz when in normal tonic state lasting for indefinite periods.[7] The sphincter is subject to fatigue if forced to a smooth tetanic contraction lasting longer than 10–15 minutes. Temporary relaxation invariably follows.

The majority of patients was in the older

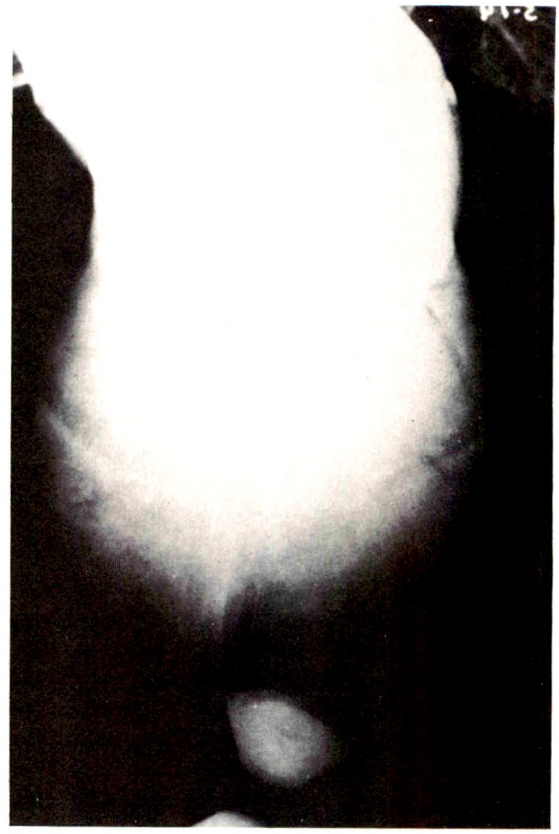

FIG. 3. End stage of barium enema examination. Note firm grip around the electronic plug with no leak despite maximal distention of the rectum.

age group. Only 5 patients were younger than 50 years. Using Ferrucci's classification,[3] it can be assumed that those patients presented a functional inability to retain the barium enema, since none of them had a known neurologic problem affecting the sphincter. It is also interesting to note that many of the patients were extremely emaciated and weak. The procedure was well tolerated by all patients.

Clinical studies using currents of various frequencies and impulse duration are in progress in order to delineate the optimal range combining the patient's comfort and effective strength of the tetanic contraction.

Electro-Catheter Corporation from Rahway, New Jersey supplied the generator as well as the electronic plugs.

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REFERENCES

1. Caldwell, K. P. S. Treatment of incontinence by electronic implants. *A. Roy. Coll. Surg.*, 1968, *41*, 447–459.
2. Clark, K. Barium enema examination using electronic technique: preliminary report. *Brit. J. Radiol.*, 1971, *44*, 970–972.
3. Ferrucci, J. T., Jr. Hypotonic barium enema examination. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 304–308.
4. Fischer, B. H., and von der Mosel, H. A. Electronic management of fecal incontinence. *J.A.M.A.*, 1969, *207*, 1897–1898.
5. Harvey, A. J., and Robertson, A. Use of anal stimulator for barium enema examinations. *Austr. Radiology*, 1968, *12*, 393–394.
6. Hopkinson, B. R., and Lightwood, R. Electrical treatment of incontinence. *Brit. J. Surg.*, 1971, *54*, 802.
7. Tokizama, T., and Shimazu, H. Functional Differentiation of Human Skeletal Muscle. Charles C Thomas, Publisher, Springfield, Ill., 1964.
8. Vodovnik, L., Long, L., Regenos, E., and Lippay, A. Pain response to different tetanizing currents. *Arch. Phys. Med.*, 1965, *46*, 187.

NON-CONTRACTILE MOVEMENT OF TANTALUM POWDER IN THE CANINE RECTUM*

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ABSTRACT:

Caudad movement of tantalum particles in the rectum was observed in 5 recumbent anesthetized beagle bitches in the absence of muscular contractions as monitored by intrarectal balloons. The movement did not occur after injections of atropine or glucagon alone but did occur when a bisacodyl suppository was introduced in addition to the injection of glucagon. Along with direct observations of rectal mucosa, this strongly suggests the role of mucus as a carrier of the particles.

COLONIC motility has been studied for many decades with fluoroscopy using both open-tip and balloon catheters,[2,6–10] cineradiography,[4,11,13] and telemetric capsules.[11] Several types of movement have been described in the colon.[1,2] The most important appear to be segmental contractions, which cause mixing and churning of fecal material, and mass movement, which results from decrease in segmental resistance before segmental contractions and can involve as much as one-third of the colon.[8] Other types of movement are peristaltic waves, which occur less frequently than segmental contractions and are probably less important;[11] haustral shuttling;[12] and type I rapid-frequency waves, which occur infrequently, last only about 5 seconds, and develop relatively low pressure.[14]

To our knowledge, there has been no documentation of movement of material along the colon wall without detectable muscular contractions. This report describes caudad movement of tantalum powder insufflated into the rectums of recumbent anesthetized dogs in the absence of pressure changes in the rectum as monitored by balloons.

MATERIAL AND METHOD

In 5 spayed female beagle dogs, the rectums were marked with tantalum wires placed in rows at measured distances into the serosa during laparotomy. The dogs were anesthetized with sodium pentobarbital (30 mg. per kg.) and placed on their sides. Initially, pressure measurements to monitor possible contractions were obtained with perfused open-tip catheters, but these were abandoned because of the possibility of partial plugging of the lumen by fecal material despite perfusion. The measurements reported here were obtained with balloons filled with 20 ml. of 50 per cent Hypaque, one placed in the proximal and the other in the distal rectum of each dog.

Studies were performed immediately after intramuscular administration of: (1) 0.2 mg. of atropine, which is known to inhibit motility and secretion of mucus; (2) 0.5 mg. of glucagon, which also inhibits motility and secretion of mucus; (3) 0.5 mg. of glucagon immediately followed by introduction of a bisacodyl suppository, which stimulates mucus secretion; and (4) no additional medication. For most studies, frontal and lateral roentgenograms were obtained at 5 to 30 minute intervals over a period of 1 to 4 hours. For others, only lateral views were taken, as these were more sensitive to changes in contour. The distances between tantalum particles and wires were measured only during periods when there were no contractions (Fig. 1, *A–C*; and 2). The films

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on which measurements were made were superimposed to ascertain that there was no change in contour of the bowel and wires. Movement of the particles was expressed in cm. per hour by adjusting the actual time to 1 hour.

RESULTS

No movement of tantalum particles was discerned after injection of atropine (Fig. 3, *A* and *B*), or glucagon (Fig. 4, *A* and *B*). When a bisacodyl suppository was introduced at the beginning of a glucagon study, particles moved (Fig. 5, *A* and *B*). The particles moved the greatest distance when the dogs were under anesthesia without additional medication (Fig. 6, *A* and *B*; and Table 1).

Movement of the tantalum particles in various areas of the rectum was not uniform: in all dogs particles moved well in the proximal rectum but in the distal rectums of 2 dogs particles appeared inspissated and conglomerated and did not move at all. By the end of most of the glucagon-plus-suppository studies, when the rectal mucosa looked completely dry as the balloons were removed, the roentgenograms showed no movement of tantalum powder, although large quantities of mucus were observed in the rectum immediately after administration of the bisacodyl suppository.

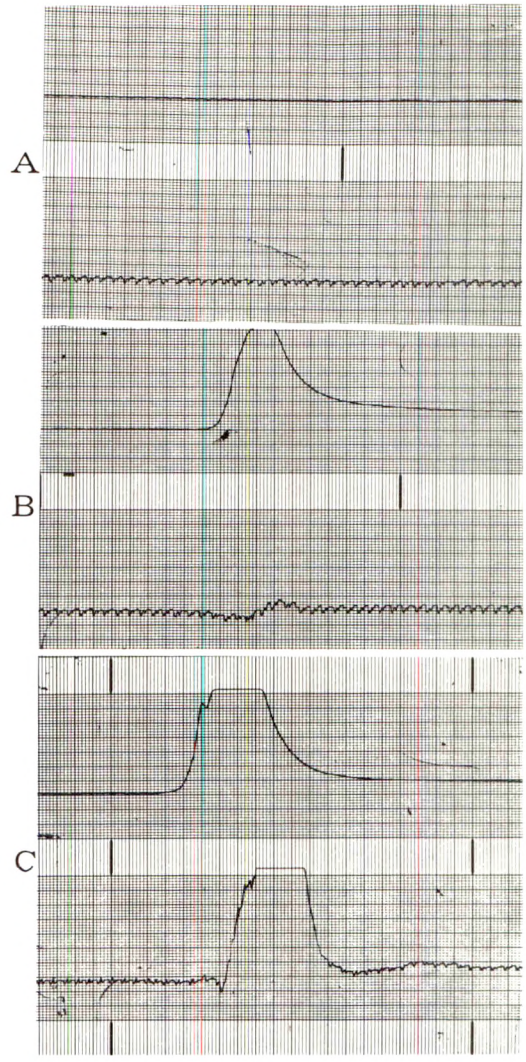

FIG. 1. Pressure recordings from the 2 intrarectal balloons filled with 20 ml. of 50 per cent Hypaque in 3 different studies: (*A*) No evidence of change of pressure. Only breathing motion is recorded. (*B*) Moderate changes of pressure. (*C*) Major changes of pressure. Measurements of particles were made only when there were no changes of pressure (*A*). The shape of the balloons varied with position in the rectum.

DISCUSSION

The mechanism of this non-contractile caudad movement of tantalum particles in the canine rectum is unknown. One possibility is that contractions of the muscularis mucosae move the particles. This is unlikely, however, because the movement described here occurred in the absence of Type I waves, which are believed to be due to contractions of the muscularis mucosae.[14] Beating of the microvilli,[15] which contain Actin, might be the true mechanism pushing the particles towards the anus. This mechanism might be similar to the orad movement of particles in the bronchial tree,[3,5] which is due to ciliary action moving a stratified mucus blanket, although coughing also plays a role. In our anesthetized dogs, there was no movement of tantalum particles unless mucus was secreted in the rectum. Mucus secretion was verified by observing the mucosa when the balloons were removed. This suggests that mucus carries the particles toward the anus. It is not clear, however, why mucus flowed only

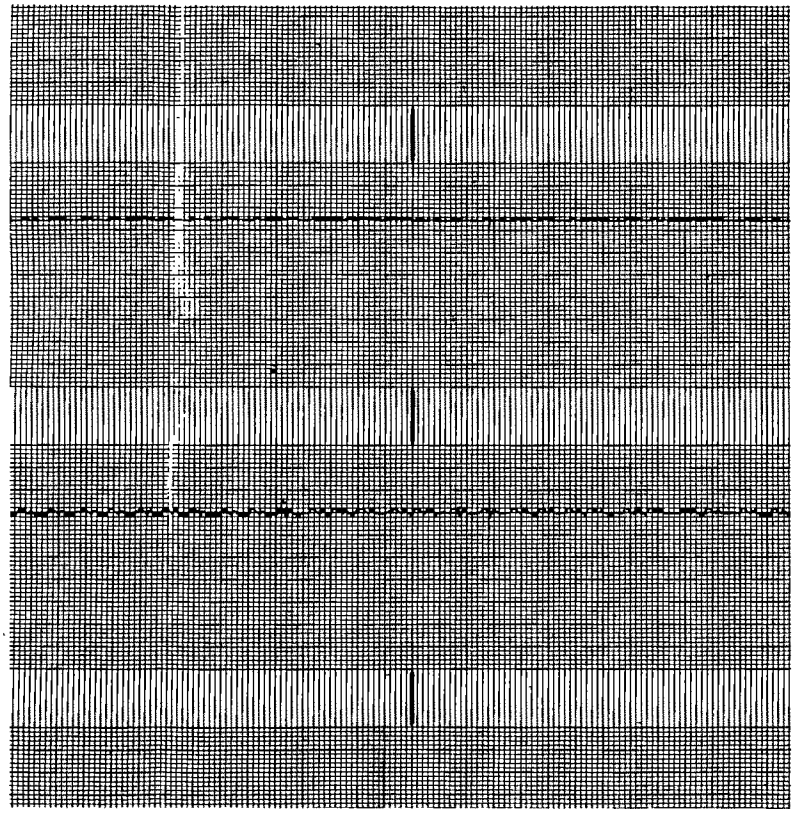

FIG. 2. Pressure recordings of upper and lower balloons showing minimal contractions of short duration. No measurements of particles were made even during these minimal changes of pressure.

in an anal direction when the effect of gravity was eliminated.

It is unknown whether this type of movement observed in beagle dogs also occurs in humans. If it does, it may be an important physiologic mechanism for movement of the fecal contents in the resting state and may account for fecal movement in diseases where there are no colonic muscular contractions.

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TABLE I

NON-CONTRACTILE MOVEMENT OF TANTALUM PARTICLES IN COLONS OF ANESTHETIZED DOGS WITH AND WITHOUT ADDITIONAL MEDICATION

Experimental Conditions	Number of Studies	Range of Particle Movement (cm. per hour)	Average Particle Movement (cm. per hour)
Atropine (0.2 mg.)	6	0–0.6*	0
Glucagon (0.5 mg.)	5	0	0
Glucagon (0.5 mg.) plus bisacodyl suppository	6	0.4–5.2†	1.8
No additional drugs	5	0.3–8.0	2.8

* Movement observed in 1 dog only.

† Movement ended after first 30 to 45 minutes in all studies, except in one, where movement was seen between 60 and 90 minutes.

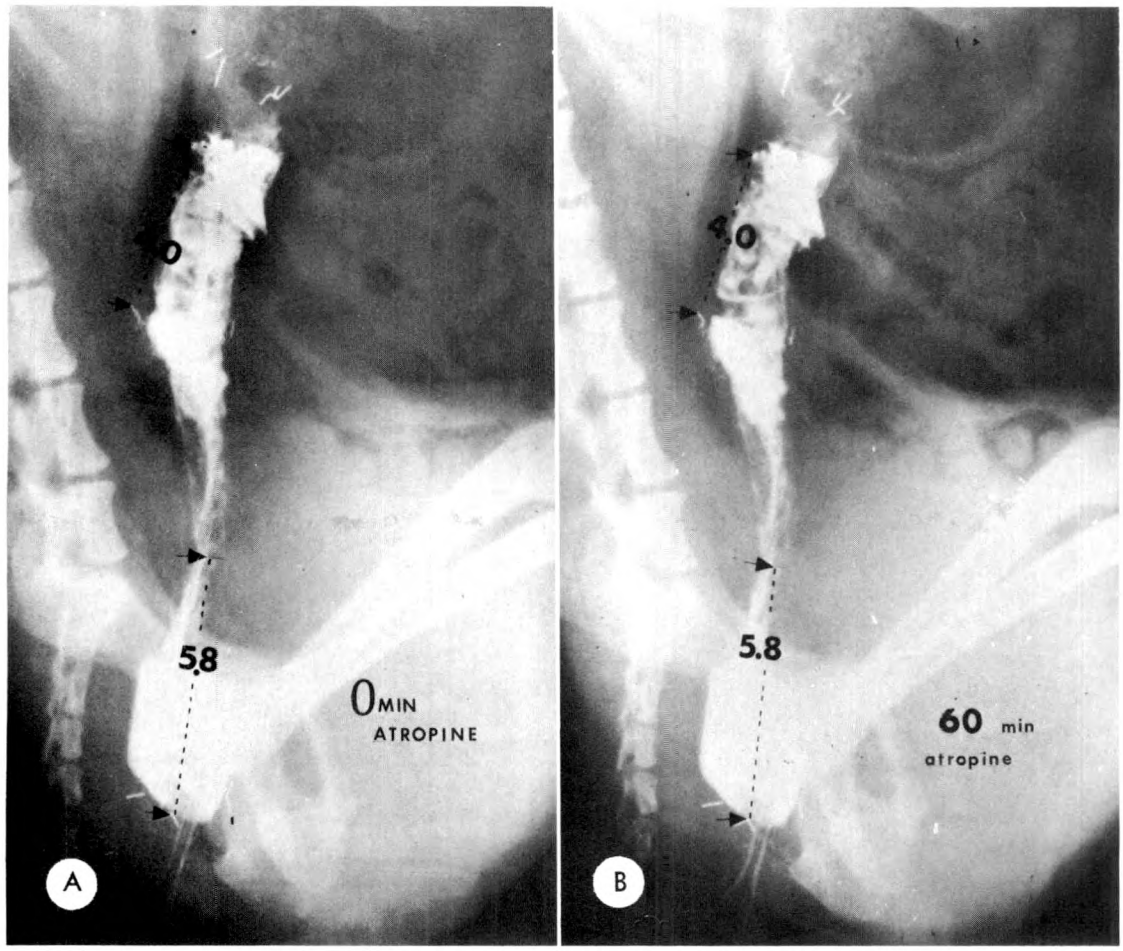
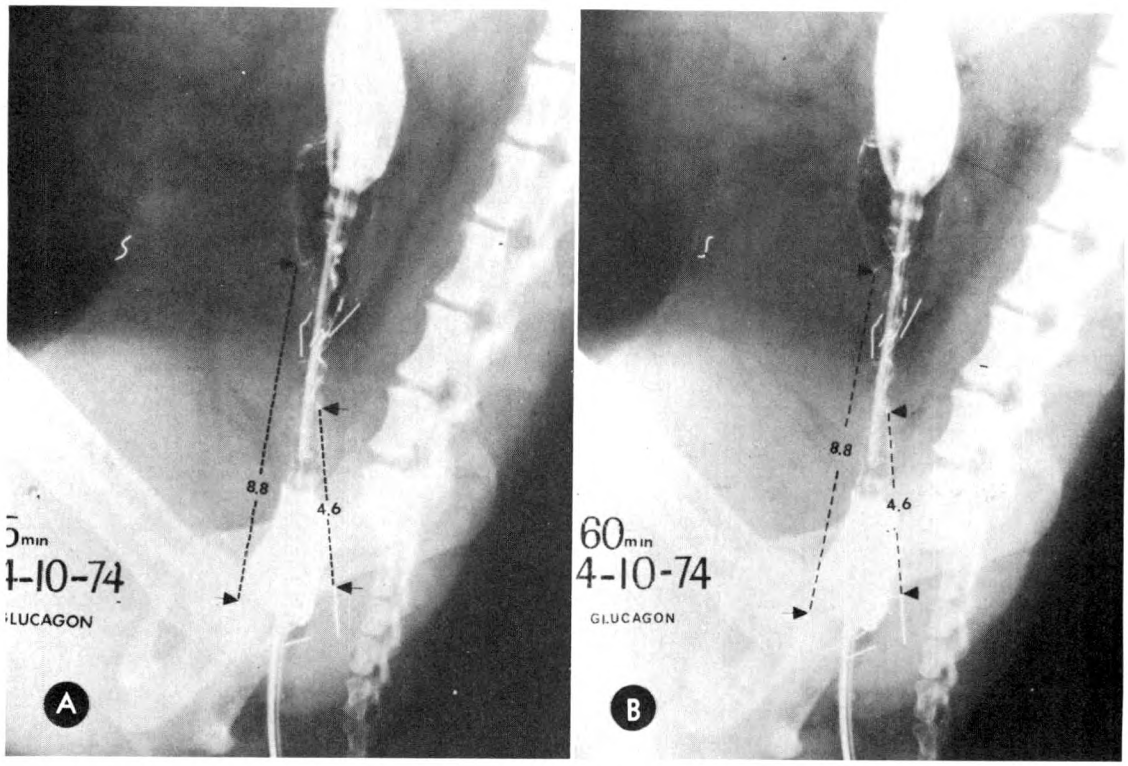


FIG. 3. Rectal roentgenograms in an anesthetized dog at (*A*) 0 and (*B*) 60 minutes after intramuscular injection of 0.2 mg. of atropine. Top arrow at each of two levels indicates tantalum particle; bottom arrow indicates tantalum marker. No movement of tantalum particles was observed.

The authors gratefully acknowledge the assistance and advice of Drs. Rudi Schmid, Henry Goldberg, and Lawrence Way, Mr. Antonio Brito, and Ms. Mimi Zeiger. This work was supported by National Institutes of Health Grant AM 15937 from the National Institute of Arthritis and Metabolic Diseases.

REFERENCES

1. CONNELL, A. M. Motor action of large bowel. In: Handbook of Physiology. Volume 4: Motility. Section 6: Alimentary Canal. Edited by C. F. Code, and W. Heidel. American Physiological Society, Washingron, D. C., 1968, pp. 2075–2091.
2. DAVENPORT, H. W. Movements of colon. In: Physiology of the Digestive Tract: An Introductory Text. Third edition. Year Book Medical Publishers, Inc., Chicago, 1971, pp. 70–78.
3. EDMUNDS, L. H., JR., GRAF, P. D., SAGEL, S. S., and GREENSPAN, R. H. Radiographic observations of clearance of tantalum and barium sulfate particles from airways. *Invest. Radiol.*, 1970, *5*, 131–141.
4. GRAMIAK, R., ROSS, P., and OLMSTEAD, W. W. Normal motor activity of human colon: combined radiotelemetric manometry and slow-frame cineroentgenography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 301–309.
5. GROSS, P., and DETREVILLE, R. T. P. Lung as embattled domain against inanimate pollutants: précis of mechanisms. *Am. Rev. Resp. Dis.*, 1972, *106*, 684–691.
6. HARDCASTLE, J. D., and MANN, C. V. Physical factors in stimulation of colonic peristalsis. *Gut*, 1970, *11*, 41–46.


8.8
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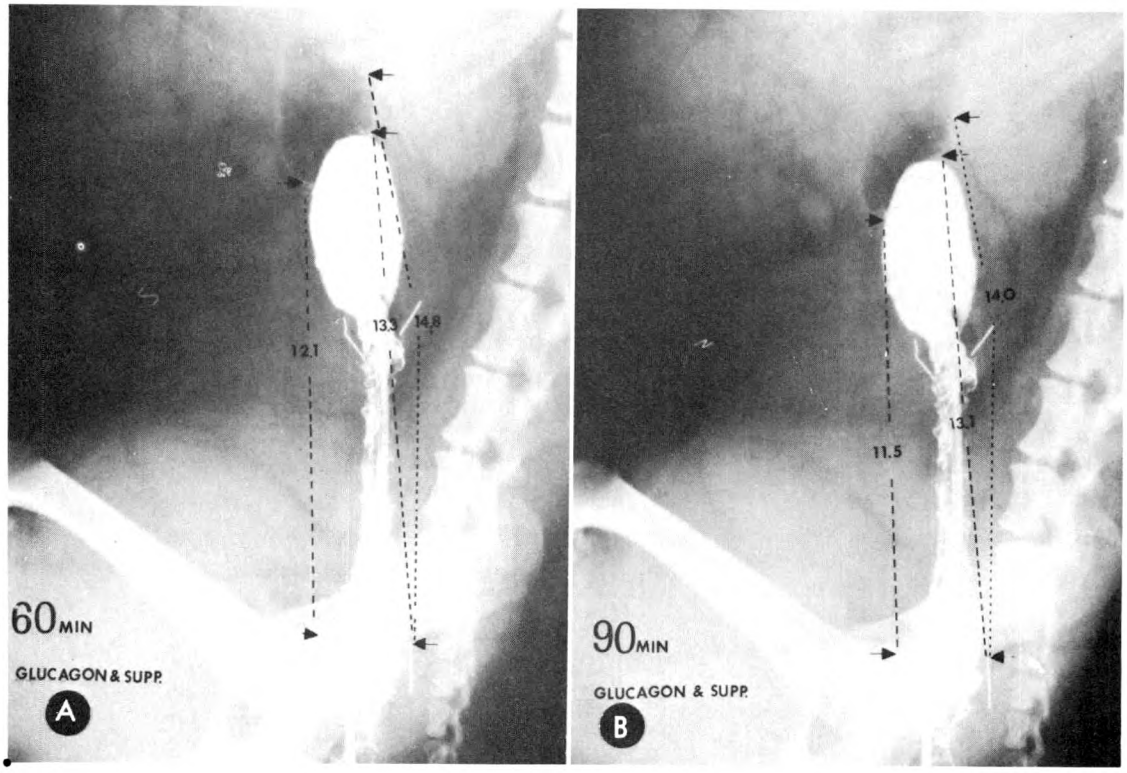
12.1
13.3
14.8
60 MIN
GLUCAGON & SUPP.
A
14.0
13.1
11.5
90MIN
GLUCAGON & SUPP.
B

FIG. 4. Rectal roentgenograms in an anesthetized dog at (*A*) 5 and (*B*) 60 minutes after intramuscular injection of 0.5 mg. of glucagon. Top arrows indicate tantalum particles; bottom arrows indicate tantalum markers. No movement of tantalum particles was observed.

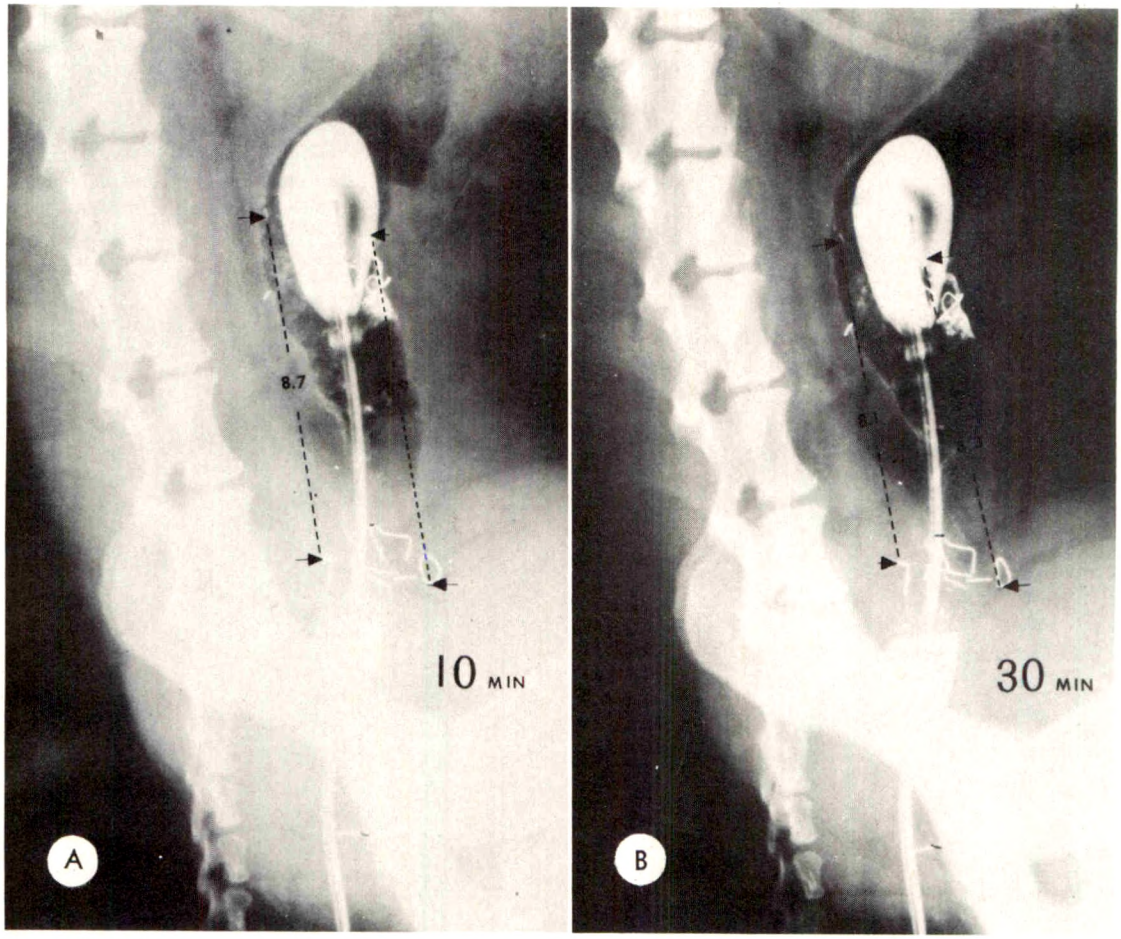

FIG. 6. Rectal roentgenograms showing movement of tantalum particles in the interval between (*A*) 10 and (*B*) 30 minutes in an anesthetized dog with no additional medication. Top arrows indicate tantalum particles; bottom arrows indicate tantalum markers. Both particles moved 0.6 cm. in 20 minutes, or 1.8 cm. per hour.

FIG. 5. Rectal roentgenograms in an anesthetized dog at (*A*) 60 and (*B*) 90 minutes after intramuscular injection of 0.5 mg. of glucagon followed immediately by introduction of a bisacodyl suppository into the rectum. Top arrows indicate tantalum particles; bottom arrows indicate tantalum markers. Significant movement of particles was observed during this interval: 0.6, 0.2, and 0.8 cm. in 30 minutes, or 1.2, 0.4, and 1.6 cm. per hour. In later roentgenograms, no movement of tantalum particles was observed.

7. HOLDSTOCK, D. J., MISIEWICZ, J. J., SMITH, T., and ROWLANDS, E. N. Propulsion (mass movements) in human colon and its relationship to meals and somatic activity. *Gut*, 1970, *11*, 91–99.
8. HOLZKNECHT, G. Die normale peristaltik des kolons. *München med. Wchnschr.*, 1909, *56*, 2401–2403.
9. Proceedings, Fourth International Symposium on Gastrointestinal Motility, Banff, Alberta, Canada, September 6–8, 1973. Edited by E. E. Daniel, K. Bowes, J. A. L. Gilbert, B. Schofield, K. T. Schnitka, and G. Scott. Mitchell Press, Vancouver, 1973.
10. RITCHIE, J. A. Colonic motor activity and bowel function. Part I. Normal movement of contents. *Gut*, 1968, *9*, 442–456.
11. RITCHIE, J. A., ARDRAN, G. M., and TRUELOVE, S. C. Motor activity of sigmoid colon of humans: combined study by intraluminal pressure recording and cineradiography. *Gastroenterology*, 1962, *43*, 642–668.
12. RITCHIE, J. A., ARDRAN, G. M., and TRUELOVE, S. C. Haustral movement in human colon. *Gut*, 1969, *10*, 1048. (Abst.).
13. RITCHIE, J. A., TRUELOVE, S. C., and ARDRAN, G. M. Propulsion and retropulsion in human colon demonstrated by time-lapse cinefluorography. *Gut*, 1968, *9*, 735–736. (Abst.).
14. SLEISENGER, M. H. Physiology of colon. In: Gastrointestinal Disease: Pathophysiology, Diagnosis, Management. Edited by M. H. Sleisenger, and J. S. Fordtran. W. B. Saunders Company, Philadelphia, 1973, p. 234.
15. TONER, P. G., CARR, K. E., and WYBURN, G. M. The Digestive System: An Ultrastructural Atlas and Review. Appleton-Century-Crofts, New York, 1971, pp. 107–118.

VOL. 125, NO. 1

A NEW CONTRAST MEDIUM FOR CHOLANGIO-CHOLECYSTOGRAPHY: MEGLUMINE IODOXAMATE*

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ABSTRACT:

A preliminary study of 18 patients following a single bolus intravenous injection of 10 to 30 cc. of meglumine iodoxamate (Cholovue), a new contrast medium for cholangio-cholecystography, showed no significant adverse clinical or laboratory effects with 10 and 20 cc., and only transient, minor changes with 30 cc. Early and persistent visualization of the subsegmental and major bile ducts and the gall-bladder was obtained. Good to excellent opacification was obtained in all cases with doses ranging between 0.11 cc./kg. (19.7 mg. iodine) to 0.59 cc./kg. (108 mg. iodine). Meglumine iodoxamate appears to be a highly effective agent for cholangio-cholecystography.

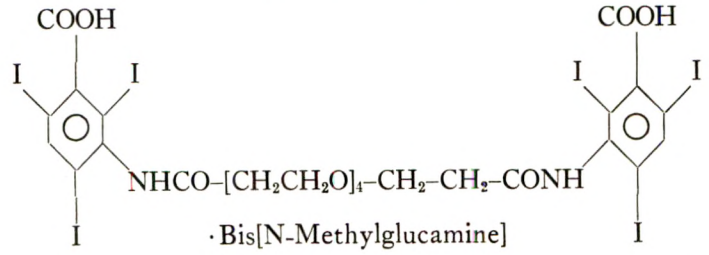
MEGLUMINE iodoxamate (Cholovue‖) is a new contrast medium for intravenous cholangio-cholecystography. The compound is an original development of the Research Laboratories of Bracco Industria Chimica of Milan, Italy and the Squibb Institute for Medical Research.

Meglumine iodoxamate is characterized by a high molecular weight of 1,678.3 and a molecular iodine content of 45.5 per cent. The chemical composition is as follows:

With alkaline metals and hydroxyalkylamines, the substance forms salts that are highly soluble in water, permitting the preparation of aqueous solutions with a high iodine content and with relatively low osmotic pressure.

Preclinical evaluations of meglumine iodoxamate in mice, rats and dogs show it to possess lower acute toxicity and better biliary excretion than meglumine iodipamide (Cholografin); thus, better tolerance

Meglumine Iodoxamate



3, 3′-[(1, 16-Dioxo-4, 7, 10, 13-tetraoxahexadecane-1, 16-diyl)-diimino] bis [2, 4, 6-triiodobenzoic acid], bis[N-methylglucamine]salt

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‖ Squibb Institute for Medical Research, Princeton, N.J.

and biliary tract visualization are possible in humans.

The purpose of the authors is to present their preliminary experience with 18 patients.

MATERIAL AND METHOD

The subjects selected had no prior history of cardiovascular, hepatic, hematopoietic, renal, or metabolic disorders. All subjects with a history of asthma, allergy, or any hypertensive experience were excluded from the study. No subject with a disease of the thyroid was allowed to participate. Specifically excluded from the study were subjects who had been treated with any contrast agents in the preceding 4 weeks before the examination. The subjects were not permitted to consume any barbiturates or chlorpromazine for 1 month, or salicylates for 1 week prior to the study.

Included in the 18 subjects studied were 16 asymptomatic, normal volunteers and 2 patients who had failure of visualization of the gallbladder following a double-dose oral cholecystogram. There were 9 females and 9 males between the ages of 18 and 47 years, with an average of 27.9 years. The body weights varied between 31.8 kg. and 92.7 kg., with an average of 68.6 kg. The heights ranged from 133 cm. to 191 cm., with an average of 167 cm. The body surface areas ranged from 1.41 m^2 to 2.21 m^2, with an average of 1.7 m^2.

No bowel preparation was used, but a low residue diet was given to each subject the day before the examination. The subjects were allowed to take nothing by mouth, with the exception of water, for approximately 8 hours prior to the examination.

A 40 per cent w/v aqueous solution of meglumine iodoxamate containing 183 mg. of organically bound iodine per ml. was used for the intravenous bolus injection.

The 18 subjects studied were divided into 3 sub-groups: 6 of the subjects received 10 cc. (1.83 gm. iodine); 6 received 20 cc. (3.66 gm. iodine); and 6 received 30 cc. (5.50 gm. iodine), according to a randomized allocation.

Each subject was given a test dose of 1 cc. of the contrast agent prior to the actual study. After waiting 5 minutes to see that no adverse reaction occurred, the total dose required for the examination was injected intravenously. A narrow-bore hypodermic needle was used to ensure a slow rate of injection of the single bolus, and the entire dose was injected steadily over a full 10 minute period.

Physical examinations were performed before the injection and were repeated at 2, 24 and 120 hours after injection of the contrast agent. Pulse, blood pressure, respiratory rate, temperature, and an electrocardiogram were obtained just prior to the administration of the contrast agent. The electrocardiogram was monitored during the entire procedure and repeated 24 and 120 hours after the procedure. Vital signs were again obtained at the conclusion of the injection and at 30 minutes, 1 hour, 2 hours, 4 hours, and 24 hours after the contrast agent administration. All patients were observed throughout the examination for any untoward reactions, such as a feeling of warmth, flushing, nausea, and skin rashes.

The following laboratory tests were performed just before the examination and at 4, 8, 24, 48, and 120 hours after the intravenous injection of the contrast agent: complete blood cell count, including differential and platelet counts; serum uric acid; serum urea nitrogen; serum glutamic-oxalic transaminase; serum glutamic-pyruvic transaminase; serum alkaline phosphatase; direct serum bilirubin; total serum bilirubin; serum cholesterol; serum calcium; serum phosphorus; total serum protein; serum albumin; serum glucose; serum lactic dehydrogenase; serum creatinine; serum creatine phosphokinase; serum cholinesterase; red blood cell cholinesterase and glucose-6 phosphate dehydrogenase (pre-injection only); serum sodium; serum potassium; serum chloride; and serum carbon dioxide. Serum creatinine clearance was also measured before and after the procedure. A complete urinalysis was also performed at these times.

Roentgenographic examinations were performed with the subject recumbent, the left side elevated, with the central beam in the anteroposterior projection, coned over the liver. Serial exposures were made at 0 time (at the end of the 10 minute injection period), and at 10, 20, 30, 40, 60, 90, 120, 150, 180, and 240 minutes after the completion of the injection. No tomography was used to enhance the visualization of the biliary tract.

Opacification of the biliary tract on the roentgenograms was evaluated on a scale of 0 to 4 (grading the bile ducts and gallbladder separately) as follows: 0—no visualization; 1—poor (faint visualization); 2—fair (visualization diagnostic but less than optimal quality); 3—good (visualization equal to previous acceptable clinical experience); and 4—excellent visualization (superior to previous clinical experience).

RESULTS

Table 1 demonstrates the dose related to body weight of the 18 subjects and the maximum grades of visualization of the bile ducts and the gallbladder, respectively, in each case. The results of this preliminary study showed good to excellent bile duct and gallbladder visualization in all subjects with all dose ranges. The doses ranged from 0.11 cc./kg. (19.7 mg. iodine/kg.) to 0.59 cc./kg. (108 mg. iodine/kg.) with an average of 0.30 cc./kg. (55.14 mg. iodine/kg.).

Table 11 shows the dose related to timing and duration of good to excellent opacification (scores of 3 or 4). Good to excellent visualization of the bile ducts was noted as early as 10 minutes in 16 subjects (Fig. 1), and as early as 20 to 30 minutes in 2 subjects. In most cases the bile ducts were opacified immediately after the injection was completed (0 time) (Fig. 2). Furthermore, the subsegmental biliary radicles were clearly seen without tomography, during the early stages of the examination (Fig. 1; and 2), often visible for as long as 1 hour after the injection (Fig. 3). In fact, persistence of the good-to-excellent visualization was found up to 60 minutes in all subjects.

The grading of the quality of visualization of the biliary tract was subjected to statistical analysis. Based on an over-all score (obtained by averaging over 10 observation points), a statistically significant ($p < 0.01$) dose-response relationship was found for bile duct visualization, with better results obtained with increasing dose. However, this relationship was not consistent at all time points and, in fact, did not begin to appear until 90 minutes after administration of the contrast agent. Up to and including 1 hour, all 3 doses gave essentially identical good to excellent results. After 1 hour, the effects of the 10 cc. dose

TABLE I

DOSE OF MEGLUMINE IODOXIMATE RELATED TO BODY WEIGHT

Case No.	cc./kg.	Mg. BI/kg.	Maximum Score 10 cc. Bile Ducts	Maximum Score 10 cc. Gallbladder	Case No.	cc./kg.	Mg. BI/kg.	Maximum Score 20 cc. Bile Ducts	Maximum Score 20 cc. Gallbladder	Case No.	cc./kg.	Mg. BI/kg.	Maximum Score 30 cc. Bile Ducts	Maximum Score 30 cc. Gallbladder
1	0.16	29.56	4	4	3	0.39	62.03	4	4	2	0.41	74.59	3	0*
6	0.19	34.14	4	4	4	0.27	48.67	4	4	5	0.45	83.93	4	4
7	0.16	29.47	4	4	9	0.27	49.26	4	4	8	0.46	83.46	4	4
10	0.11	19.74	4	4	11	0.27	50.20	4	4	13	0.39	70.47	4	4
12	0.14	25.41	3	4	14	0.30	54.22	4	4	15	0.49	90.37	4	4
16	0.15	27.89	4	4	18	0.28	52.14	4	4	17	0.59	107.96	4	4
Average	0.15	27.70	3.83	4.0		0.30	52.75	4.0	4.0		0.47	84.98	3.83	4.0

* Patient 2 had many small stones in gallbladder and a stone in cystic duct. Therefore, scoring for this patient was omitted from calculating average scores.

Ranges

0.11 cc./kg.—0.59 cc./kg. (Av. 0.30 cc./kg.)

19.74 mg. bound iodine (BI)/kg.—107.96 mg. bound iodine (BI) kg. (Av. 55.14 mg./kg.)

TABLE II

DOSE OF MEGLUMINE IODOXIMATE RELATED TO MAXIMUM TIME OF OPACIFICATION

Case No.	Maximum Time* of Opacification (min.)		Case No.	Maximum Time* of Opacification (min.)		Case No.	Maximum Time* of Opacification (min.)	
	10 cc.			20 cc.			30 cc.	
	Bile Ducts	Gall-bladder		Bile Ducts	Gall-bladder		Bile Ducts	Gall-bladder
1	20–60	60–240	3	10–240	20–120	2	10–60	—
6	10–60	20–240	4	10–150	30–240	5	10–240	40–240
7	10–60	20–240	9	10–150	30–240	8	10–150	20–240
10	10–60	40–240	11	10–120	30–240	13	30–180	40–240
12	10–120	20–240	14	10–90	60–240	15	10–240	120–240
16	10–60	30–240	18	10–60	10–240	17	10–240	30–240

* Scores of 3 or 4.

dropped off sharply, those of the 20 cc. dose did less so, while the 30 cc. dose continued to produce good roentgenograms up to $2\frac{1}{2}$ to 3 hours (Fig. 4).

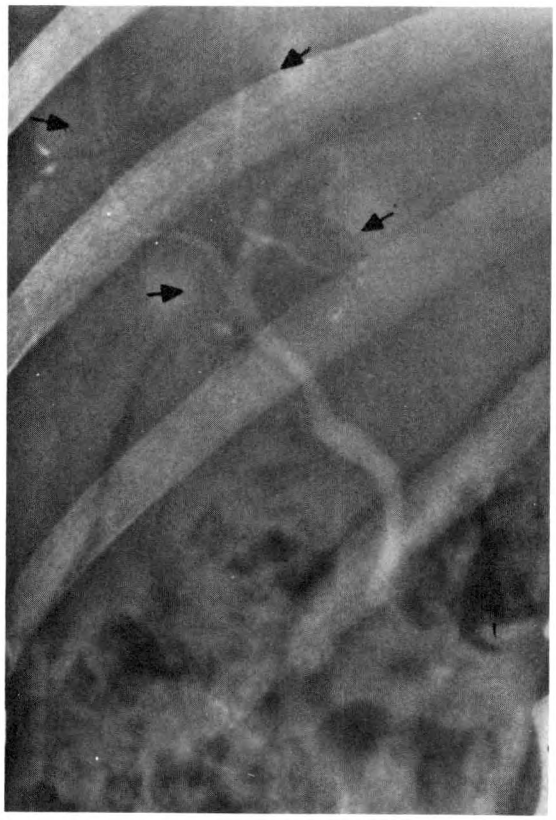

FIG. 1. Excellent visualization of bile ducts and subsegmental radicles (arrows) 10 minutes after the end of injection (20 cc.).

Good-to-excellent gallbladder visualization was obtained in all 18 cases (except 1 subject with an obstructed cystic duct). Good to excellent opacification appeared as early as 20 to 40 minutes in 15 subjects (Fig. 5), and as early as 60 to 120 minutes in 2 subjects. Persistent good-to-excellent visualization was obtained up to 4 hours in

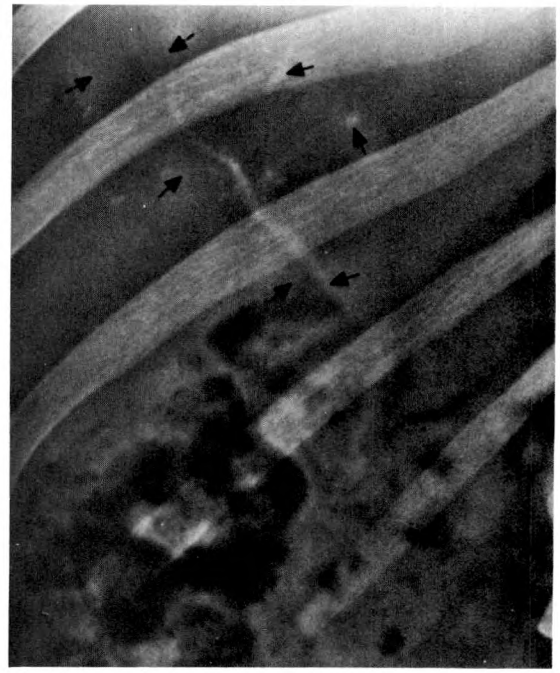

FIG. 2. Good visualization of bile ducts (lower arrows) and subsegmental radicles (upper arrows) without the use of tomography, seen immediately after the end of an injection, 0 time (10 cc.).

16 subjects (Fig. 6) and 2 hours in 1 subject. Statistical analysis of gallbladder visualization showed no dose-response relationships either over-all or at individual time points. (Case 2, in the 30 cc. group, had no filling of the gallbladder because of a stone in the cystic duct and was omitted from the statistical evaluation.)

In the higher dose ranges (30 cc.) excretion of the contrast agent by the kidneys was readily visible immediately after the end of the injection, reaching a maximum in 10 minutes and beginning to fade away after 20 minutes (Fig. 7, *A–C*).

Meglumine iodoxamate 40 per cent solution, in doses ranging from 10 to 30 cc., appears to be a well-tolerated preparation for use in intravenous cholangio-cholecystography. There were no significant changes in the physical examinations and vital signs at the 10 and 20 cc. dose levels, and only minor, transient changes at 30 cc. Three of the 18 participants experienced adverse effects, none of which was clinically significant. These included 1 case of mild nausea when a 6 minute injection time instead of 10 was employed; 1 case with mild, self-limiting facial urticaria; and 1 case of mild transient pain in the arm above the injection site.

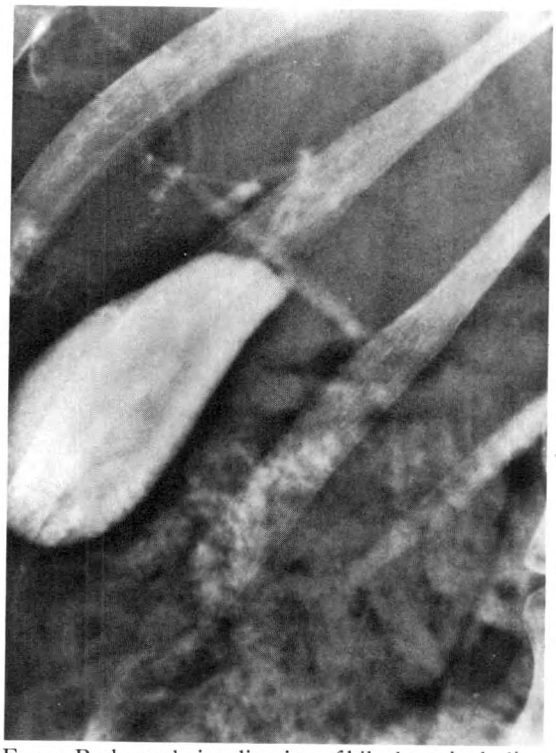

Fig. 4. Prolonged visualization of bile ducts including segmental radicles 3 hours after injection (30 cc.).

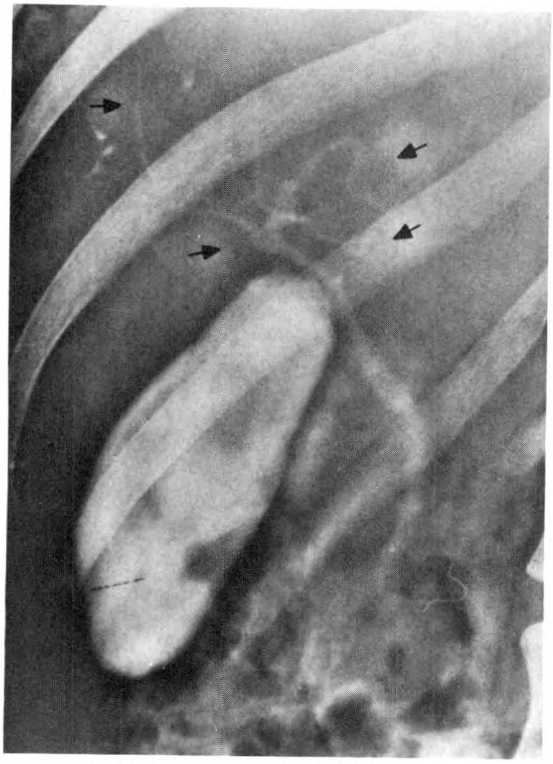

Fig. 3. Excellent visualization of bile ducts, subsegmental radicles (arrows) and gallbladder 1 hour after the end of injection (20 cc.).

There were few significant alterations of the laboratory findings. The only findings which might be attributable to the contrast agent were minimal elevations of total serum bilirubin 4 and 8 hours postinjection in 1 subject, minor elevations in lactic dehydrogenase activity in 2 subjects who received a 30 cc. dose, and minor elevations of creatinine phosphokinase activity in 1 subject who received a 20 cc. dose. These changes were generally transient in nature, therefore it is difficult to assign any clinical significance to these data. However, it was apparent that fewer changes in laboratory values were encountered with the lower doses (10 and 20 cc.).

DISCUSSION

The slow, intravenous injection of 10–30

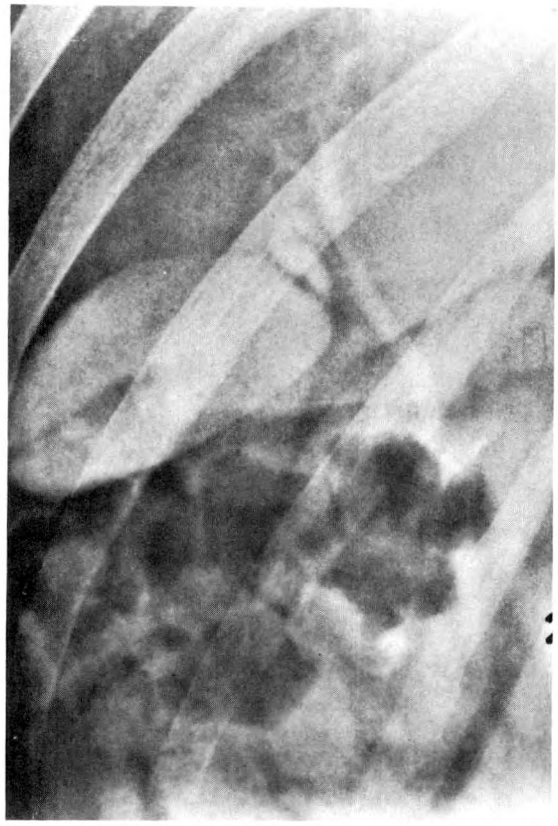

FIG. 5. Excellent visualization of gallbladder as early as 20 minutes after the injection (20 cc.).

cc. of meglumine iodoxamate over a 10 minute period was not associated with any significant adverse effects. There were no significant renal, hepatic, myocardial or hematopoietic changes. All doses of meglumine iodoxamate were well tolerated and only 3 subjects experienced mild adverse effects. No severe reactions were encountered. The adverse effects were those which are frequently experienced in intravenous cholangiography.

The new contrast medium, meglumine iodoxamate, which was used in this study, is formulated in a 40 per cent w/v solution and contains 183 mg. bound iodine/cc. Meglumine iodipamide (Cholografin), the most commonly used intravenous contrast medium for cholangio-cholecystography, as presently formulated in a 52 per cent w/v solution, contains 260 mg. bound iodine/cc.

The acute intravenous toxicity of the new compound is considerably lower than that of iodipamide in all animal species investigated. The MLD 50 in mice for meglumine iodoxamate is $5,580 \pm 160$ mg. free acid/kg., and the MLD 50 of meglumine iodipamide is $2,700 \pm 120$ mg. free acid/kg.; *i.e.*, the former is about one-half as toxic as the latter.*

The new compound is excreted principally in the bile, and studies in the dog showed a maximum biliary transport value twice that of iodipamide. Preliminary studies of meglumine iodoxamate in dogs showed that it is not metabolized to any significant extent when given either as a rapid intravenous injection or as a drip infusion. Most of the contrast medium (about 92 per cent), administered by either method, is excreted in the bile and urine as unchanged compound. The isolation and identification of trace amounts of metabolites possibly present in the urine has not yet been attempted.*

Meglumine iodoxamate appears to be a

* Squibb Research Investigators Brochure for SQ 21, 982.

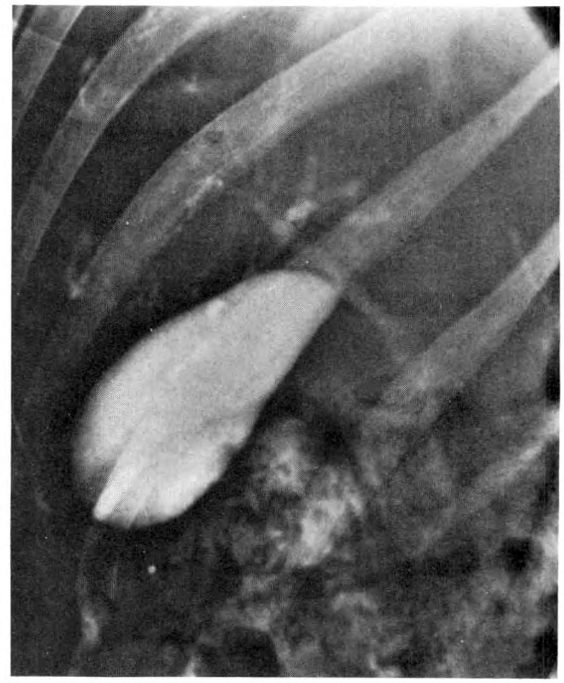

FIG. 6. Excellent visualization of gallbladder 4 hours after injection (20 cc.). Persistent visualization of bile ducts.

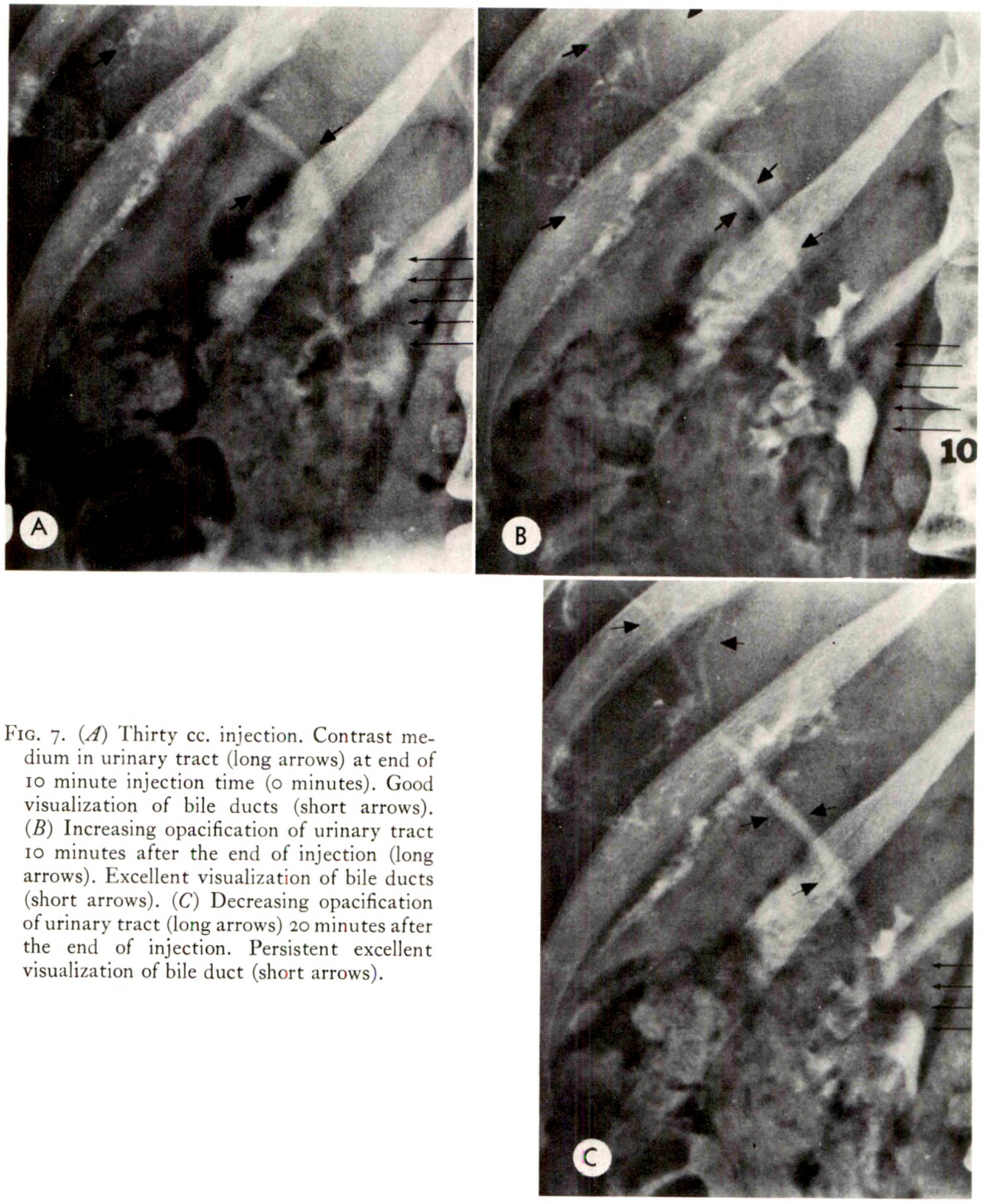

Fig. 7. (*A*) Thirty cc. injection. Contrast medium in urinary tract (long arrows) at end of 10 minute injection time (0 minutes). Good visualization of bile ducts (short arrows). (*B*) Increasing opacification of urinary tract 10 minutes after the end of injection (long arrows). Excellent visualization of bile ducts (short arrows). (*C*) Decreasing opacification of urinary tract (long arrows) 20 minutes after the end of injection. Persistent excellent visualization of bile duct (short arrows).

highly effective contrast agent for cholangio-cholecystography. Roentgenographic visualization of the major and subsegmental bile ducts and gallbladder is excellent for all 3 doses: 10, 20 or 30 cc. A more prolonged visualization of the bile ducts is achieved with the 20 and 30 cc. doses. For visualization of the gallbladder, no time-dose relationship was apparent, with all doses yielding excellent results up to 4 hours. With the 30 cc. dose, a pyelogram was observed in most subjects suggesting that the maximum rate of biliary excretion had been exceeded; thus excess contrast

agent was undergoing urinary excretion. This phenomenon is commonly seen during high dose cholangiography with meglumine iodipamide.

On the basis of this study, 20 cc. of the 40 per cent solution of meglumine iodoxamate, equivalent to 3.66 gm. of iodine, will be the recommended dose. It is further recommended that the roentgenologic examination of the bile ducts be made 10–60 minutes after injection of the contrast agent, and that the gallbladder be examined 30–240 minutes after injection. A double blind comparison of meglumine iodoxamate (Cholovue) and meglumine iodipamide (Cholografin) is now in progress.

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The authors wish to thank Barbara Oliver for the preparation of this manuscript; Neal Duenas, Gloria Love and Hayano Yagi for their technical assistance; and Dr. Arthur Itkin for his statistical analysis.

VOL. 125, NO. 1

ANGIOCARDIOGRAPHY WITH SMALL-SIZE RAPID CASSETTE CHANGER FILMS*

By MARIO CORNALI, M.D., GAETANO AZZOLINA, M.D., *and* SOFIA REDAELLI, M.D.
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ABSTRACT:

The disadvantages of small-size 10×10 cm. films in angiocardiography do not preclude correct diagnosis in a well equipped cardiac laboratory. On the contrary, the advantages are such as to recommend a more extensive use. In our department the method has been limited to pediatric patients, mainly because of excessive scattered radiation at high kVp. It is hopeful that improvement in recording and combination with image intensification will make the system available in the future for adult use.

AN angiocardiographic examination can be performed according to various techniques each of which has its advantages and disadvantages. Full size roentgenograms have the most desirable definition, but are difficult to handle and relatively expensive. Spot films are less expensive, more practical to handle but, at the present time, with the equipment generally available, are lacking in definition. Cine-films offer the advantage of a dynamic approach, but they require an extensive investment and, for publication purposes, have limited value.

For patients weighing less than 25 Kg we have been using a system that utilizes small-size, 10×10 cm. films. This Rapid Cassette Changer method, based on photofluorography, has several advantages.

TECHNIQUE

The two recording cameras, for horizontal and vertical projections, are of the kind commercially available for mass chest surveys and are manufactured according to photofluoroscopic principles. The cameras allow 6 programs of 5 films each, at a speed of 6, 4, 2, 1, or ½ films per second. A specially designed electronic device allows exposure of 1 film while another, in the other projection, is being moved in position. Roentgenographic events are recorded on 10×10 cm. films which can be automatically processed. Fast processing, 90 second films are presently available. The power generators have a capacity of 1200 mAs, 130 kVp and can be triggered alternatively for each direction. For a 10 Kg patient, 40 mAs and 85 kVp, at a focus distance of 85 cm. with a 12:1 grid, are required. Catheterization is usually performed through a brachial vessel, the internal jugular vein, or the common carotid artery.[1] Injections are made with power pressure injectors. Studies are always made in anteroposterior and lateral projections. For right-sided injection, 25 films in each projection are usually sufficient in our experience. In order to record a small field-size and to avoid unnecessary scattered radiation, the area to be recorded is previously viewed on a monitor. The tube centering light must coincide with this area. Small pen-marks are printed on the skin at the light field edges. These marks are used, subsequently, as reference points, when the table top and tubes are moved in position over the cameras. This kind of centering is possible because the table top to image amplifier distance coincides with table top to camera grid distance. Thus, no geometrical error is introduced by centering. Following exposure, the 10×10 cm. films are removed from the receiving magazine, loaded in a new magazine and automatically processed. An entire examination is available within 16 minutes.

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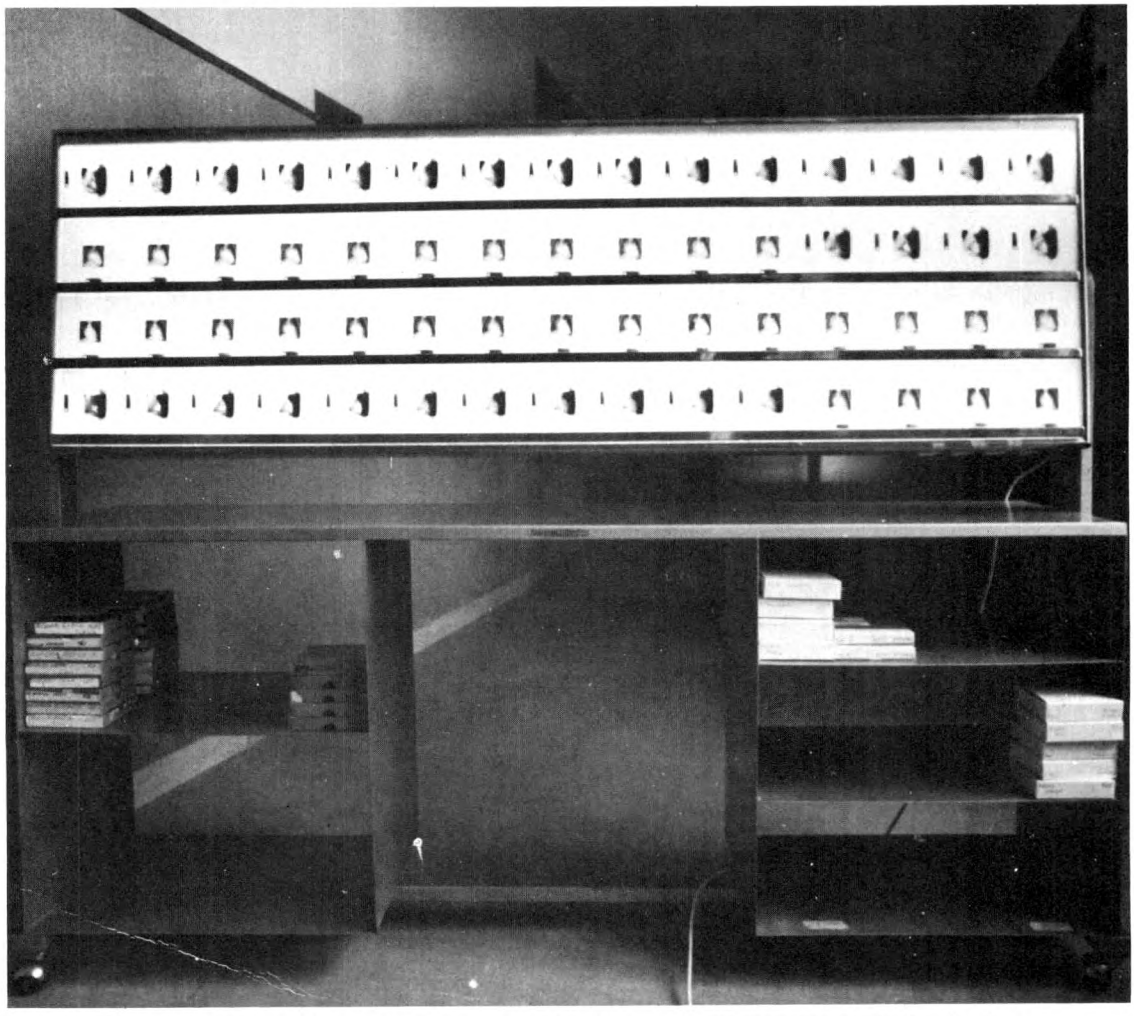

FIG. 1. An entire right and left heart examination can be displayed on a single view box.

DISCUSSION

In evaluating an angiocardiographic study, a complete view of all the films available is most desirable. This is, however, not possible with full-size 24×30 cm. or 35×35 cm. roentgenograms. On the other hand, 10×10 cm. films make this feasible. The 10×10 cm. films used for children by our group (a) are very easy to handle, (b) permit roentgenographically and diagnostically acceptable studies, (c) are quite economical (as compared to full size films and (d) are extremely convenient to file.

A radiologist can view an entire right and left heart examination on a single view box (Fig. 1). The time required for handling films is reduced to a minimum, a mean time of 45 seconds being required for displaying films as opposed to 20 seconds to take them down. Furthermore, given the facility with which films can be handled, the surgeon can be offered the unique opportunity of having the entire study in the operating room.

Definition, sharpness, and contrast, because of minification and the use of an optical rather than an electronic system, are roentgenologically acceptable. Figure 2 shows a 1:1 reproduction of 6 roentgenograms. With the aid of a magnifying lens,

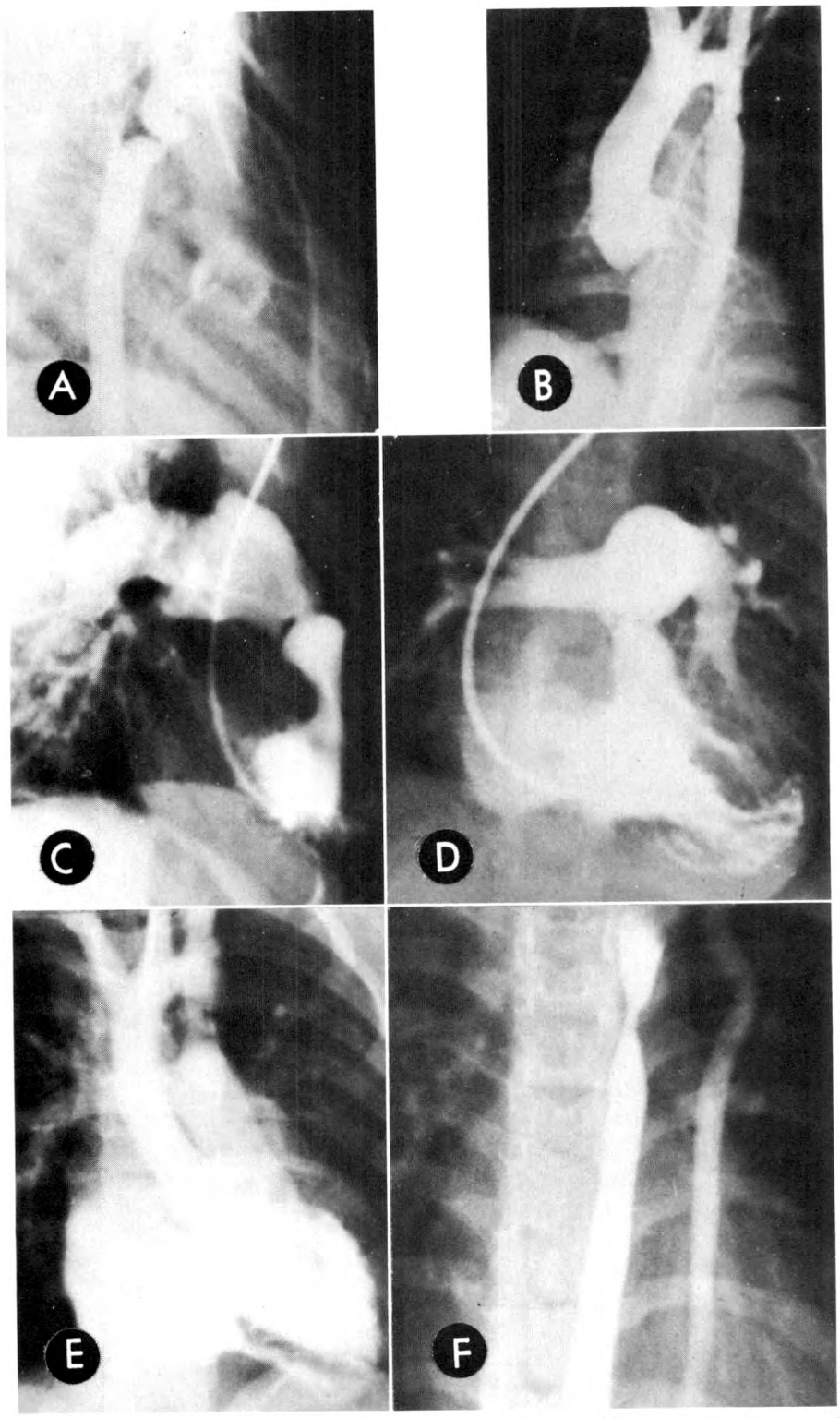

Fig. 2. A 1:1 reproduction of 6 cases of congenital heart disease. With the aid of a magnifying lens minor details can be sufficiently evaluated.

even minor details can be sufficiently evaluated. A magnifying view box is also commercially available. Over 3,000 examinations have been performed since 1967 with this method. Since most of the patients have subsequently been operated upon, it can be confidently stated that good diagnostic anatomic control has been obtained.

The average cost of an examination with this method is less than 1/10 that of conventional roentgenography. Furthermore, the system being fully automated, an x-ray technician can load, unload, and process 50 small-size films much faster than an equal number of 35×35 cm. films. This leaves him free for more practical, and useful work.

The space required for filing full-size films is considerable. With our method over 3,000 examinations can be filed in order in 3 small cabinets. The compactness of storage also reduces retrieval time and is of help to busy file-clerks.

As compared to roentgenography, photofluorography has the following disadvantages.

The resolution achieved by the presently available photofluorographic system is inferior to that achieved in roentgenography. The modulation transfer function for photofluorography is inferior to that offered by roentgenography.[2] Nonetheless, as our experience proves, the system allows highly acceptable diagnostic opportunities. Since angiocardiographic examinations are correlated with oxygen saturation studies, electrocardiograms, phonocardiograms, and vectorcardiograms, this disadvantage can be offset by these additional data. In our department 70 mm. spot films and 16 mm. cine-films are also available. This allows us to switch to these methods when needed.

Scattered radiation, if excessive, may become a serious problem for the diagnostician. For patients weighing more than 25 Kg, our camera requires more than 100 kVp in order to have sufficient penetration and to avoid excessive tube loads. This, of course, entails scattered radiation in an amount that may become roentgenographically undesirable. Furthermore, the use of high kVp reduces contrast, increases latitude, and makes the film appear too "flat." For these reasons we limit the use of the system to patients weighing less than 25 Kg. In addition, for the purpose of reducing secondary radiation from the second tube, an electronic device triggers the 2 x-ray tubes alternately. This circuitry permits exposure of 1 film while the other is being moved in position.

In roentgenography it is necessary to keep the radiation dose to a minimum. Our method requires a higher radiation dose than conventional roentgenography. For instance, at a focus-film distance of 85 cm. with a 12:1 grid, 40 mAs and 85 kVp are required for a 10 Kg patient. However, given the undisputed advantages of small-size films, a higher radiation dosage is not considered detrimental for congenital cardiac patients where thorough diagnosis is an essential feature.

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The authors wish to thank Miss Anna Frezzato for her assistance in the preparation of the manuscript.

REFERENCES

1. AZZOLINA, G., EUFRATE, S., and ALELLA, A. New approach to cardiac catheterization in infants and children. *Brit. Heart J.*, 1973, *35*, 643–646.
2. MORGAN, R. H. Frequency response function: valuable means of expressing informational recording capability of diagnostic x-ray systems. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1962, *88*, 175–186.

VOL. 125, NO. 1

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Published by: CHARLES C THOMAS, Publisher, 301–327 East Lawrence Avenue, Springfield, Illinois 62717.

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EDITORIAL

UROGRAPHY, SHOCK REACTION AND REPEATED UROGRAPHY

AT unexpected and unforeseen moments an excretory urogram may be followed by profound hypotension, shock, and unconsciousness.[21,22] Such a result elicits an appropriate resuscitative effort, employing drugs such as epinephrine.[2] The period of hypotension and unconsciousness may vary, but the response of the examining physician and the resuscitated patient is predictable. The patient is frightened and unwilling to undergo the risk of a similar repeated experience; and the physician, relieved that the patient did not die, is unwilling to risk the death of the patient in a subsequent examination, and advises the patient never to have a urogram again. When the patient's clinical situation subsequently indicates urographic evaluation, the physician called upon to execute the examination is thus faced with the decision of whether the risk can be satisfactorily undertaken or not. This Editorial is designed to provide a report of our experience with this problem.

During the past five years (January, 1970, through December, 1974) a modified point of view has been applied in the execution of urograms in the Outpatient Department of the Cleveland Clinic. While the prior approach included premedication with an antihistamine, close questioning of allergies and previous experience with contrast media, and an intravenous test dose, the current method is one of simply informing the patient that a urogram is to be performed, injecting the contrast medium straight away in a brisk and rapid fashion and completing the examination as individually indicated. If the patient raises questions these are discussed, but there is no attempt to provide an informed consent unless the patient requests it. No forms are completed nor signatures obtained. The injections are all made by two nurses who have been carefully selected and trained, and the examinations are carried out in a carpeted and pleasantly decorated area used exclusively for urography, with an ambience of calm, quiet, and friendly sociability. All is done to make the patients feel at ease, and the nurses and technicians avoid anxiety-causing words such as "dye," "allergy," "reactions," etc. During this period of time 21,198 outpatients have been examined. Experimental groups previously reported are not included in these numbers.[13,14,20] Sodium iothalamate has been used throughout as the contrast medium employing a dose of at least 50 milliliters with increasing volume according to body size and severity of renal failure. No drip infusions were used. All patients were adults, and all were prepared with a cathartic agent and dehydration from approximately 6:00 P.M. of the day before the examination. They were examined at scheduled intervals during the day from 8:00 A.M. to approximately 3:30 P.M.

During the same time period there were four patients who were frightened about the examination and were not reassured. The examination was not performed on these patients and they represent the only group that was not examined. Two of the four patients had never had a urogram and were fearful of the examination, whereas the other two had had urticaria or nausea and vomiting. I believe that patients who cannot be reassured should not have urography and the patients should be able to assent to the examination willingly once

their problems have been adequately discussed.

Observed reactions such as nausea and vomiting and urticaria have decreased progressively over the five year period of time as greater experience with this anxiety reducing method was acquired; however, the total reactivity is summarized in Table 1.

Included in this group were nine patients previously examined at other institutions whose urograms had resulted in shock and unconsciousness, varying in length from a few hours to three days, with total recovery. These experiences had occurred following the employment of sodium and methylglucamine iothalamate and sodium and methylglucamine diatrizoate and had occurred at intervals of seven months to four years prior to the subsequent urogram performed in our department. None of these patients experienced any reactivity of any type.

The reactivity following our approach is slightly better than that described by Witten, Ochsner or Ansell.[1,19,24] It is perhaps impossible, in the current state of the art, to perform urograms on everyone without risking at least a minor reactivity.

Very little treatment was involved in dealing with the reactions described in Table 1. The patients with thrombophlebitis had the characteristic changes in the veins at the site of injection and these were followed by repeated visits to the Radiology Department and consultation in a few cases with the Department of Peripheral Vascular Diseases. All, I believe, are the result of the direct irritation of the intima of the vein by the contrast medium and all resolved slowly and completely.

The three patients who experienced asthma were all *bona fide* asthmatics who recovered with small injections of epinephrine. The three patients who experienced mild hypotension did not require medication and regained a normotensive state within 20 minutes to two hours. I would describe these episodes as being syncopal attacks or vasovagal responses.[21] The one patient who was confused and hypotensive had been given by error intravenous benadryl, 50 milligrams, immediately prior to the sodium iothalamate by a new resident unfamiliar with our methods. This patient was elderly with a poor command of the English language. He spontaneously got up from the examining table and proceeded to defecate on his way to the nearby bathroom. No treatment was given and he recovered shortly thereafter so that the examination could be satisfactorily completed. The patients with nausea and vomiting received no treatment and while some of them developed erythema of the face and neck, in most instances the problem was confined to slight nausea which passed quickly and was dealt with by reassurance. The urticaria was usually mild and did not require treatment. During the period of five years reported here only 18 patients were given a small dose of parenteral histamine to ease the discomfort more rapidly than would spontaneous regression. One patient did experience conjunctivitis with the urticaria which was still present in mild form 24 hours later. Minor problems such as a metallic taste in the mouth and arm pain at the completion of the injection were not recorded.[24]

Table 1

REACTIONS TO CONTRAST MEDIA

Reaction	Percent
Urticaria	1.1
Nausea and emesis	3.6
Asthma	0.014
Mild hypotension	0.014
Confusion and mild hypotension	0.005
Thrombophlebitis	0.05
No reactions	95

Of great interest to us and forming the primary reason for reporting this experience were the nine patients who had previously been unconscious and hypotensive following previous urography. In every instance these patients raised questions and objections to the performance of this examination because of their earlier urogram. This was discussed with a physician

who offered reassurance and indicated a willingness to perform the examination despite this previous problem if the patient was willing to have it. The patients were told that the contrast medium that we used was newer and less capable of producing reactions and that we had no reason to fear that a similar reaction would occur on the current occasion. These patients had all had well documented episodes of severe hypotension and unconsciousness and seven had also had a preceding negative intravenous test dose with two receiving no previous test dose.[9] There was no response of any kind identified in these patients following the injection of the contrast medium in our department. None of them received any premedication or test dose.[22]

My purpose is to document in the medical literature that one can undertake repeated urography in patients with severe reactions, even those that would appear life-threatening, without a repetition of the previous experience. This is not calculated to encourage a cavalier approach but would serve to demonstrate that in a clinically indicated situation one very severe reaction does not necessitate a recurrence.[24] All of these nine patients were examined in separate institutions of varying character and it is difficult to categorize them. It does emerge that the only essential difference that could be determined is one of approach, as in all of the previous examinations these patients had been questioned in varying degrees about reactivity to previous urography, allergies and sensitivity to iodides or seafoods.

Data have been accumulated which would indicate that patients can indeed be "frightened to death."[7,10] One would wish to avoid this occurrence in the performance of a diagnostic examination. It is my conclusion, supported by evidence reported elsewhere, that the most significant aspect of undesirable reactions to urographic contrast media is fear.[13,14] The contrast medium produces undeniable physiologic effects, one of which is translatable as a sensation of heat.[11,18] These have been and are being investigated.[6,15–17] Perhaps one day there will be data that implicate an enzymatic defect or other mechanism which causes these physiologic changes in a given person to be extreme and life-threatening.[4,5,23] At present, however, I would suggest that the fearful patient responds to these physiological changes or indeed even to the needle penetration of the skin and vein in such a fashion as to produce shock, cardiac arrest and death.[20] It is the fear, I believe, which leads to an unpredictable or apparently idiosyncratic reaction to the contrast medium.

Recent reports have suggested that there is a remote possibility of "natural occurring antibodies" to urographic contrast media or that the contrast medium may interfere with the complement system.[12,17] If these recent hypotheses prove to be more useful than those reported in the past, perhaps these patients can be satisfactorily identified and treated prior to urography. Until that time, each of us must operate our departments and make decisions on a daily basis based on personal experience and conviction.

It has been a well documented experience that there is a higher incidence of fatality with urography beyond the age of 50 and it may be that the fear reaction combined with greater myocardial susceptibility contributes to this result.[1] We have examined the problem of electrocardiographic changes in urography and have not detected any statistically significant changes when patients with a history of heart disease were compared with patients without a similar history.[3,20] Advanced age had no effect, and while infusion urography produced more frequent changes, similar results could be produced when patients were infused with 5 percent dextrose and water, following the application of the tourniquet and the intravenous insertion of the needle. Electrocardiographic changes that we observed were deemed to be of no clinical significance and heart disease and

advanced age were not contradictory to urography.[20] It has also been established that asthmatics represent a particular group wherein a higher incidence of reaction, namely asthmatic attack, is found following the injection of the urographic contrast medium.[1] It is possible that these patients and this higher incidence coincide due to a learned response to a stressful situation.

What recommendation can one make? It would be my advice that a Radiology Department be willing to consider urography on any patient regardless of previous reactive history. If the examination is clinically indicated the patient should be prepared in the routine fashion but with no premedication such as steroids, antihistamines, or other medication, as these have proved to be of no benefit.[8,25] The Radiology Department should be prepared and equipped to deal with the most severe reactions, and the drug of choice that should be readily at hand is epinephrine.[2] These preparations, however, should be covert and the patient should be offered reassurance and an atmosphere calculated to reduce anxiety. The examination should then be done in a routine fashion. The demeanor and behavior of all personnel are, I believe, of enormous importance in the outcome of these examinations. While it may be difficult to think in terms of shock, severe reactions, and death occurring as a result of a fear reaction during urography, it is my approach to act in this fashion until someone has proved that there is a useful mechanism independent of the patient's psyche which can be satisfactorily dealt with.

Anthony F. Lalli, M.D.

REFERENCES

1. Ansell, G. Adverse reactions to contrast agents: scope of problem. *Invest. Radiol.*, 1970, *5*, 374–384.
2. Barnhard, H. J., and Barnhard, F. M. Emergency treatment of reactions to contrast media: updated 1968. *Radiology*, 1968, *91*, 74–84.
3. Berg, G. R., Hutter, A. M., Jr., and Pfister, R. D. Electrocardiographic abnormalities associated with intravenous urography. *New England J. Med.*, 1973, *289*, 87–88.
4. Bergman, H., and Ellner, H. Severe reactions following previously uneventful intravenous pyelograms. *New York J. Med.*, 1960, *60*, 105–106.
5. Boecemann, H. H. Komplikationen bei deo intravenösen urographic. *Urol. Int.*, 1966, *21*, 500–510.
6. Brasch, R. C., Rockoff, S. D., Kuhn, C., and Chraplyvy, M. Contrast media as histamine liberators. II. Histamine release into venous plasma during intravenous urography in man. *Invest. Radiol.*, 1970, *6*, 510–513.
7. Engel, G. L. Sudden and rapid death during psychological stress: folklore or folk wisdom? *Ann. Int. Med.*, 1971, *74*, 771–782.
8. Evans, R. R., and Rackemann, F. M. Allergy-corticotropin and cortisone: review of literature from September, 1950 to January, 1952. *Arch. Int. Med.*, 1952, *90*, 96–127.
9. Fischer, H. W., and Doust, V. L. Evaluation of pretesting in problem of serious and fatal reactions to excretory urography. *Radiology*, 1972, *103*, 497–501.
10. Greene, W. A., Goldstein, S., and Moss, A. J. Psychosocial aspects of sudden death: preliminary report. *A.M.A. Arch. Int. Med.*, 1972, *129*, 725–731.
11. Hodson, C. J. Physiological changes in size of human kidney. *Clin. Radiol.*, 1961, *12*, 91–96.
12. Klein-knecht, D., Deloux, J., and Homberg, J. C. Acute renal failure after intravenous urography: detection of antibodies against contrast media. *Clin. Nephrol.*, 1974, *2*, 116–119.
13. Lalli, A. F. Use of chlordiazepoxide hydrochloride (librium) in preparation for urography. *J. Canad. A. Radiologists*, 1974, *25*, 44–46.
14. Lalli, A. F. Urographic contrast media reactions and anxiety. *Radiology*, 1974, *112*, 267–271.
15. Lasser, E. C., and Lang, J. H. Inhibition of acetylcholinesterase by some organic contrast media: preliminary communication. *Invest. Radiol.*, 1966, *1*, 237–242.
16. Lasser, E. C., Walters, A., Reuter, S. R., and Lang, J. Histamine release by contrast media. *Radiology*, 1971, *100*, 683–686.
17. Lasser, E. C., Kolb, W. P., and Lang, J. H. Contrast media activation of serum complement system. *Invest. Radiol.*, 1974, *9*, 6A.
18. Lindgren, P. Hemodynamic response to contrast media. *Invest. Radiol.*, 1970, *6*, 424–435.
19. Ochsner, S. F., and Calonje, M. A. Reactions

to intravenous iodides in urography. *South. M. J.*, 1971, *64*, 907–911.
20. Reich, N. E., Seidelmann, F. E., Lalli, A. F., Castle, L. W., and Alfidi, R. J. Electrocardiographic changes during urography. *Cleveland Clin. Quart.*, 1974, *41*, 66–66.
21. Sandström, C. Secondary reactions from contrast media and allergy concept. *Acta radiol.*, 1955, *44*, 233–242.
22. Svendson, P., and Wilson, J. Adverse reactions during urography and modification by atropine. *Acta radiol.* (Diag.), 1971, *11*, 427–433.
23. Toniolo, G., and Buia, L. Risultati di una inchiesta nazionale sugli incidenti mortali da iniezone di mezzi di contrasto orgno-iodate. *Radiol. Med.*, 1966, *52*, 625–657.
24. Witten, D. M., Hirsch, F. S., and Hartman, G. W. Acute reactions to urographic contrast medium: incidence, clinical characteristics and relationship to history of hypersensitivity states. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *119*, 832–840.
25. Wolfromm, R., Dehouve, A., Degaud, F., Waltez, E., Lang, R., and Crehalet, A. Les accidents graves par injection intraveineuse de substances iodees pour uragraphie. *J. de radiol., d'électrol. et de méd. nucléaire*, 1966, *47*, 346–362.

NEWS ITEMS

RADIOLOGY AND NUCLEAR MEDICINE

The George Washington University and the University of Utah Departments of Radiology will present a course, "Continuing Education in Radiology Nuclear Medicine," April 1 through 4, 1976.

While a number of courses in nuclear medicine has been available, all have utilized the lecture format. As in our previous courses, this course will be conducted in seminar fashion. Nine 1½–2 hour seminars will be given. Scheduled seminars will stress up-to-date aspects of radionuclide imaging of liver, spleen, bone marrow, bone, brain, thyroid and heart. Radionuclide imaging of the lung with radiographic and angiographic correlation will be included. Additionally, a seminar session and lectures on computerized tomography will be included. These topics will be covered in small group sessions led by nationally recognized, experienced leaders in the field.

This course is approved for 20 hours of AMA Category 1 credit.

For further information, please contact: David O. Davis, M.D., Department of Radiology, George Washington University Medical Center, 901 23rd Street, NW, Washington, D.C. 20037. (202) 331-2710.

NEOPLASTIC DISEASE

The Tenth Annual Seminar organized by the Mexican Society of Radiology, "Radiological Topics in Neoplasia," will be held January 12–16, 1976 in Mexico City, Mexico. Sponsored by The Mexican Federation of Radiological Societies, Loyola University of Chicago, the Interamerican College of Radiology, and the American College of Radiology, the Seminar will demonstrate advances and limitations of radiology in diagnosis of neoplastic disease and establish data that will permit a differential diagnosis.

There will be simultaneous translation in English and Spanish.

The program is approved for Category 1 with 28 credit hours by The American Medical Association.

For further information, please contact: Rogelio Moncada, M.D., Loyola University Medical Center, 2160 South First Avenue, Maywood, Illinois 60153. (312) 531-3928.

RESPIRATORY DISEASE

The American Thoracic Society, the medical section of the American Lung Association, invites submission of papers on all scientific aspects of respiratory disease for presentation at the 1976 annual meeting in New Orleans, Louisiana, May 16–19, 1976. Membership in the Society is not a prerequisite to participation on the program.

Abstracts of papers should be submitted before January 6, 1976, to the Chairman of the Annual Meeting Committee. Abstracts of papers from investigators outside the United States will be welcome provided they are in English and are submitted with the understanding that all papers accepted must be presented clearly in English at the Annual Meeting.

The required forms and instructions may be obtained by writing to: Marvin A. Sackner, M.D., Chairman, ATS Annual Meeting Committee, American Thoracic Society, 1740 Broadway, New York, New York 10019.

ULTRASOUND AND NUCLEAR MEDICINE

The first annual postgraduate seminar, Ultrasound and Nuclear Medicine, "Interrelated Roles in Medical Diagnosis," will be held January 4–7, 1976 in Key Biscayne, Florida. This Seminar is sponsored by the Department of Radiology, Division of Nuclear Medicine at the University of Miami School of Medicine (at Jackson Memorial Hospital), Miami, Florida.

For further information about this Sem-

inar, please contact: Mrs. Lucy R. Kelley, Department of Radiology, Jackson Memorial Hospital, Miami, Florida.

GENITOURINARY RADIOLOGY COURSE

The Twelfth Annual Postgraduate Course in Radiology (G.U. Tract), sponsored by the Department of Radiology, Virginia Commonwealth University—Medical College of Virginia, will be held February 29–March 4, 1976, at the Williamsburg Conference Center in Williamsburg, Virginia.

The current concepts in diagnostic radiology of the genitourinary tract will be reviewed with emphasis on the applications of angiography, ultrasonography and nuclear medicine.

The program, open to all physicians interested in the subject, offers 20 hours of A.M.A. Category 1 accreditation. A certificate of attendance will be awarded to registrants.

For further information, please contact: Gary G. Ghahremani, M.D., MCV—Station, P.O. Box 728, Richmond, Virginia 23298.

DIAGNOSTIC RADIOLOGY

The George Washington University Department of Radiology announces The Washington Bicentennial Radiology Post-Graduate Conference at the Statler-Hilton Hotel, Washington, D.C., June 27 through July 4, 1976.

This will combine high quality diagnostic radiology education with the many special events commemorating our country's Bicentennial at the Smithsonian, Kennedy Center, Mount Vernon, and in the U.S. Congress.

For further information, please contact: S. David Rockoff, M.D., Professor and Chairman, Department of Radiology, George Washington University Medical Center, 901 23rd Street, N.W., Washington, D.C. 20037 (202) 331-6281.

NUCLEAR MEDICINE

The Basic Radiation Sciences Division of the Medical University of South Carolina will offer a one week course entitled "Quality Control for Nuclear Medicine" from November 3, 1975 through November 8, 1975.

The course will stress both didactic lectures and laboratory experience in quality control procedures. The material will be of interest to nuclear medicine technologists and physicians specializing in nuclear medicine. The course is being supported by a grant from the U. S. Department of Health, Education, and Welfare.

For further information, please contact: G. Donald Frey, Ph.D., Basic Radiation Sciences, Medical University of South Carolina, 80 Barre Street, Charleston, South Carolina 29401.

THE FLEISCHNER SOCIETY

The Fleischner Society's Sixth Annual Course on Chest Radiology will be held in Zagreb, Yugoslavia, May 30 through June 1, 1976. Exciting new concepts related to the normal and diseased chest will be presented by a large well-known international faculty. All presentations will be in English with earphone translations. A variety of teaching methods will be used, including small group colloquies. The Fleischner Society is an international fellowship of physicians dedicated to advancing knowledge in the field of chest radiology.

For further information, please contact: Dr. Benjamin Felson, President, Fleischner Society, General Hospital, Cincinnati, Ohio 45267.

CONCEPTS IN THE DIAGNOSIS AND TREATMENT OF CANCER

The Harvard Medical School announces a course in "Concepts in the Diagnosis and Treatment of Cancer," to be given November 24–26, 1975 at the Massachusetts General Hospital.

This selected-topics course covers the diagnosis and treatment of cancer, with emphasis on newer developments. Presentations will include panel discussions and symposia, with participants represent-

ing many disciplines focusing upon specific clinical cases. Emphasis will be placed upon diagnostic and practical therapeutic measures of general interest. The course should be valuable to general surgeons, general internists, and others with a broad interest in malignant disease.

This course has Category 1 accreditation for approximately 21 hours toward the A.M.A.'s Physicians' Recognition Award.

For further information, please contact: Associate Dean, Department of Continuing Education, Harvard Medical School, 25 Shattuck Street, Boston, Massachusetts 02115.

SPRING ROENTGEN CONFERENCE

The Spring Roentgen Conference of the New Orleans Radiology Society is to be held in New Orleans on May 6, 7 and 8, 1976.

The meeting will present several well known guest speakers, and the subjects will be general radiology, nuclear medicine, and ultrasound.

For further information, please write to: The New Orleans Radiology Society, Post Office Box 51706, New Orleans, Louisiana 70151.

ANGIOGRAPHY

The Departments of Radiology of Shadyside Hospital and the University of Pittsburgh School of Medicine will present a seminar "Advances in Angiography, Technical and Clinical," January 28 to 30, 1976 in Las Vegas, Nevada. Three full day sessions will cover new equipment, new techniques, and other subjects such as extraction of biliary calculi, ultrasound, and computer assisted tomography.

For further information, please contact: Shadyside Hospital, Radiology Seminar, 5230 Centre Avenue, Pittsburgh, Pennsylvania 15232.

BOOK REVIEW

RADIOLOGY OF SYNDROMES. By Hooshang Taybi, M.D., M.Sc., Clinical Professor of Radiology, University of California School of Medicine, San Francisco; Chief, Department of Radiology, Children's Hospital Medical Center, Oakland; Consultant Radiologist, Children's Hospital of San Francisco and Letterman General Hospital, San Francisco. Cloth. Pg. 333, with many illustrations. Year Book Medical Publisher, Inc., 35 E. Wacker Drive, Chicago, Ill. 60601, 1975.

Radiology of Syndromes by Hooshang Taybi, M.D. is a compilation of 541 syndromes, including hereditary disorders and congenital malformations. The syndromes are listed alphabetically in the text along with their numerous synonyms. The mode of inheritance and clinical and radiological manifestations for each syndrome are briefly but concisely presented with the most important in italics. Numerous radiographic reproductions of good quality illustrating pertinent findings are interspersed throughout the text. References are included in the text after each syndrome and selected references of a more general nature are listed in the back of the book. A gamut index presented at the end of the book should be quite useful. The index is divided into 15 anatomic systems beginning with Abdomen. Under each of the 15 anatomic systems particular clinical or radiographic findings are alphabetized and a list of possible syndromes is included below the finding. For instance, under the Alimentary System, the first finding listed is Anorectal Anomaly followed by a list of 7 syndromes beginning with Cat-Eye syndrome and ending with Rieger's syndrome. The syndromes in which the particular clinical or radiographic findings are most constant are printed in italics.

This book should prove a handy and practical guide to the recognition and classification of malformation syndromes and genetically determined disease.

JOHN A. COUZENS, M.D.

BOOKS RECEIVED

INFLAMMATORY BOWEL DISEASE. Edited by Joseph B. Kirsner, M.D., Ph.D., Louis Block Distinguished Service Professor of Medicine: Chief of Staff and Deputy Dean for Medical Affairs, University of Chicago, Department of Medicine, Chicago, Ill.; and Roy G. Shorter, M.D., Consultant, Departments of Pathology and Medicine, Mayo Clinic and Mayo Foundation; Professor of Pathology and Medicine, Mayo Medical School, Rochester, Minn. Cloth. Pp. 465, with 240 illustrations. Price, $35.00. Lea & Febiger, Washington Square, Philadelphia, Pa. 19106, 1975.

FUNDAMENTALS OF RADIATION THERAPY AND CANCER CHEMOTHERAPY. By Sidney Lowry, M.D., Professor of Cancer Studies, The Queen's University of Belfast; formerly, Director of Radiation Therapy, Maine Medical Center, Portland, Maine; Assistant Professor of Therapeutic Radiology, Tufts-New England Medical Center, Boston, Mass. Paper. Pp. 198, with some figures. Price, $7.50. Arco Publishing Company, 219 Park Avenue South, New York, N. Y. 10003, 1974.

AN INTRODUCTION OF PHYSICS FOR RADIOLOGIC TECHNOLOGISTS. By Billie J. Graham, Ph.D., Associate Professor of Physics, United States Naval Academy, Annapolis, Md.; and William N. Thomas, M.D., Assistant Professor of Radiology, University of Maryland School of Medicine, Baltimore, Md. Cloth. Pp. 331, with 154 illustrations. Price, $12.50. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1975.

THE EPIDEMIOLOGY OF CANCER IN PAPUA NEW GUINEA. Edited by L. Atkinson, J. K. Clezy, P. S. Heay-Young, G. C. Scott, and S. C. Wigley. Paper. Pp. 184, with some illustrations. Price, $10.00. Department of Public Health, Box 2084, Konedobu, Papua New Guinea, 1974.

RADIOGRAPHIC ANATOMY. By James McInnes, F.S.R., T.E., F.R.P.S. Editor of the Ninth Edition of Clark's Positioning in Radiography. Cloth. Pp. 71, with many illustrations. Price, $16.50. Appleton-Century-Crofts, 292 Madison Avenue, New York, N. Y. 10017, 1975.

ULTRASOUND IN MEDICINE. Volume I. Proceedings of the 19th Annual Meeting of the American Institute of Ultrasound in Medicine. Edited by Denis White, Department of Medicine, Queens University, Kingston, Ontario, Canada. Cloth. Pp. 611, with some figures. Price, $47.50. Plenum Publishing Corporation, 227 West 17th Street, New York, N. Y. 10011, 1975.

SARCOIDOSIS: A CLINICAL APPROACH. By Om P. Sharman, M.B., B.S., F.A.C.P., F.C.C.P., D.I.M. and H.(Eng.), Assistant Professor of Medicine, University of Southern California School of Medicine; Physician at Los Angeles County Hos-

pital, University of Southern California Medical Center, Los Angeles, Calif. Cloth. Pp. 228, with many figures. Price, $17.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1975.

Health Protection of Radiation Workers. By W. Daggett Norwood, M.D., Consultant in Environmental Health, HEJF Battelle Northwest, U. S. Navy, U. S. Air Force, Hanford Environmental Health Foundation, Richland, Wash. Cloth. Pp. 442, with some figures. Price, $27.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1975.

Immunity and Cancer in Man: An Introduction. Edited by Arnold E. Reif, Mallory Institute of Pathology and Boston University School of Medicine, Boston City Hospital, Boston, Mass. Cloth. Pp. 160, with some illustrations. Price, $12.50. Marcel Dekker, Inc., 270 Madison Avenue, New York, N. Y. 10016, 1975.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. James Franklin Martin, 300 S. Hawthorne Rd., Winston-Salem, N. C. 27103. Annual Meeting: Marriott Hotel, Atlanta, Ga., Sept. 30–Oct. 3, 1975.

AMERICAN RADIUM SOCIETY
Secretary, Richard H. Jesse, Dept. of Surgery, M. D. Anderson Hosp. and Tumor Institute, Houston, Tex. 77025. Annual Meeting: Vancouver, B. C., Canada, May 9–14, 1976.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Theodore A. Tristan, Harrisburg, Pennsylvania. Annual Meeting: McCormick Place on the Lake, Chicago, Ill., Nov. 30–Dec. 5, 1975.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Written Examination will be held in 14 selected sites, June 21, 1975.

Oral examinations will be held: Pittsburgh Hilton, Pittsburgh, Pa., June 2–7, 1975; Statler Hilton, Dallas, Texas, Dec. 8–13, 1975; Marriott Motor Hotel, Chicago, Ill., June 14–19, 1976; Sheraton Biltmore, Atlanta, Ga., Dec. 6–11, 1976.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1976 is September 30, 1975.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Dr. Robert G. Waggener, Dept. Radiol., Univ. Tex. Health Sciences Center, San Antonio, Tex. 78284 Annual Meeting, Aug. 3–7, 1975.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wis., 54601. Annual Meeting: Hyatt Regency Hotel in the Embarcadero Center, San Francisco, Calif., Oct. 8–12, 1975.

AMERICAN NUCLEAR SOCIETY
Program Chairman, Werner K. Kern, IRD, Space Nuclear Systems, F 309, U. S. Atomic Energy Commission, Washington, D.C. 20545.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

FOURTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Rio de Janeiro, Brazil, Oct. 23–29, 1977.
President Elect: Prof. Dr. Nicola C. Caminha; *General Secretary:* Dr. Durval Couto. Av. Churchill, 97-50 and-LC-39 20,000 Rio de Janeiro, Brazil.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

TWELFTH INTER-AMERICAN CONGRESS OF RADIOLOGY
President, Dr. Publio Vargas Pazzos, Casilla 5227, Guayaquil, Ecuador. Meeting in Quito, Ecuador, 1979.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Manuel Viamonte, Jr., M.D., Mt. Sinai Medical Center, 4300 Alton Rd., Miami Beach, Fla. 33140.
Secretary, Gaston Morillo, M.D., Jackson Memorial Hosp., 1700 N.W. 10th Ave., Miami, Fla. 33152.
Counselor for the United States, J. A. del Regato, M.D., Univ. of So. Fla. College of Medicine, 13000 No. 30th St., Tampa, Fla. 33612.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Secretary-General, P.O. Box 14, 79 Lauriston Place, Edinburgh BHI IDB, Scotland.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Secretary, Dr. Buenaventura U. V. Angtuaco, Congress Secretariat Headquarters, 2161 Taft Ave., Manila, Philippines. Meeting: Manila, Philippines, Nov. 10–14, 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lawrence E. Fetterman, 1720 Springhill Ave., Suite 201, Mobile, Ala. 36604. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY, CHAPTER ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

AMERICAN OSTEOPATHIC COLLEGE OF RADIOLOGY
Annual Scientific Meeting: Americana Hotel, Bar Harbor, Fla. Program Administrator: Lawrence J. Jordan, D.O., Suite 21, Ponce American Bldg., 4601 Ponce de Leon Blvd., Coral Gables, Fla. 33146.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Irwin M. Freundlich, 1501 N. Campbell Ave., Dept. Radiol., Tucson, 85724. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Arch W. Templeton, Univ. of Kansas Med. Center, 39th and Rainbow Blvd., Kansas City, Kansas 66103. Annual Meeting.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Marshall J. Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on fourth Thursday evenings during the academic year at a

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Maj. Marco J. DiBiase, M.D., Radiology Service, 130 Station Hosp., APO N. Y. 09102. Next Meeting: Berlin, March 14–16, 1975.

Blockley Radiological Society
Secretary-Treasurer, Dr. William H. Green, 616 East 24th St., Chester, Pa. 19013.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Ralph Brancaccio, 7901 4th Ave., Brooklyn, N. Y. 11209. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. George J. Alker, Jr., E. J. Meyer Mem. Hosp., Buffalo, N. Y. 14215. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. Duane J. Gillum, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Alfred Brettner, Auburn Mem. Hosp., Auburn, N. Y. 13021. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Daniel E. Lewis, 309 East State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, March, and May at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer Dr. E. Jerome Schoolar, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harold J. Lasky, 55 E. Washington St., Suite 1735, Chicago, Ill. 60602. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Charles M. Greenwald, 7007 Powers Blvd., X-Ray Dept., Parma, Ohio 44129. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. John Pettigrew, Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Robert R. Burns, 1400 Stemmons Ave., Dallas, Tex. 75208. Meets the 3rd Monday of every month at 6:30 P.M., at the Airport Hotel, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Ekkehard S. Schubert, Wilmington Med. Center, P. O. Box 1951, Wilmington, Del. 19899.

East Bay Radiological Society
Secretary-Treasurer, Dr. Robert L. Stein, 43 Dormidera Ave., Piedmont, Calif. 94611. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary. Dr. Paul J. Popovich, 1350 S. Hickory St., Melbourne, Fla. 32901. Meets twice annually, in the spring with the Annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Herbert Johnson, Davis Blvd., Tampa, Fla. 33606. Meets on 4th Thursday of the months January, March, May, October, and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Jay I. Rozen, Suite 216, 6400 Prospect, Kansas City, Mo. 64132. Meets 5 times a year.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary, Dr. David C. Hillman, P. O. Box 610544, North Miami, Fla. 33161. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Sidney B. W. Wong, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Robert D. Dooley, Hinsdale Med. Center, Hinsdale, Ill. 60521. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John A. Knote, Home Hosp., Lafayette, Ind. 47902.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Dale L. Roberson, 1948 First Ave., NE, Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific session is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

KENTUCKY CHAPTER OF ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph Infirmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Harry L. Stein, North Shore Univ. Hosp., 300 Community Dr., Manhasset, N. Y. 11030.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Isaac Sanders, 1429 Valley View Rd. 13, Glendale, Calif. 91202. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference. Century Plaza Hotel, Los Angeles, Calif.

LOUISIANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

LOUISIANA-TEXAS GULF COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Arnold G. Manske, P.O. Box 3008, Port Arthur, Tex. 71670.

MAINE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Peter E. Giustra, Department of Radiology, Penobscot Bay Medical Center, Rockland, Maine 04841. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

MASSACHUSETTS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Joseph T. Ferrucci, Jr., Massachusetts Gen. Hosp., Boston, Mass. 02114.

MEMPHIS ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Thomas P. Coburn, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38163. Meets first Monday Oct. through May at the University Club.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

MICHIGAN RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Francis P. Shea, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

MICHIGAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Marvin E. Goldberg, Box 292, Mayo Memorial Health Sciences Center, Minneapolis, Minn. 55455.

MISSISSIPPI STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

MISSOURI RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

MONTANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

NEBRASKA CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert Bodmer, Suite 622 Doctors Bdg., Omaha, Neb. 68131. Meets the third Wednesday of the month, Oct. through March, at the Clarkson Hosp., Omaha, Neb. and the fourth Friday of April at the Lincoln Country Club, Lincoln, Neb.

NEVADA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

N.E. SOCIETY FOR RADIATION ONCOLOGY
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Melvin E. Clouse, 185 Pilgrim Rd., Boston, Mass. 02115. Meets the third Friday of Oct. and Nov., and third Thurs. of each month. Jan. through May, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass. at 3:00 P.M. Annual Meeting: May 20, 1976.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

NEW MEXICO SOCIETY OF RADIOLOGISTS, CHAPTER OF ACR
Secretary, Dr. W. M. Jordan, 1100 Central Ave. S.E., Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

NEW ORLEANS RADIOLOGICAL SOCIETY, INC.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference.

NEW YORK ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Thomas C. Beneventano, 111 East 210th St., Bronx, N. Y. 10467. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., April 22–24, 1976. A.M.A. Cat. I credit. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

NEW YORK STATE CHAPTER OF ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 6 Secor Dr., Port Wash., N. Y. 11050.

NORTH AMERICAN SOCIETY OF CARDIAC RADIOLOGY
Secretary-Treasurer, Dr. Erik Carlson, Univ. Calif., San Francisco, Calif. 94143.

NORTH CAROLINA CHAPTER OF ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

NORTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Marshall Landa, P. O. Box 6001, Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Donald R. Morton, Dept. of Radiology, St. Clare's Hosp., Schenectady, N. Y. 12304. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. John D. Earle, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Patrick J. Grinsell, 1207 Fairchild Ct., Woodland, Calif. 95695. Meets on the fourth Monday, Sept. through May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Richard W. Siders, 421 Michigan, Toledo, Ohio 43624. Meets 3rd Wednesday from September to May.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. James Farmer, Cleveland, Ohio.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. William E. Danko, 2601 E. Chapman, Orange, Calif. 92667. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ray F. Friedman, 3324 S. W. 44th St., Portland, Ore. 97221. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Kenneth D. Moores, 1118 9th Ave., Seattle, Wash. 98101. Annual Meeting: Benson Hotel, Portland, Ore., May 7–9, 1976.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Joseph A. Marasco, Jr., St. Francis General Hosp., Pittsburgh, Pa. 19141.

Philadelphia Roentgen Ray Society
Secretary, Dr. Marvin E. Haskin, 230 N. Broad St., Philadelphia, Pa. 19102. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Klaus M. Bron, Presbyterian-Univ. Hosp., Pittsburgh 15213. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary, Dr. Gerald L. Baker, 85 Jefferson St., Hartford, Conn. 06106. Meets in Oct., Jan., March, and June.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Fred M. Palace, 11 Dale Dr., Morristown, N.J. 07960. Monthly meetings in East Orange and Trenton, N.J. Semi-Annual Meeting in Nov.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

Radiological Society of Southern California
Secretary-Treasurer, Dr. A. Franklin Turner, LAC-USC Medical Center, Room 3418, 1200 North State St., Los Angeles, Calif. 90033. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Frederick S. Vines, 11830 Rothbury Dr., Richmond, Va. 23235. Meets third Thursday of each month at the Aberdeen Barn.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, Rochester General Hosp., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 21–23, 1975.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

San Diego Radiological Society
President, Dr. Donald J. Fleischli, 7849 Fay Ave., La Jolla, Calif. 92037. Meets the first Wednesday of each month Sept. through June at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. Robert L. Koch, 3838 California St., Suite 106, San Francisco, Calif. 94118.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section of Radiology, National Medical Association
Secretary, Dr. Ruby Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broadway, Baltimore, Md. 21205. Annual meeting: Marriott Hotel, Atlanta, Ga., Sept. 28–29, 1975.

SOCIETY OF GASTRO-INTESTINAL RADIOLOGISTS
Secretary-Treasurer, Dr. John R. Amberg, University Hospital, 225 W. Dickinson St., San Diego, Calif. 92103.

SOCIETY OF NUCLEAR MEDICINE
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual Meeting.

SOUTH BAY RADIOLOGICAL SOCIETY
Secretary, Dr. Robert Mindalzun, Valley Radiologists, Inc., 100 O'Connor Dr., San Jose, Calif. 95128. Meets the 2nd Monday of each month at the Santa Clara County Medical Society Bldg., 700 Empey Way, San Jose, Calif.

SOUTH CAROLINA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

SOUTH COAST RADIOLOGICAL SOCIETY CHAPTER OF ACR
Secretary-Treasurer, Dr. Richard T. Love, Santa Barbara Med. Clin., 215 Pesetas Lane, Santa Barbara, Calif. 93110. Meets fourth Tuesday of alternate months, Sept., Nov., Jan., March and May.

SOUTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

SOUTHERN CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

SOUTHERN RADIOLOGICAL CONFERENCE
Secretary-Treasurer, Dr. J. W. Maxwell, P.O. Box 2144 Mobile Ala. 36601. Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 30–Feb. 1, 1976.

TENNESSEE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Jerry W. Grise, Methodist Hosp., 1265 Union Ave., Memphis, Tenn. 38104. Meets annually at the time and place of the Tennessee State Medical Association meeting.

TEXAS STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bdg., San Antonio, Tex. 78205.

THE FLEISCHNER SOCIETY
Secretary, Dr. E. Robert Heitzman, State University of New York Upstate Medical Center, Syracuse, N. Y. 13210. Annual Meeting: St. Francis Hotel, San Francisco, Calif., May 28–31, 1975.

TRI-STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Alfred Lessure, Welborne Hosp., Evansville, Ind. 47714. Meets bimonthly on the third Wednesday at Arco Hospitals

UPPER PENINSULA RADIOLOGICAL SOCIETY
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

UTAH STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Patrick D. Lester, Dept. Radiol., University of Utah Medical Center, Salt Lake City, Utah 84112. Meets quarterly at Holy Cross Hospital.

VERMONT RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

VIRGINIA CHAPTER OF ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 3500 Kensington Ave., Suite 2-A, Richmond, Va. 23221.

WASHINGTON, D. C., CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

WASHINGTON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Donald J. Hesch, 3216 N.E. 45th Place, Seattle, Wash. 98105.

WEST VIRGINIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

WESTCHESTER COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Leonard Cutler, 16 Guion Place, New Rochelle, N. Y. 10802. Meets on second Tuesday of October, December, February and May.

WISCONSIN RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. June Unger, Wood VA Hosp., Wood, Wis. 53193. Meets twice a year, May and September.

WISCONSIN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wis. 53706. Meets quarterly.

WYOMING RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

MEXICO, PUERTO RICO AND CENTRAL AMERICA

ASOCIACIÓN COSTARRICENSE DE RADIOLOGÍA
Secretary, Dr. Rafael Umaña-Umaña, Apartado 8-5340, San José, Costa Rica.

ASOCIACÓN DE RADIÓLOGOS DE CENTRO AMERICA Y PANAMÁ. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

SOCIEDAD DE RADIOLOGÍA DE SALVADOR
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

SOCIEDAD DE RADIOLOGÍA DE GUATEMALA
Secretary, Dr. Carlos E. Escobar, 9ª, Calle A 0–05, Zona 1, Guatemala

SOCIEDAD MEXICANA DE RADIOLOGÍA, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

SOCIEDAD RADIOLÓGICA PANAMEÑA
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

SOCIEDAD RADIOLÓGICA DE PUERTO RICO
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

BRITISH COMMONWEALTH OF NATIONS

ASSOCIATION OF RADIOLOGISTS OF THE PROVINCE OF QUEBEC
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

BRITISH INSTITUTE OF RADIOLOGY
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England.

CANADIAN ASSOCIATION OF PHYSICISTS, DIVISION OF MEDICAL AND BIOLOGICAL PHYSICS
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

CANADIAN ASSOCIATION OF RADIOLOGISTS
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St. Catherine St. West, Montreal 107, Que., Canada. Thirty-eighth Annual Meeting: Toronto, Ont., May 11–16, 1975.

EDMONTON AND DISTRICT RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada.

Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, P. D. Thomson, 28 Portland Pl., London, W1N 4DE.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. José Marcos Pires de Oliveira, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

Sociedad Argentina de Radiología
Secretary-General, Dr. Alba de la Torre, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

Sociedad Colombiana de Radiología
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Luis Blum, Casilla 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiología
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
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Société Européenne de Radiologie Pédiatrique
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Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 730, Paris Cadet 15, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

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Nederlandse Vereniging voor Radiologie
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Scandinavian Radiological Society
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Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
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Africa

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Honorary Secretary, C. A. Beetlestone, M.B., B.S., D.M.R.D., Dept. Radiol., University College Hosp., Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. A. Visser, P. O. Box 8850, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

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ROENTGEN DIAGNOSIS

ABDOMEN

HOARE, A. M. Comparative study between endoscopy and radiology in acute upper gastrointestinal haemorrhage. *Brit. M. J.*, Jan., 1975, *1*, 27–30. (From: King's College Hospital, London S. E. 5, England.)

This study describes 158 patients with acute upper gastrointestinal hemorrhage admitted to the King's College Hospital in London. The admissions policy at that hospital was such that 1 group of admitting physicians had a policy of immediate endoscopy, and the others requested endoscopy only if the emergency barium meal examination findings were negative. Both groups used the same surgeons and surgical criteria throughout the study.

The endoscopy group consisted of 53 patients in all of whom examination was performed within 48 hours of admission and usually within 12 hours. In 43, positive evidence of the site of bleeding was found.

Where roentgenologic examination was employed in the 105 remaining cases, a firm localization of bleeding site was made in only 39 cases. Additional findings such as hiatus hernia or deformed duodenal bulb were seen in others but were not included in the bleeding-site statistics.

The unusually high incidence of positive diagnosis in the endoscopy group was probably due to performance of endoscopy early in the admission and to the group being limited to patients with proved hematemesis. Of the 21 patients in the roentgenologic group who subsequently had endoscopy performed more than 48 hours after admission, a diagnosis was made only in 12.

The author feels that if roentgenology is performed before endoscopy, it delays the endoscopic examination until barium has left the stomach completely, after which the chance of a positive endoscopic diagnosis is considerably reduced. The disappointing yield of the roentgenographic examination stems from its inability to portray superficial mucosal erosions. On the basis of these findings and those of similar studies, the author recommends prompt emergency endoscopy in patients with acute upper gastrointestinal bleeding.—*R. H. Troupin, M.D.*

GYNECOLOGY AND OBSTETRICS

ROUSSEAU, J., FENTON, J., and MATHIEU, G. Épithéliomas invasifs du col utérin: résultats du traitement de 1962 cas traités à la Fondation Curie de 1956 à 1967. (Invasive carcinoma of the cervix: results of treatment of 1962 cases at the Curie Foundation from 1956 to 1967.) *Bull. du cancer*, Oct.–Dec., 1974, *61*, 457–472. (Address: Dr. J. Rousseau, Fondation Curie-Institut du Radium, 26, rue d'Ulm, 75231 Paris Cedex 05, France.)

Cervical carcinoma can be treated with satisfactory, but not yet excellent results, benefiting from close co-ordination of surgery and radiation therapy treatment.

Five year survival rates were available for 1962 women treated during a 12 year period. By international staging there were 28 per cent Stage I, 41 per cent Stage II, 27 per cent Stage III, and 4 per cent Stage IV under consideration. Endotherapy was by radium by the method of Regaud. External radiation therapy was generally delivered by 4 rectangular ports by 600 kV x-ray or telecobalt.

Stage I was treated by either radium application followed by surgery, or radium application plus moderate external beam followed by surgery, or pure external and internal radiation therapy. Stage II was treated by either external and internal radiation therapy followed by surgery, or by pure radiation therapy by both modalities. Stages III and IV received external beam and, when possible, internal radium.

The over-all 5 year cure rate in the series was 53 per cent, improving somewhat in the latter years of treatment.

Stage I rate was 81 per cent. This became 88 per cent in those cases in which the cervix was negative after irradiation and the lymph nodes not involved, and reduced to 62 per cent for histologically proved positive lymph nodes.

Stage II rate was 55 per cent survival at 5 years. However, in patients with only slight parametrial or vaginal involvement the rate was 71 per cent, and with extensive involvement 41 per cent. "Slight" Stage II with negative lymph nodes had 81 per cent rate.

Stage III rate was 30 per cent. It was 40 per cent if only 1 parametrium was involved and the intravenous urogram was normal; 24 per cent if there was bilateral parametrial involvement; and 7 per cent when ureters were compressed on urography.

Stage IV survival rate was only 10 per cent, emphasizing the need for earlier detection.—*Alan E. Oestreich, M.D.*

GENITOURINARY SYSTEM

DALINKA, MURRAY K., LALLY, JAMES F., RANCIER, LEE F., and MATA, JULIO. Nephromegaly in hemophilia. *Radiology*, May, 1975, *115*, 337–340. (Address: Murray K. Dalinka, M.D., Department of Radiology, Thomas Jefferson University Hospital, 11th and Walnut Streets, Philadelphia, Pa. 19107.)

Hematuria is a well recognized manifestation of the generalized bleeding diathesis in the hemophiliac patient. Various renal abnormalities consequent to

this urinary tract bleeding have been detailed previously in the literature. Some of these abnormalities consist of: obstructive uropathy secondary to calculi or blood clots; calyceal defects resembling papillary necrosis; bilateral renal enlargement secondary to numerous cortical cysts; unilateral renal enlargement secondary to intrarenal hematoma; and amyloidosis of the kidneys.

Changes in renal size have been noted often as an incidental finding in hemophiliac patients and renal enlargement without obstruction is not an uncommon finding.

The authors reviewed the urograms on 12 patients with hemophilia seen during the past 12 years and noted that renal enlargement was described in 4 cases. Three of these cases also showed a pseudotumor in an iliac wing. The relationship of the pseudotumors of the ilium to the urinary tract findings was not apparent. The 1 factor common to all of their patients with nephromegaly is multiple transfusions. This has also been noted in patients with sickle cell anemia or thalassemia. The cause of the nephromegaly has not been explained.—*Donald N. Dysart, M.D.*

Nervous System

Dunnick, N. Reed, and Castellino, Ronald A. Arteriographic manifestations of ganglioneuromas. *Radiology*, May, 1975, *115*, 323–328. (Address: Ronald A. Castellino, M.D., Department of Radiology, Stanford University Medical Center, Stanford, Calif. 94305.)

Ganglioneuromas are rare, benign tumors arising from sympathetic ganglia, and are usually found in the neck, posterior mediastinum or retroperitoneal space. The roentgenographic findings are those of a soft tissue mass, often ovoid in shape, which at times contains mottled calcifications. They may attain sufficient size to displace adjacent organs, and occasionally may present as a "dumbbell tumor" with neuroforaminal widening and spinal cord compression. Ganglioneuromas are more common in females and 60 per cent of the patients are under the age of 20. Although patients are usually asymptomatic; chest pain, abdominal distention, ptosis and altered gait have been reported. Also, hormonally active benign ganglioneuromas may cause diarrhea or episodic hypertension, sweating and flushing suggestive of a pheochromocytoma.

The authors present the angiographic findings of 4 patients with ganglioneuromas. In each of the authors' 4 cases, a large vascular mass was demonstrated on arteriography. The vessels supplying the tumors were variable and dependent on the location of the mass. Stretching and displacement of the adjacent opacified vessels were helpful in defining the extent of the tumor. Abundant neovascularity was present in each case. The neovascularity was not typical of that seen in the usual vascular malignant tumor, in that no sudden changes in arterial caliber, arteriovenous shunting, contrast laking or puddling, or fine, bizarre vessels were seen. Instead, the appearance was more like extremely serpiginous and meandering vessels, tending to cluster in nodular areas, with adjacent avascular lakes. Arterial encasement was not noted on any of the angiograms. Arterial encasement, however, may be seen on angiograms of neuroblastomas. Nevertheless, at present the authors do not believe it is possible to distinguish with confidence the benign ganglioneuroma from the malignant neuroblastoma on the basis of the arteriographic findings.—*E. J. Schoolar, M.D.*

Skeletal System

Wiltse, Leon L., Widell, Eric H., Jr., and Yuan, Hansen A. Chymopapain chemonucleolysis in lumbar disk disease. *J.A.M.A.*, Feb., 1975, *231*, 474–479. (Address: Leon L. Wiltse, M.D., 2840 Long Beach Boulevard, Suite 410, Long Beach, Calif. 90806.)

"Chemonucleolysis" to indicate a chemical dissolution of the nucleus pulposus by intradiskal injection of the enzyme, chymopapain, has had a worldwide use in more than 15,000 cases to date. Since 1968 the Orthopedic Department of Long Beach Memorial Hospital Medical Center, Long Beach, California and the Department of Orthopedic Surgery, College of Medicine, University of California, have performed over 1,200 chemonucleolysis. The experience in the first 500 cases, now over 3 years post injection, were reviewed.

The enzyme, chymopapain, acts on the central contents of the intervertebral disk by hydrolyzing and dissolving the noncollagenous protein that interconnects long chain mucopolysaccharides and is highly specific for the nucleus pulposus as compared to the surrounding fibrous tissue of the annulus. Toxicologic studies have been performed on rabbits, dogs and monkeys. Doses up to 100 times greater than required to consistently dissolve the nucleus pulposus were well tolerated when injected intravenously, intradiskally and epidurally. Intrathecal injections can be toxic but injection at the L3 level requires a dose 3 times that of injection at the cisternal level to affect the same toxic effect. Other studies utilizing injection of sub-lethal intrathecally administered chymopapain revealed no evidence of arachnoiditis anywhere along the spinal cord or about the brain in dogs killed up to one year post injection. Massive epidural doses caused no neural adhesions, dural thickening nor epidural scarring. This is extremely important because in about 1 in 4 humans there is leaking of chemopapain into the extradural space at the time of injection.

The same criteria were used in selecting patients for chemonucleolysis as have traditionally been used in deciding on laminectomy, with the following ex-

ceptions or contraindications: history of allergy to papaya or meat tenderizers; presence of a rapidly progressing neurological defect; neurological deficit producing bowel or bladder disturbance; complete block on myelogram; and any possibility of spinal cord tumor or pregnancy. No patient who would not also have been considered for laminectomy received chymopapase injection. Each candidate was thoroughly examined including roentgenograms, myelogram, spinal fluid analysis and cytological testing. Diskography was done on all cases as part of the injection procedure.

Diskography at the level considered for chymopapain injection is done at the time of chemonucleolysis to insure proper needle placement, to avoid injecting fairly normal appearing disks as seen on diskography, and very rarely to demonstrate a direct communication between the nucleus pulposus and subarachnoid space. Iothalamate has been shown to be considerably less toxic than diatrizoate when given intrathecally and therefore iothalamate was used for all diskograms.

An especially equipped roentgenographic room, utilizing image intensified fluoroscopy and a C-arm fluoroscopy unit to afford biplane fluoroscopic control for needle insertion, was utilized with the patient placed in the left decubitus position. Six to 8 mg. of chymopapain were injected by a 15 cm. 22 gauge spinal needle threaded through a previously placed 10 cm. 18 gauge disposable spinal needle with the tip of the longer needle centered within the disk. Six to 8 mg. of chymopapain were then injected into each appropriate disk space. Following the procedure the patients were allowed to walk as soon as they felt able. The average hospital stay was 5 days.

During the procedure, done under local anesthesia, an anesthesiologist and an especially trained nurse were available in the event that anaphylaxis occurred. This has been observed in approximately 1 per cent of the patients world-wide. There were 9 cases of anaphylaxis in this entire series of 1,200 patients. Fifteen others developed a rash, usually associated with a moderate drop in blood pressure, and 11 patients reported delayed urticaria at 9 to 14 days post injection. In the last 600 cases the patients were pre-medicated with 250 mg. hydrocortisone and 50 mg. of Benadryl and only 1 case each of moderately severe and very mild anaphylaxis occurred. Three patients suffered foot drop and 1 continues to have some dorsiflexor weakness. There have been no disk space infections, but there was 1 case of diskitis occurring after diskography.

Seventy-four per cent of the patients stated they achieved their optimum results within the first month following injection. Seventy-two per cent of the patients undergoing 1 level injection were given excellent, good, or fair ratings while 80 per cent of those with 2 level injections and 77 per cent of those with 3 level injections were given similar ratings. Statistics were not compiled but it is the feeling of the authors that chances of successful relief of pain are at least as good if an extruded fragment is present prior to treatment. After chemonucleolysis, disk space narrowing occurs rapidly. Careful follow-up examinations indicate that the patient who has had several previous back operations, especially if he has had a spinal fusion, does not often profit from chemonucleolysis.

Of the 112 cases of chymopapain therapy, 56 patients subsequently required surgery and there was good follow-up information available on 30 patients. There was a single case of sequestered disk fragment found at subsequent surgery. In 17 of 25 levels a definite hard bulge of annulus was described and this combined with disk space narrowing was thought by the operating surgeon to be causing nerve root pressure. At 5 levels spinal stenosis was diagnosed and at 5 other levels a previously injected disk contained a normal appearing nucleus pulposus indicating the enzyme either failed to act or the needle was not accurately positioned prior to injection. It is the authors' belief that chances of relieving pain by laminectomy after chemonucleolysis is as good as if chemonucleolysis had not first been performed.

One hundred and twenty-five patients randomly selected, who originally had definite neurological deficits including diminished deep tendon reflexes and motor weakness of sensory deficits, were re-evaluated neurologically 1 year post injection. Sixty-one per cent of those with initially absent deep tendon reflexes were rated normal, 84 per cent with gross muscle weakness reverted gradually over several months and 77 per cent with sensory deficits reverted within 3 weeks.—*John T. Underberg, M.D.*

McALLISTER, V. L., KENDALL, B. E., and BULL, J. W. D. Symptomatic vertebral haemangiomas. *Brain*, March, 1975, *98*, 71–80. (From: The National Hospital for Nervous Diseases, Queen Square, London WC1, England.)

The authors point out that the majority of vertebral hemangiomas are silent clinically and exhibit characteristic vertical striations in involved vertebral bodies. Nine cases are discussed to point out that neurological complications may occur and the roentgenological changes may be atypical. Most spinal hemangiomas are in the thoracic region but occasionally they occur in the cervical and lumbar areas. Expansion of an involved vertebra can lead to narrowing and deformity of the spinal canal. Compression fracture is rare as is extradural hematoma secondary to the hemangioma. Occasionally plain roentgen rays are negative and although myelography can be helpful if there is tumor compression of the theca, spinal angiography can confirm the diagnosis. It is pointed out that vertebral collapse is rare in hemangiomas and differentiation from Paget's disease is usually not difficult. Angiography is done by the intercostal arterial route.

It is pointed out that laminectomy is hazardous because of the possibility of hemorrhage, but the danger is lessened if spinal angiography defines the arterial supply preoperatively. Embolism or ligation can be employed to reduce the vascularity also. Radiation therapy is discussed and this has produced good results in vertebral hemangiomata in the past.

The authors summarize that most of the hemangiomata, which became evident clinically in the cases which they described, did fill because of spinal cord or nerve root compression from extradural tumor, bony expansion or a combination of these. The classical roentgenologic pattern of accentuated vertical striation or honeycomb pattern is easily recognized but atypical features such as apparent pedicle erosion, paravertebral soft tissue mass and bone expansion can occur. The great importance of preoperative spinal angiography is stressed.—*Robert P. Andrews, M.D.*

Tenner, R. Röntgenologische Veränderungen bei der Materialwanderung am künstlichen Hüftgelenk. (Roentgenological changes due to displacement of alloplastic materials of artificial hip joints.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1974, *121*, 591–597. (Address: AK. St. Georg, 2 Hamburg 1, Lohmühlenstrasse 5, Germany.)

Migration of alloplastic material of artificial hip joints occurs when a disproportion of weight and load capacity exists. Pathophysiologically as well as roentgenographically 3 types of migration can be differentiated: decreased load capacity of bone; inadequate mechanical anchoring of the alloplastic material; and a combination of both.

Decreased load capacity of bone is most frequently caused by infection, whereby not only osteolysis but also sclerotic bone, which is softer than normal bone, open the way for migration. Frequently the infection-related migration of the alloplastic material is combined with loosening of the material. Osteoporosis is another although rare cause of migration due to reduced load capacity. Insufficient mechanical anchoring is caused by a too small contact area between prosthesis and bone. Roentgenographically, the socket projects either too far laterally or protrudes centrally. A combination of inadequate load capacity and mechanical anchoring is usually caused by infection superimposed on an inadequately anchored prosthesis. Artificial hip joints that have been replaced previously because of infection are prone to develop this complication. The combination of osteoporosis and insufficient mechanical anchoring is rare.

The direction of migration depends on the contour of the pelvis and femur, construction of the prosthesis, surgical technique, and acting forces. The alloplastic material migrates craniomedially if the inner cortex of the pelvis is destroyed by infection or surgical manipulation, and craniolaterally if the mechanical anchoring is inadequate laterally. Rotation of the socket can also occur. In the femur, the prosthesis migrates caudad. If the femoral shaft is affected by osteomyelitis, the stem can protrude through the destroyed cortex either medially or laterally.—*Peter F. Winter, M.D.*

Huvos, Andrew G., and Marcove, Ralph C. Adamantinoma of long bones: a clinicopathological study of fourteen cases with vascular origin suggested. *J. Bone & Joint Surg.*, March, 1975, *57-A*, 148–154. (Address: Andrew G. Huvos, M.D., 1275 York Avenue, New York, N. Y. 10021.)

The authors review 14 cases of adamantinoma seen at the Memorial Sloan-Kettering Cancer Center, between 1920 and 1973, all of which were histologically proved. Malignant hemangioendothelioma was carefully excluded. The tumors have a cellular pattern with vascular spaces adjacent to, and merging with, solid tumor cells which suggests angioblastic proliferation in addition to the monophasic epithelial pattern. Two cases had tissue cultures which suggested a mesenchymal origin, rather than epithelial or synovial, which have been postulated in previous cases.

Twelve tumors were in the tibia and 2 in the humerus. The age range was 13–67, equally divided as to sex. The usual roentgen-ray appearance was a sharply defined eccentric defect in the diaphysis of the tibia with marginal sclerosis. The cortical bone loss over the lesion was typically "saw-tooth." There may be an expanding honeycomb lesion with coarse trabeculae. In 6 cases there were extra cortical soft tissue extensions. Occasionally, there is marked periosteal bone formation. The tumor may be multifocal and of greater extent than obvious from the films. The differential diagnosis includes chondrosarcoma, metastatic carcinoma, eosinophilic granuloma and angiosarcoma.

Curettage was done 11 times in 8 patients and in only 1 small lesion did this result in long-term cure. Since the tumor has a long natural course with localized disease, amputation even after recurrence was curative in 7 of 9 cases. Two patients developed pulmonary metastases, one at 2 and the other at 5 years. One case had a metastasis to the humerus from the tibia. In addition, metastases have been reported to lymph nodes and abdominal viscera. Curettage and *en bloc* resection were unreliable and amputation is considered to be the preferred definitive treatment.—*Martha Mottram, M.D.*

Duncan, Jan W., Schrantz, James L., and Nasca, Richard J. The bizarre stiff hip: possible idiopathic chondrolysis. *J.A.M.A.*, Jan., 1975, *231*, 382–385. (Address: Richard

J. Nasca, M.D., The Orthopaedic Clinic, 30 Evergreen Place at 1100 N. University, Little Rock, Ark. 72207.)

Chondrolysis is a well-recognized clinical entity with an over-all reported incidence of 8.2 per cent in slipped capital epiphysis and is infrequently found secondary to immobilization or severe trauma.

The authors, from the Department of Orthopaedics, University of Arkansas Medical Center, report 5 cases of previously undescribed idiopathic chondrolysis of the hip. Diagnosis was based on the following criteria; onset in early adolescence, painful hip and limp, progressive stiffness of the hip, narrowing of the articular cartilage space with maintenance of femoral head contour, nonspecific synovitis on biopsy, and no identifiable cause, such as chronic monoarticular arthritis, tuberculosis, fungus, pyrogenic arthritis or slipped capital femoral epiphysis.

Roentgenographically the earliest findings consisted of capsular distention and osteoporosis with the thigh held in an abducted, externally rotated position which led to pelvic obliquity on the anteroposterior roentgenogram. As the disease progressed there was narrowing of the joint space and finally there could be premature closure of the physis, subchondral cysts and osteophyte formation.

The cases involved a 13 year old boy and 4 girls, ages 11 to 13, with bilateral hip involvement in 1 patient. The patients presented with a painful hip. Laboratory work-ups, including sedimentation rates, were generally within normal limits. Ankylosis occurred in 4 of the 6 involved hip joints. In unilateral cases the authors believe immobilization in a position of function is the treatment of choice, as without immobilization ankylosis with excessive flexion results. —*John T. Underberg, M.D.*

SIM, FRANKLIN H., DAHLIN, DAVID C., and BEABOUT, JOHN W. Osteoid-osteoma: diagnostic problems. *J. Bone & Joint Surg.*, March, 1975, *57-A*, 154–159. (Address: Franklin H. Sim, M.D., 200 First Street, S. W., Rochester, Minn. 55901.)

Osteoid-Osteoma (OO) has typical clinical and roentgen findings and is surgically curable. In some patients, however, surgery fails to identify the nidus and it will not be shown on the histological examination. To determine whether such patients are cured or have persistent symptoms, 54 cases were investigated at the Mayo Clinic. All of the patients had typical pain, usually accentuated at night and relieved by aspirin. Some had swelling, tenderness or muscle atrophy, but no clinical evidence of inflammation. Forty-eight per cent of the patients had symptoms for less than 1 year. Forty-three lesions were in the lower extremities or pelvis, mainly in the femur and tibia. Seven lesions were located in the upper extremities and 4 in the spine. In cancellous bone, there was central rarefaction with surrounding sclerosis. Cortical lesions showed sclerosis with a central lucent zone in 47 and a calcified nidus in 4.

The lesions were excised and 2 were Brodie's abscesses. The other 52 had non-diagnostic pathological specimens; sclerotic bone, no inflammation and negative cultures. Of this group, 29 had relief of symptoms. Because of persistent symptoms still suggesting OO and roentgen-ray appearances compatible with this diagnosis, 18 patients had a second operation 5 months to 6 years later (average 2.5 years). Thirteen of these patients had relief of symptoms with a nidus found in 7 instances. Of the 5 who had no relief following the second operation, 1 was later found to have a parosteal osteogenic sarcoma. Even though 1 patient had had a radical *en bloc* resection, no nidus was found. A nidus was eventually found in 2 others after a third operation with relief of symptoms. The fifth patient had extensive excision of the sclerotic area in the femoral neck with grafts; no nidus was found and symptoms persisted 13 months later. Of the 5 patients having no relief following the initial surgery and refusing further operative procedure, 1 was found to have a gastric carcinoma as the cause of pain; one had sclerotic bone and probably a degenerative cyst; the other 3, in spite of *en bloc* excisions, had persistent symptoms.

The 36 patients who had no nidus found at surgery (29 initially, 6 with a second operation and 1 with a third) could have had an extremely small nidus which was overlooked by the surgeon and pathologist or the specimen may have been damaged in surgery so as to obscure the nidus. *En bloc* resection was shown to be the most successful procedure with relief of symptoms in 38 of 50 patients and curettage gave relief in 7 of 22.

Since symptoms persist whenever the nidus is not completely removed, the authors urge verification of the lesion by roentgen ray after *en bloc* resection. Serial blocks may be necessary to identify a small nidus.—*Martha Mottram, M.D.*

LARSSON, SVEN-ERIK, LORENTZON, RONNY, and BOQUIST, LENNART. Giant-cell tumor of bone: a demographic, clinical, and histopathological study of all cases recorded in the Swedish Cancer Registry for the years 1958 through 1968. *J. Bone & Joint Surg.*, March, 1975, *57-A*, 167–173. (Address: Dr. Sven-Erik Larsson, University of Umeå. 901 85 Umeå, Sweden.)

This is a report of all giant-cell tumors (GCT) recorded in the Swedish Cancer Registry for the years 1958–1968. Seventy-five cases were registered and 73 were available for review. The authors found that 53 were true GCT and 20 were variants. Seven of the latter were reclassified as non-osteogenic fibroma, 6 as aneurysmal bone cysts and others as: fibroxanthomatous giant-cell tumor, reparative

granuloma, pigmented villonodular synovitis, benign chondroblastoma, myeloma or undifferentiated sarcoma.

For the true GCT there was good correlation between the initial and final histopathological diagnosis as regards malignancy. Out of 5 initially considered benign, only 4 were reclassified Grade III and one initially considered malignant was determined to be Grade I. Of the GCT, 11.3 per cent were malignant.

The incidence of GCT was 7.8 per cent of all reportable bone tumors (not including myeloma). There was a higher incidence in the urban population than in rural Sweden. There were 3 age peaks: 15–25, 30–40 and 55–70. Twelve patients were under 20 and 17 of these were males. The skeletal distribution was not unusual, with the greatest number located around the knee and this area also had the highest recurrence rate.

The signs, symptoms and roentgen findings did not vary from the usual. It was noted that 3 of the GCT less than 2 cm. in size recurred after primary treatment. There was no correlation between tumor size and recurrence rate, but tumors extending into the soft tissues showed a higher rate of recurrence than those confined to bone. Primary therapy varied but resection was most successful. Results were related to the type of treatment and not to the histopathological grade of the tumor. The recurrence rate was 42 per cent. Curettage, alone, had a recurrence rate of 28 per cent and resected tumors 14 per cent.

There were 9 deaths, 6 of which were due to the malignant course of the tumor; 2 were due to other malignancies and 1 to an abscess in the region of the tumor.

Curettage and grafting are advocated only for patients under 25. Resection is urged whenever possible as the initial treatment in all others. When the soft tissues are involved, either radical resection or amputation should be the primary treatment.—*Martha Mottram, M.D.*

BLOOD AND LYMPH SYSTEM

SPIERS, A. S. D., BAIKIE, A. G., GALTON, D. A. G., RICHARDS, H. G. H., WILTSHAW, E., GOLDMAN, J. M., CATOVSKY, D., SPENCER, J., and PETO, R. Chronic granulocytic leukaemia: effect of elective splenectomy on the course of disease. *Brit. M. J.*, Jan., 1975, *1*, 175–179. (Address: Dr. A. S. D. Spiers, M. R. C. Leukaemia Unit, Royal Postgraduate Medical School, London W12 OHS, England.)

Fifty years ago the median survival from diagnosis of untreated patients with chronic granulocytic leukemia was 19 months. More recent studies following treatment with radiation therapy and bulsulphan or with dibromannitol indicate that current therapy improves the quality of life and extends its duration about 20 months. In a study of the Medical Research Council, over 70 per cent of the patients died with uncontrolled leukemia after the disease had undergone metamorphosis to a more refractory acute or subacute stage.

Despite some recent improvements, once metamorphosis has occurred treatment is not strikingly effective in prolonging life. The authors previously have suggested elective splenotomy early in the course of the disease which might prolong survival by deferring metamorphosis. Patients with chronic granulocytic leukemia who have had particularly long survival, seem to include an unusual proportion with a relatively slight splenomegaly before treatment or prolonged regression of splenic enlargement after treatment, and a few patients who have undergone splenectomy for a variety of reasons have had hematological remissions without chemotherapy. Pharmacodynamic or immunological factors may make the spleen a particularly suitable site for the emergence of aberrant clones. If so, its removal might be expected to postpone metamorphosis although not prevent it.

The authors report their experience 9 years following the inception, with a study involving 26 patients between the ages of 16 and 64. Patient acceptability for the study was based upon an unequivocal diagnosis of chronic granulocytic leukemia still in its chronic phase, a satisfactory clinical and hematological response to initial treatment, willingness to undergo splenectomy and absence of other illness making for poor surgical risk in patients below the age of 65 years. Prior to surgery, an attempt was made to reduce the platelet count to less than 200×10^9 per liter. The Ph chromosome was identified and bone aspiration done to exclude the presence of myelofibrosis.

There were no operative or postoperative deaths. Deep venous thrombosis and pulmonary embolism occurred in 1 patient, pleural effusion, peritonitis and paralytic ileus in another, and sterile wound discharge in a third. A series of hematologic changes were observed with consistency in almost every patient. The total leukocyte count rose 3 to 6 times its preoperative value peaking at 24 to 48 hours and then returning spontaneously to the original value in a similar period of time; the neutrophil alkaline phosphatase level rose 5 to 10 times its preoperative value within 72 hours and remained raised for weeks or months; and the platelet count rose to a peak at 7 to 10 days to a value 4 to 6 times the preoperative value and required weeks or months to return to its former level. Clinical and hematological control after splenectomy were satisfactory so long as the disease remained in its chronic phase just as it is in patients who have not undergone splenectomy. Treatment with bulsulphan or other drugs was no more difficult than in nonsplenectomized patients; in particular, the doses required did not differ significantly. Apart from the occurrence of thrombocytosis exceeding

100×10^9 in 1 of 14 patients, the hematologic data were unremarkable. It remains uncertain if the accepted indications for resumptions of interrupted bulsulphan therapy in nonsplenectomized patients also apply to those who have undergone splenectomy.

Nineteen patients underwent splenectomy within 12 months of diagnosis. Since 21 patients remained alive at the time of the review article, the eventual median survival was unknown. Two of 5 deaths were due to intercurrent unrelated disease and 3 patients died with uncontrolled chronic granulocytic leukemia in metamorphosis; 2 further patients have undergone metamorphosis of their disease. Statistical comparison of the results in the experience of bulsulphan treated patients in the Medical Research Council study, who also had a satisfactory response to initial treatment with bulsulphan, suggested that 10 cases of metamorphosis would have been expected in the period of observation had a 4 month correction factor been introduced to compensate for any selectivity effects based upon mild symptoms and lack of hematologic deviations preceding clear-cut metamorphosis. Even with this correction, the difference is believed significant.

The mechanism whereby splenectomy might postpone the metamorphosis is unknown, but the removal of a site particularly suitable for the development of aneuploid cell clones with anaplastic properties is hypothesized. The results of the study would appear to justify its further evaluation by a controlled clinical trial. It appears that splenectomy postpones the metamorphosis from the chronic to acute phase of granulocytic leukemia. No patients have developed myelofibrosis, but the authors accept that myelofibrosis is a relative contraindication to elective splenectomy. It is not yet established whether splenectomy significantly prolongs the duration of life after metamorphosis has occurred, but the quality of life appears improved. Along with the absence of distressing symptoms of splenic origin, the patients show less need for blood and platelet transfusions. Tolerance to antileukemic drugs seems to have been improved and patients were hospitalized for shorter durations following metamorphosis.—*John T. Underberg, M.D.*

RADIATION THERAPY

CHAM, WILLIAM C., FREIMAN, ALVIN H., CARSTENS, PER H. B., and CHU, FLORENCE C. H. Radiation therapy of cardiac and pericardial metastases. *Radiology*, March, 1975, *114*, 701–704. (Address: William C. Cham, M.D., Department of Radiation Therapy, Memorial Sloan-Kettering Cancer Center, 1275 York Avenue, New York, N. Y. 10021.)

Thirty-eight patients with disseminated malignant neoplasm and cardiac metastases received external beam roentgen-ray therapy for their cardiac involvement. Neoplasms most frequently responsible were breast, lung, lymphoma, and leukemia. Clinical manifestations were determined by location and size of the tumor.

The most common manifestation was pericardial involvement with effusion. Dyspnea was the earliest and most striking symptom. Roentgenographic enlargement of the heart silhouette occurred in 30 patients, and 16 had cytology positive pleural effusion. Electrocardiographic results were non-specific, and low voltage was the most frequent abnormality. Thirty-seven patients had clinical and roentgenographic evidence of pericardial involvement. Nine also had evidence of myocardial involvement with arrhythmias and heart block. Angiocardiography was the most reliable diagnostic procedure in establishing the presence of pericardial effusion, and ultrasound was also useful. Pericardiocentesis was performed on 27 patients. Twenty had hemorrhagic effusion, 6 had serosanguineous fluid, and 1 had solid tumor around the heart. All effusions contained malignant cells.

Radiation therapy included use of opposing anterior and posterior portals, daily doses of 150–200 rads, and a total tumor dose of 2,500–3,500 rads in 3–4 weeks. Lymphoma and leukemia patients received a lower dose (1,500–2,000 rads). A significant response to treatment was based on relief of cardiac symptoms and reversal of abnormal chest roentgen ray and EKG findings. The over-all response rate was 61 per cent (23/38).

Ten patients had postmortem examinations, and most showed microscopic involvement of pericardium and epicardium. No severe late radiation changes were observed.

The cancer patient with sudden onset of tachycardia, arrhythmia or congestive failure should be suspected of cardiac metastases. An enlarged cardiac silhouette on chest roentgenogram and conduction defects and low voltage on EKG are virtually confirmatory. Ultrasonic and angiographic demonstration of effusion or pericardiocentesis establishes the diagnosis. Irradiation of cardiac metastases is worthwhile. The total tumor dose will depend on any previous radiation therapy to the mediastinum and the radiosensitivity of the tumor.—*O. D. Skinner, M.D.*

MILL, WILLIAM B. Radiation therapy in multiple myeloma. *Radiology*, April, 1975, *115*, 175–178. (Address: Division of Radiation Oncology, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.)

Prior to 1947, the only specific treatment for multiple myeloma was radiation therapy. Experience showed its effectiveness in relieving bone pain, healing pathologic fractures, decreasing the size of extramedullary "plasmacytomas," and prolonging survival in patients with extradural myeloma. After

1947, however, chemotherapeutic agents were shown to be effective in treating myeloma—melphalan and cyclophosphamide have assumed a major role in producing relief of symptoms and prolongation of survival.

The purpose of this retrospective study is to review the indications for radiation therapy in this disease and analyze its value in a group of 76 patients treated at the Mallinckrodt Institute of Radiology, Division of Radiation Oncology, between January 1, 1961 and December 31, 1970. Eleven patients presented with localized disease, the remaining 65 had disseminated myeloma.

Those patients with localized disease (solitary plasmacytoma) received doses of irradiation between 1,440 and 5,000 rads. Six patients are alive with no clinical evidence of myeloma; 3 are alive with recurrence and 2 died with tumor. It is observed that those patients receiving higher doses did better.

Patients with disseminated myeloma were treated for palliation only. Doses over 2,000 rads in 2 weeks were more effective than smaller doses and produced complete relief of pain in 58 per cent. Only 2 patients received no relief of pain. The median survival was 20 months from the time of diagnosis.

Complications from the irradiation are described. It is noted also that only 5 patients in the entire series had a second primary malignancy.

It is concluded that radiation therapy is effective in the treatment of localized plasmacytoma and doses of 4,500–5,000 rads in $4\frac{1}{2}$ to 5 weeks are recommended. In patients with disseminated involvement, the primary treatment is chemotherapy. Radiation therapy is indicated for palliation, and doses of 2,000–2,500 rads in $2\frac{1}{2}$ weeks are adequate to relieve symptoms and still preserve some bone marrow function in the irradiated area. Because the disease permeates the marrow, the entire affected bone should be treated.—*Paul M. Kroening, M.D.*

Brands, Th., and Wittig, G. Klinik und Strahlentherapie der Hämangiome und Lymphangiome: Pathologie und Histologie. (Clinical manifestations and radiotherapy of hemangiomas and lymphangiomas; pathology and histology.) *Med. Welt.*, Oct., 1974, *25*, 1697-1704. (Address: Dr. Th. Brands, Chefarzt des Radiologischen Zentralinstituts, Städtisches Krankenhaus, 7700 Singen/Hohentwiel, Germany.)

Clinical features and radiation therapy are discussed in 27 superficial and 26 subcutaneous hemangiomas and in 12 lymphangiomas. These 65 patients have been followed for the past 10 years. The angioblastomas usually appear in early infancy or at birth. They may develop almost anywhere in the body but are most frequently observed in the face and neck. There is a moderate preponderance of girls over boys.

The tumors may occasionally regress spontaneously but most often present a gradual increase in size. Therefore, aggressive radiation therapy is indicated to halt further spread. An acute emergency may also develop when a tumor in the neck presses on the trachea and the larynx, thereby causing a severe dyspnea. Fortunately, properly administered radiation therapy will bring about prompt relief.

Irradiation of the superficial hemangioma is best performed with a strontium dermaplate; single doses varying from 100 to 1,000 reps at an average of 300 reps and a total dose of 500 to 3,000 reps. Conventional radiation therapy is indicated when the tumor response remains poor. In cases with recurrence, therapy with radiocobalt is suggested with single doses varying from 50 to 300 rads at an average of 100 rads and a total dose up to 7,700 rads (or reps for treatment with Sr^{90}).—*Ernest Kraft, M.D.*

RADIOISOTOPES

Go, Raymundo T., Tonami, Norihisa, Schapiro, Rolf L., and Christie, James H. The manifestations of diaphragmatic and juxtadiaphragmatic diseases in the liver-spleen scintigraph. *Radiology*, April, 1975, *115*, 119–127. (Address: Raymundo T. Go, Department of Radiology, University of Iowa, Iowa City, Iowa 52242.)

The superior aspects of the liver and spleen normally lie in apposition to the inferior surface of the diaphragm. A variety of clinical entities may present as primary intrathoracic, diaphragmatic disease or abnormalities of sufficient magnitude to change this normal relationship. The change should be reflected in the liver-spleen scintigraph. Changes will be manifested by an exaggeration of, or a reversal of, the normal anatomical relationships of the superior margins of the liver and spleen, or by alterations in contour or axis of the organ involved.

Rectilinear scintigraphs were obtained after the intravenous injection of 4 mCi of Technetium (^{99m}Tc) sulfur colloid. The dual-5″ scintillation scanner was used primarily because the liver-spleen complex could be easily demonstrated on each scintigraph. Other advantages are the ease of obtaining opposing views of the liver-spleen, and the ability to obtain opposing transmission scintigraphs concurrently by simply inserting a 4 mCi ^{99m}Tc source in the center hole of each upper and lower probe collimators without moving the patient.

When viewing the liver-spleen scintigraph, especially the posterior, disparity in their respective positions becomes readily apparent. This may be due to cephalad or caudad displacement of either organ, and is an indication of unilateral disease. It is difficult to lateralize the organ that is displaced unless there is associated change in its contour or axis. Associated change on the chest roentgenogram may

be helpful, and a transmission scintigraph may be required. Bilateral changes may be even more difficult to appreciate without a transmission scintigraph, but are frequently heralded by "flattening" of the superior margins of both the liver and the spleen image.

It is worthwhile to compare the liver-spleen scintigraph to chest roentgenograms as a primary approach, for most cephalad displacement of either organ can be explained in this manner. Caudad displacement of either, or both, may also be explained by chest changes, but may require closer inspection of the scintigraphs for contour or axis changes of the organs, or transmission scintigraphs to define the true relationship of the displacement.—*F. C. Petty, M.D.*

UGOLOTTI, G., MAGNANI, C., and CELLA, C. La flebografia periferica mediante Tc^{99m}. (Peripheral phlebography with Tc^{99m}.) *Ann. radiol. diag.*, 1974, *45*, 123–133. (From: Istituto di Radiologia dell'Università di Parma, Italy.)

The authors report in this preliminary note the results obtained in a series of patients who were subjected to standard phlebography and then to radionuclide phlebography following the injection of Tc^{99m} pertechnetate.

To prevent uptake by the thyroid of the radionuclide, the patients were given 300 to 500 mg. of potassium perchlorate orally shortly before the examination. The amount of pertechnetate introduced varied between 0.3 cc. to 10 cc., with a concentration of the radionuclide between 10 to 200 millicuries per cc. respectively. Serial scintiphotographs of the areas under study were performed every 1 to 2 seconds in the early phase of the examination, and then at longer intervals of time to record the most significant findings. The scintillation camera employed was a Pho-Gamma III, Nuclear Chicago.

The series is still rather limited but it is felt that the following considerations on radionuclide phlebography can be presented: (1) the examination can be safely performed in patients who are allergic to the usual contrast agents or who might still suffer from an acute episode of thrombophlebitis; (2) satisfactory information is obtained on the normal or abnormal course of the vessels and on their patency; and (3) the persistence of the image of the veins on the oscilloscope allows a more detailed and unhurried evaluation of the morphology of the vessels. The standard phlebography, calling for preset times of exposure of the phlebograms, at times results in an incomplete documentation.

A disadvantage of the technique is related to the limited power of resolution of the gamma camera. In radionuclide phlebograms of the lower leg and arm, where the veins are in greater proximity one with another, the vessels would appear in the scintiphotograms as a single "trace," having lost their individual image.

Finally, it is felt that this technique should be used as a screening procedure in the selection of cases to be evaluated by standard phlebography, with particular attention brought therefore, to the areas of greater interest and importance. Similar conclusions were also drawn by L. Rosenthal in his paper which appeared in the August issue, 1966, Vol. *97*, of this JOURNAL.—*A. F. Govoni, M.D.*

CHISHOLM, G. D., SHORT, M. D., and GLASS, H. I. The measurement of individual renal plasma flows using ^{123}I-Hippuran and the gamma camera. *Brit. J. Urol.*, Dec., 1974, *46*, 591–600. (From: The Urology Unit, Department of Surgery, and Department of Medical Physics, Hammersmith Hospital, Royal Postgraduate Medical School, London, England.)

The clearance of iodine-labelled orthoiodohippurate (Hippuran) by the functioning renal parenchyma represents the effective renal plasma flow (E.R.P.F.). A method was described for the measurement of the E.R.P.F. by Ram, Evans and Chisholm in 1968, using a single injection of ^{125}I-Hippuran. The authors extended that method to measure total and individual E.R.P.F. using the gamma camera and ^{123}I-Hippuran. The data were recorded on computer compatible magnetic tape. The kidneys were localized using 40 μCi ^{197}Hg-chlormerodrin. The kidney images were positioned in the lower portion of the field to facilitate the selection of a soft tissue background region. Four hundred μCi of ^{123}I-Hippuran were injected and the data were recorded for 20 minutes and a 5 ml. blood sample was taken 15 minutes after the injection.

The radiation dose to the kidneys was about 0.07 m rads/μCi which is one-fifth the dose if ^{131}I-Hippuran was used.

Fifty patients were studied. Sixteen had equal kidney function as judged by the excretory pyelograms and normal blood urea levels, 25 had unequal renal function due either to unilateral or bilateral renal disease and normal blood urea levels and 9 had renal disease with blood urea levels between 82 and 297 mg. per cent. In the 16 normal patients the E.R.P.F. was 216 to 6.95 ml./min. In the second group the uncorrected E.R.P.F. was from 110 to 670 ml./min. The E.R.P.F. in the uremic group was 32 to 150 ml./min.

The clinical value of this technique has been in 2 main groups of patients: (a) those with asymmetrical renal disease; and (b) those requiring serial measurement of renal function such as long-term follow-up of a damaged kidney. Intra-renal blood flow and renal transit time, *i.e.*, the rate of transport of Hippuran through the kidneys, are also areas of potential value.—*Charles W. Cooley, M.D.*

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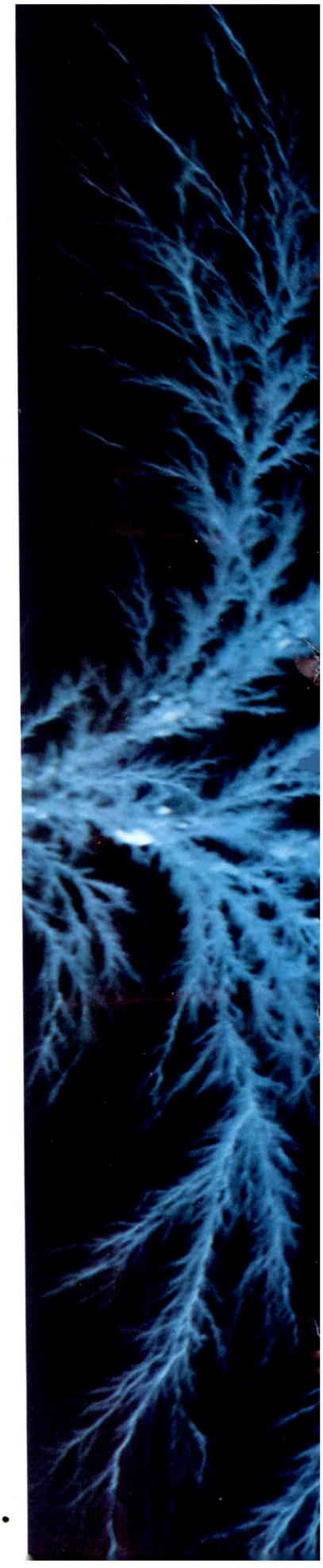

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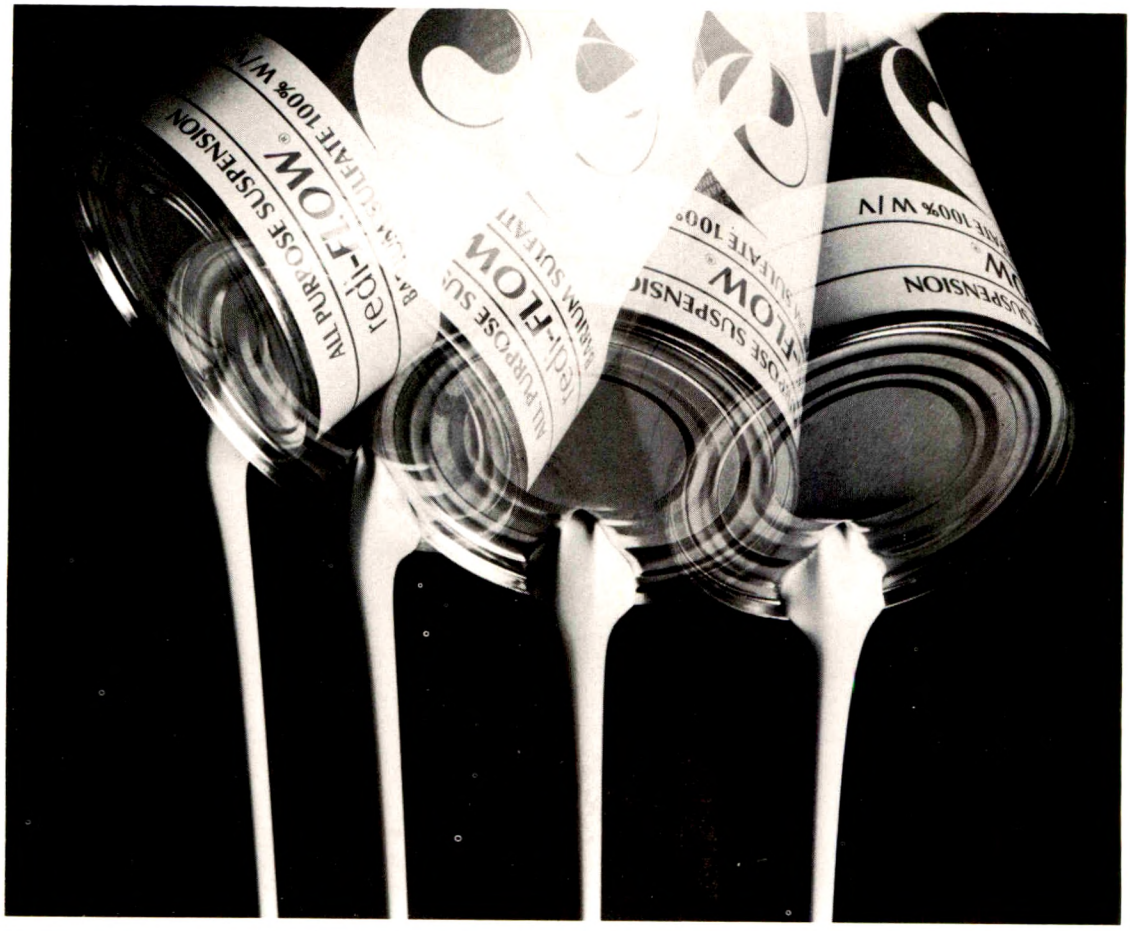

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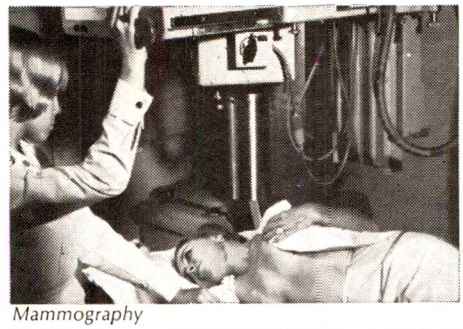
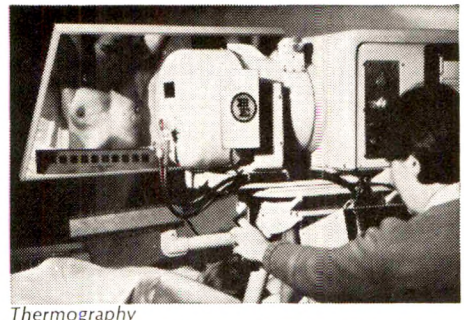


Mammography



Thermography

And we *do* have the means to achieve earlier diagnosis. We do have an *earlier warning system.* Mammography and thermography can detect breast cancer *before* a lump is discernible by palpation. To demonstrate that it is practical and feasible to detect breast cancer earlier by using these modalities, the American Cancer Society and the National Cancer Institute are funding a network of breast cancer demonstration projects. Supported by grants of $2-million from the ACS and $4-million from the NCI, 20 such centers are expected to be operative across the country by the end of the year. Each will screen at no charge, approximately 5,000 women annually, in what is considered to be the ideal detection program — to include clinical examination, mammography and thermography. Each of these detection methods contributes independently to the detection of breast cancer, and none can be dispensed with in the search for early disease.

At present we cannot prevent breast cancer, but the potential for saving more lives is immense. The five-year survival rate changes dramatically from 53% when axillary nodes are positive, to 85% when the disease is localized, to nearly 100% for in-situ cancer.

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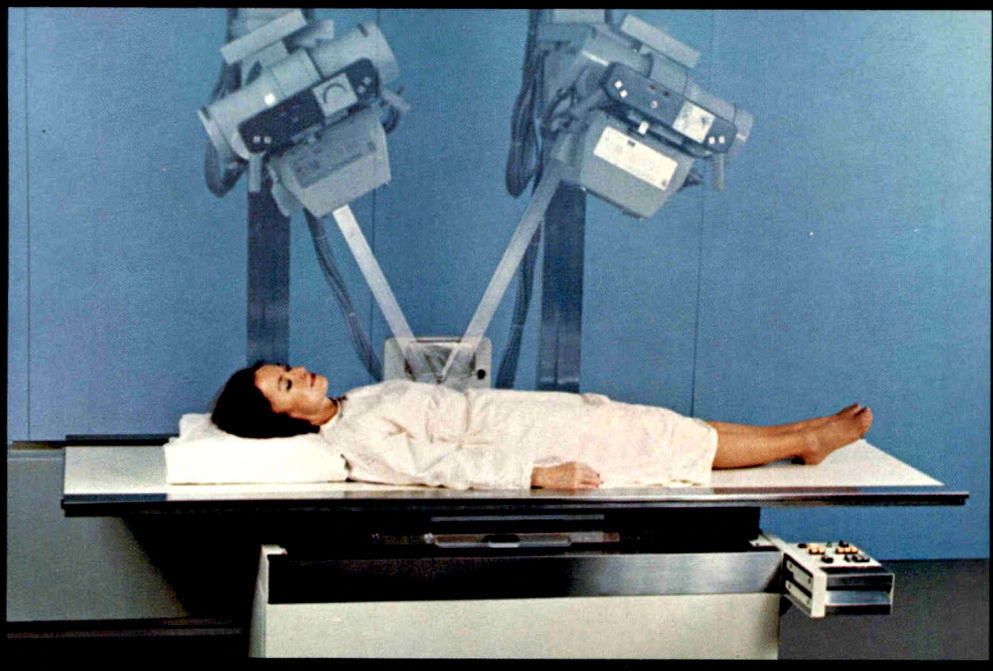

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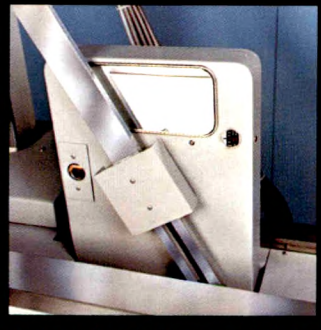

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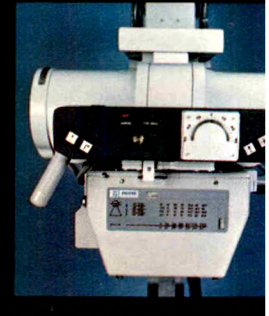

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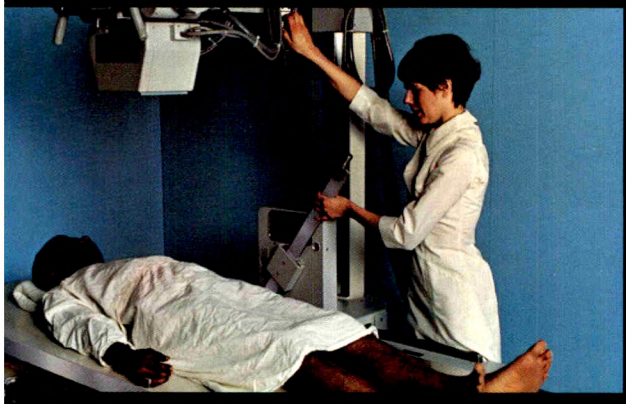

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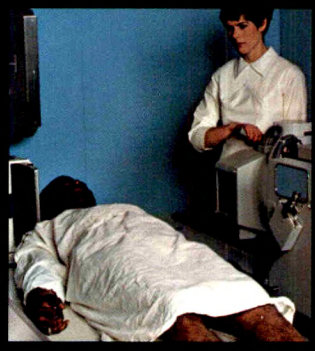

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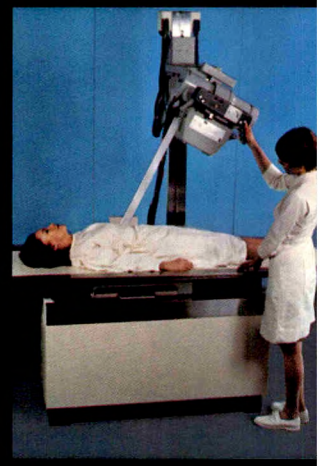

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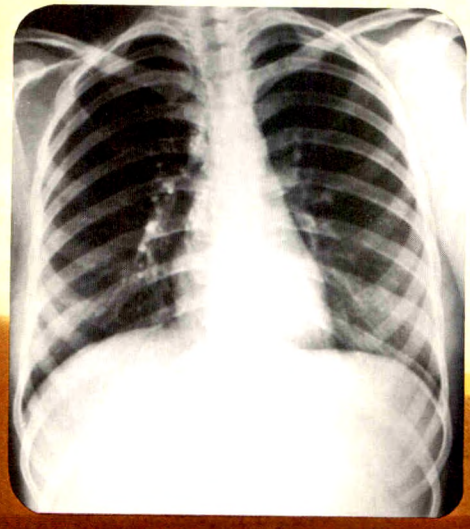

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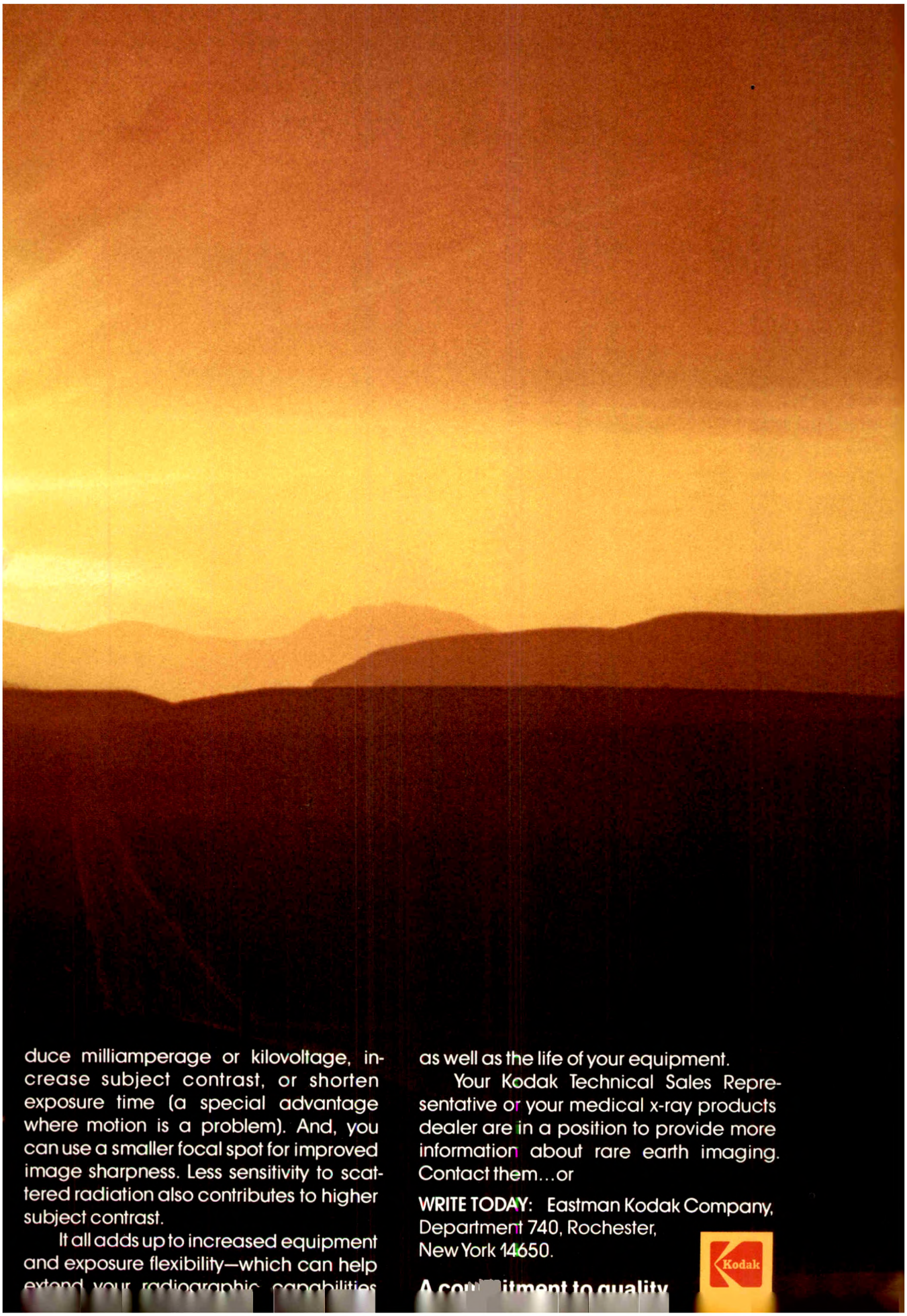

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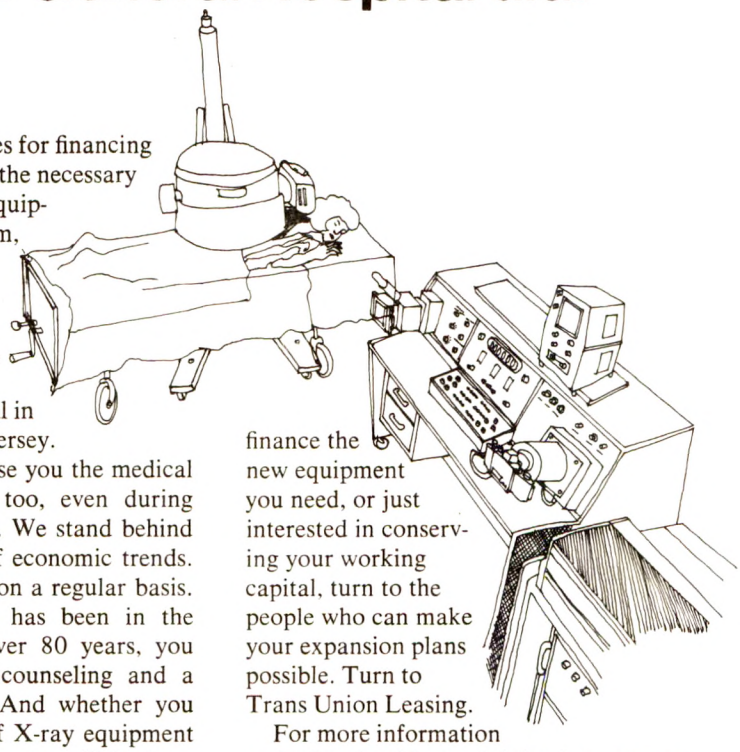

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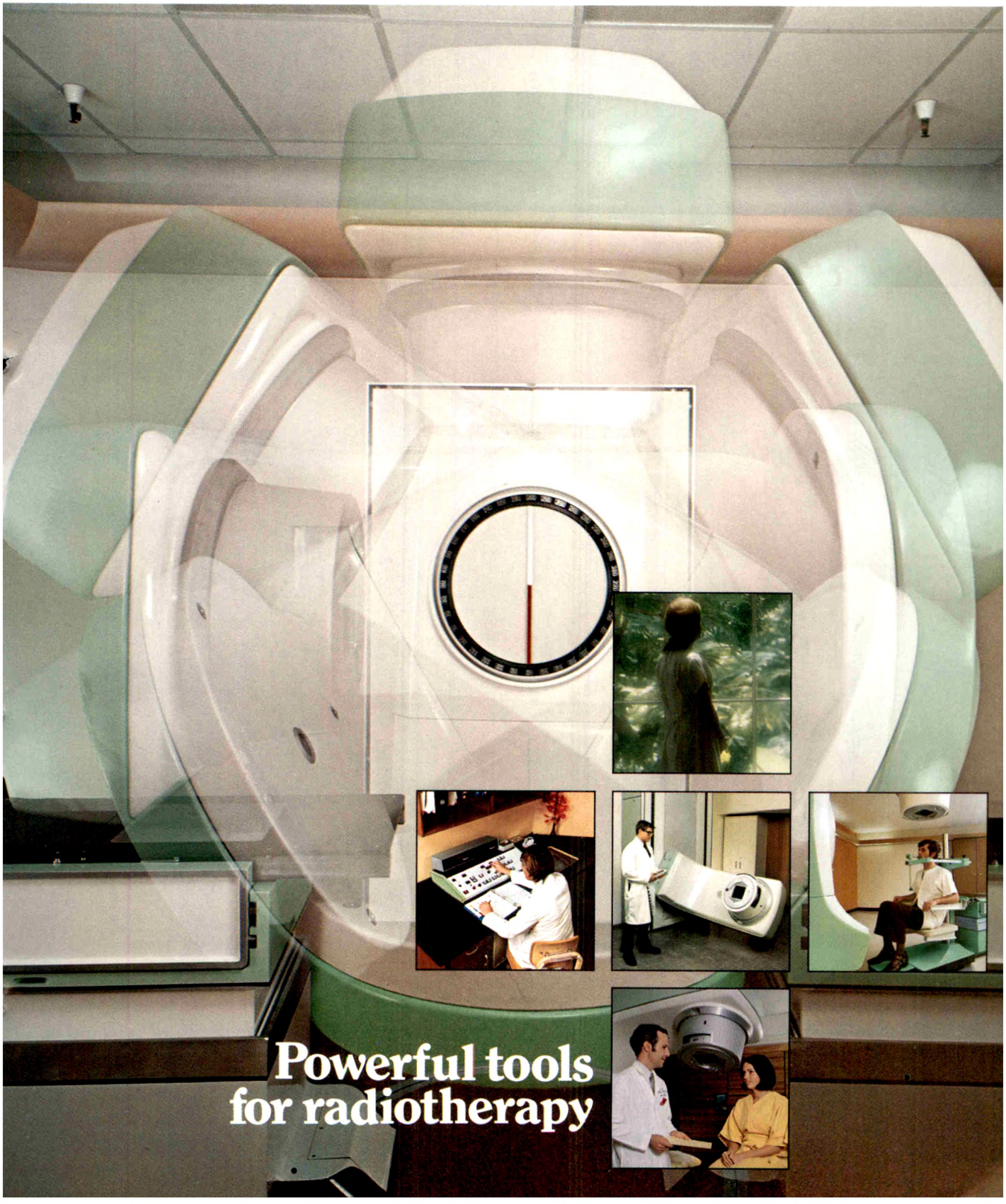

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Clinac 12

Dual Modality in an Economical Package

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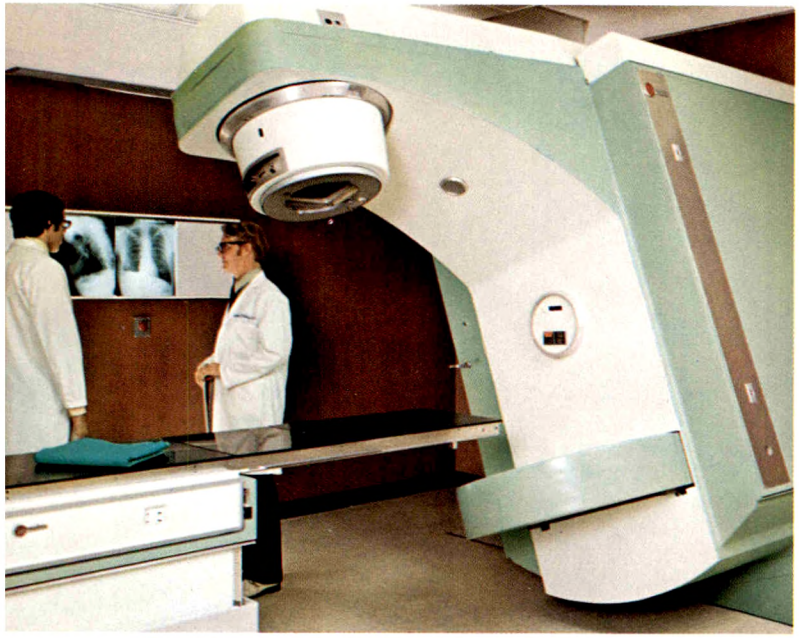

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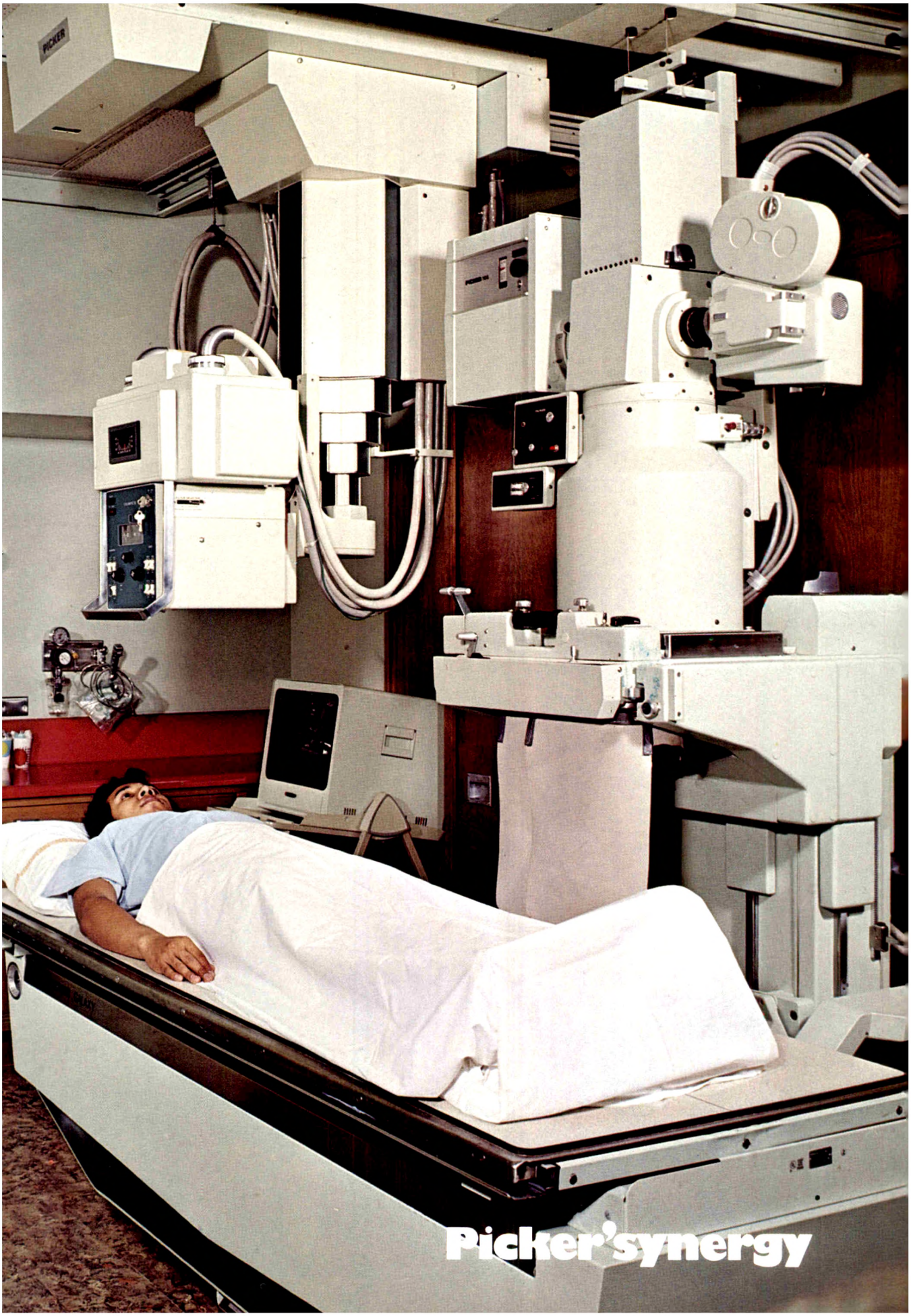
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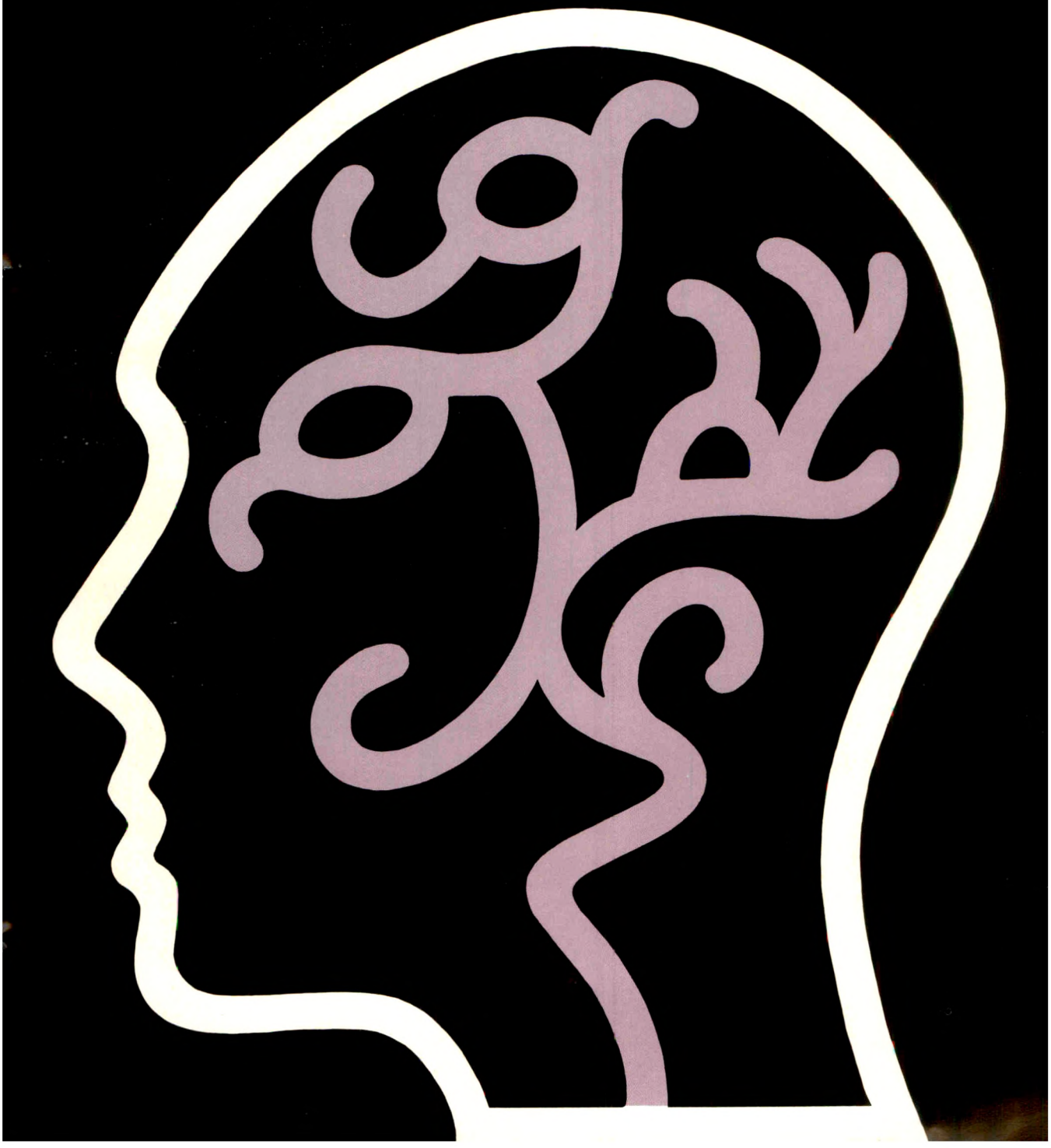

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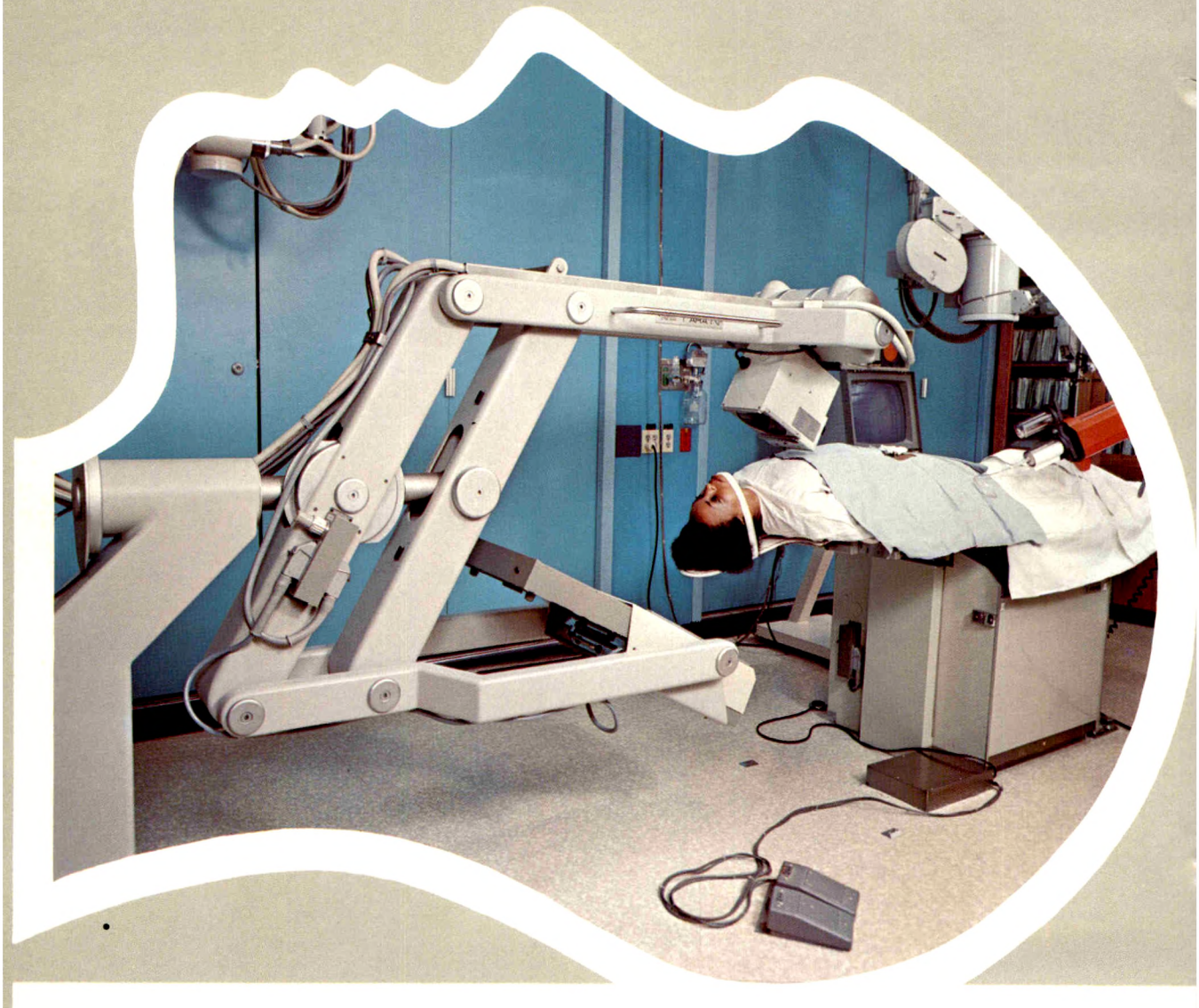

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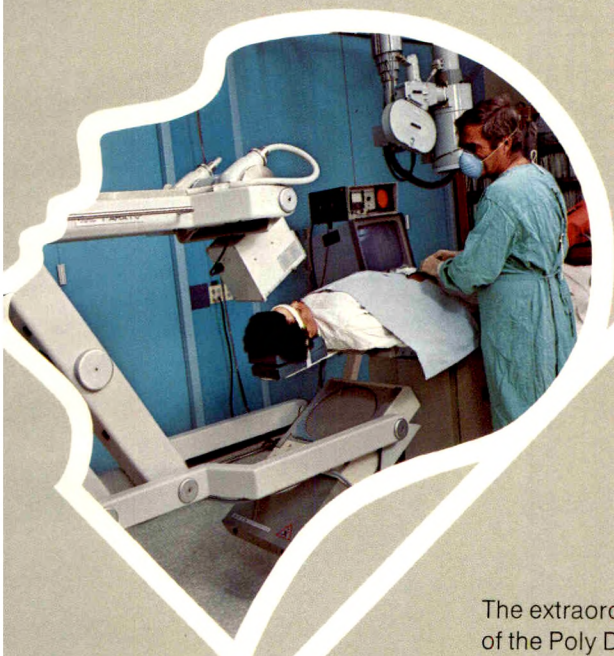

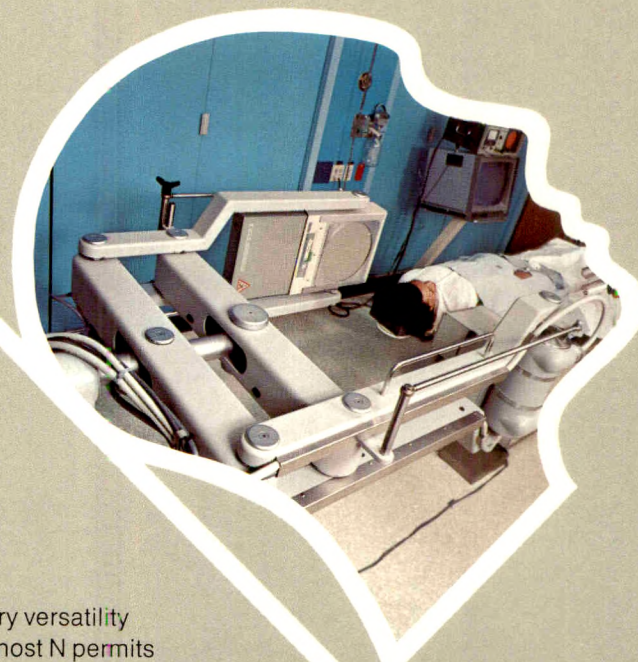

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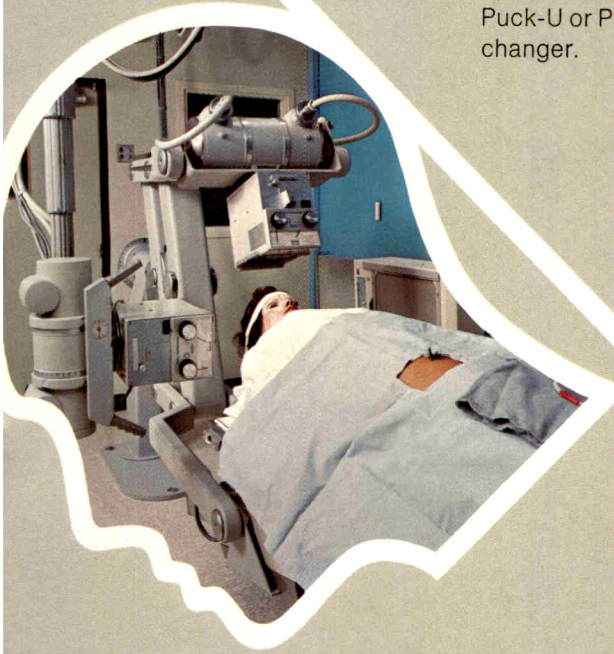

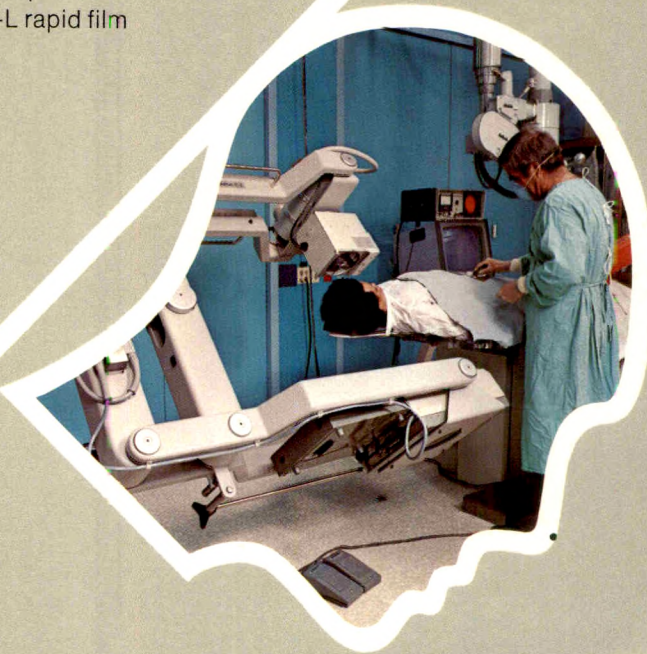

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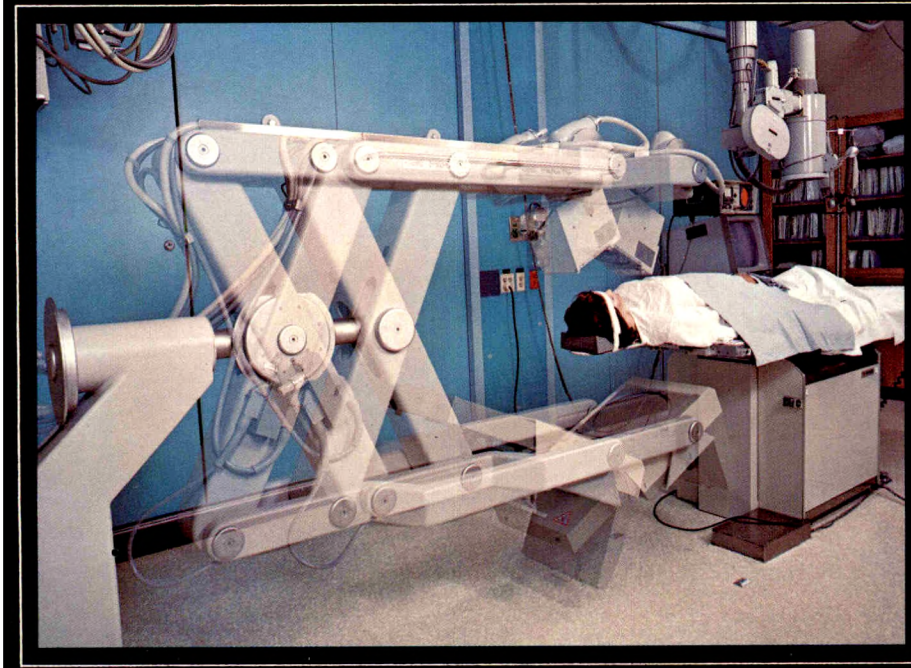

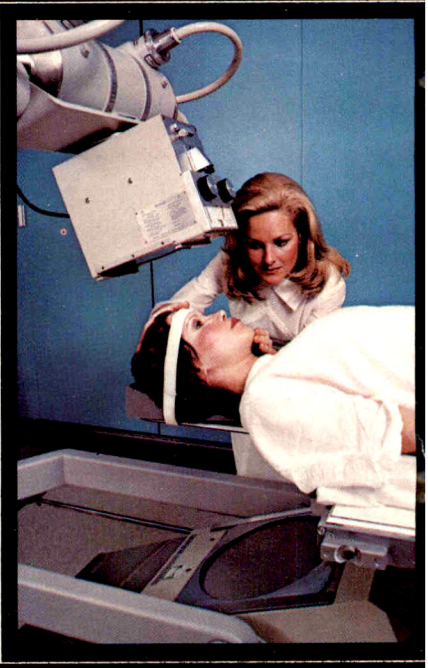

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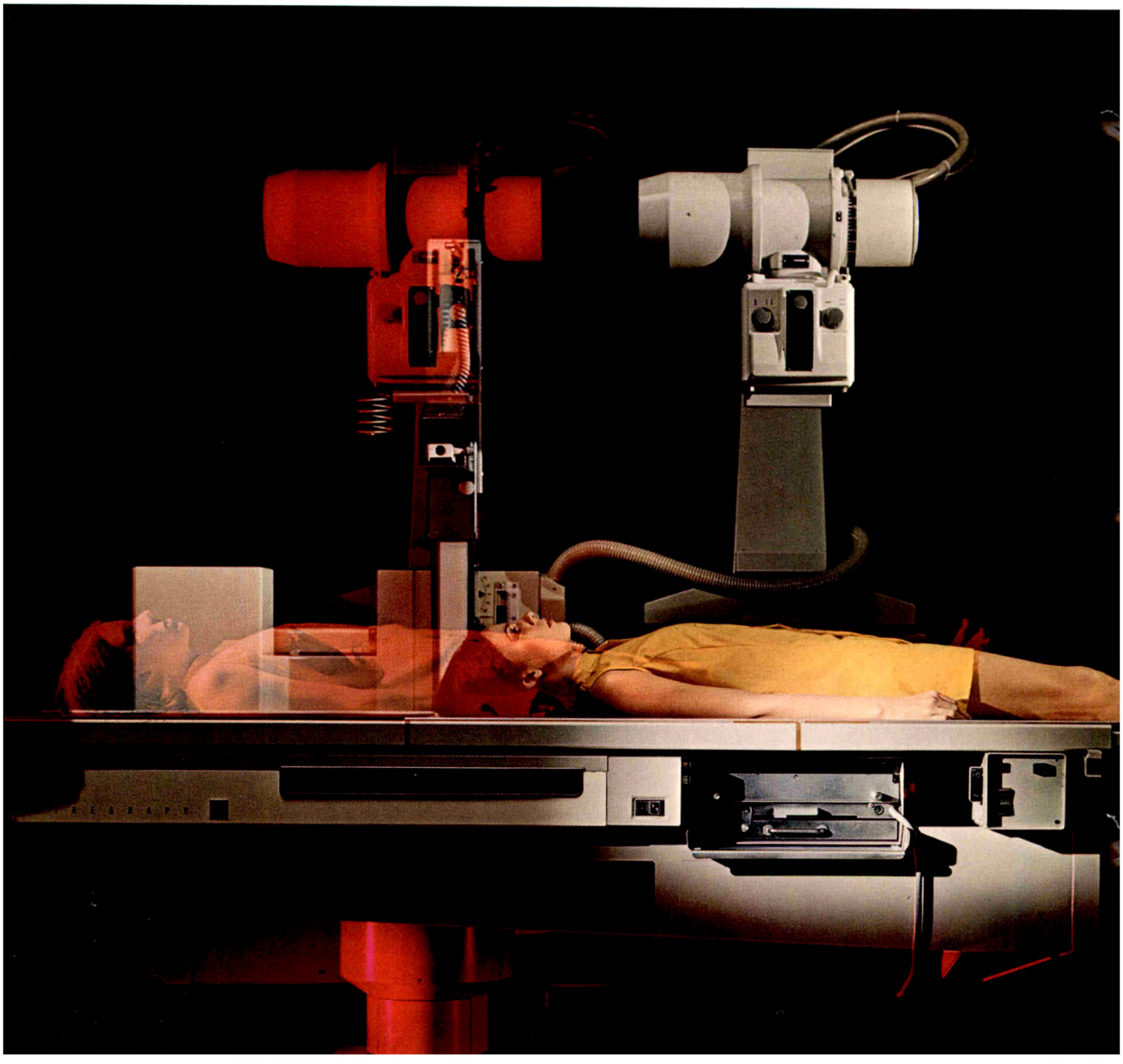

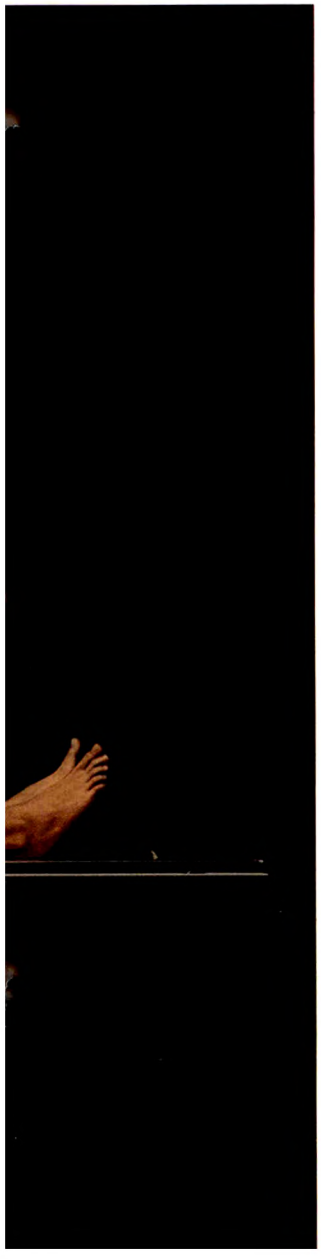

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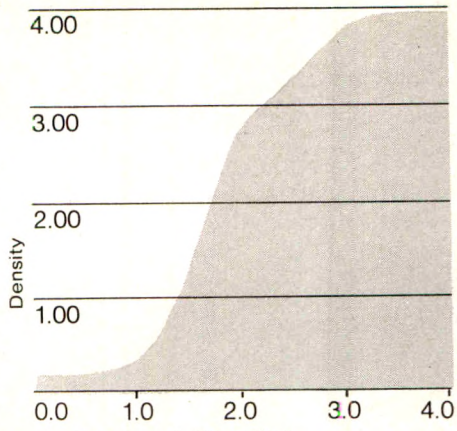

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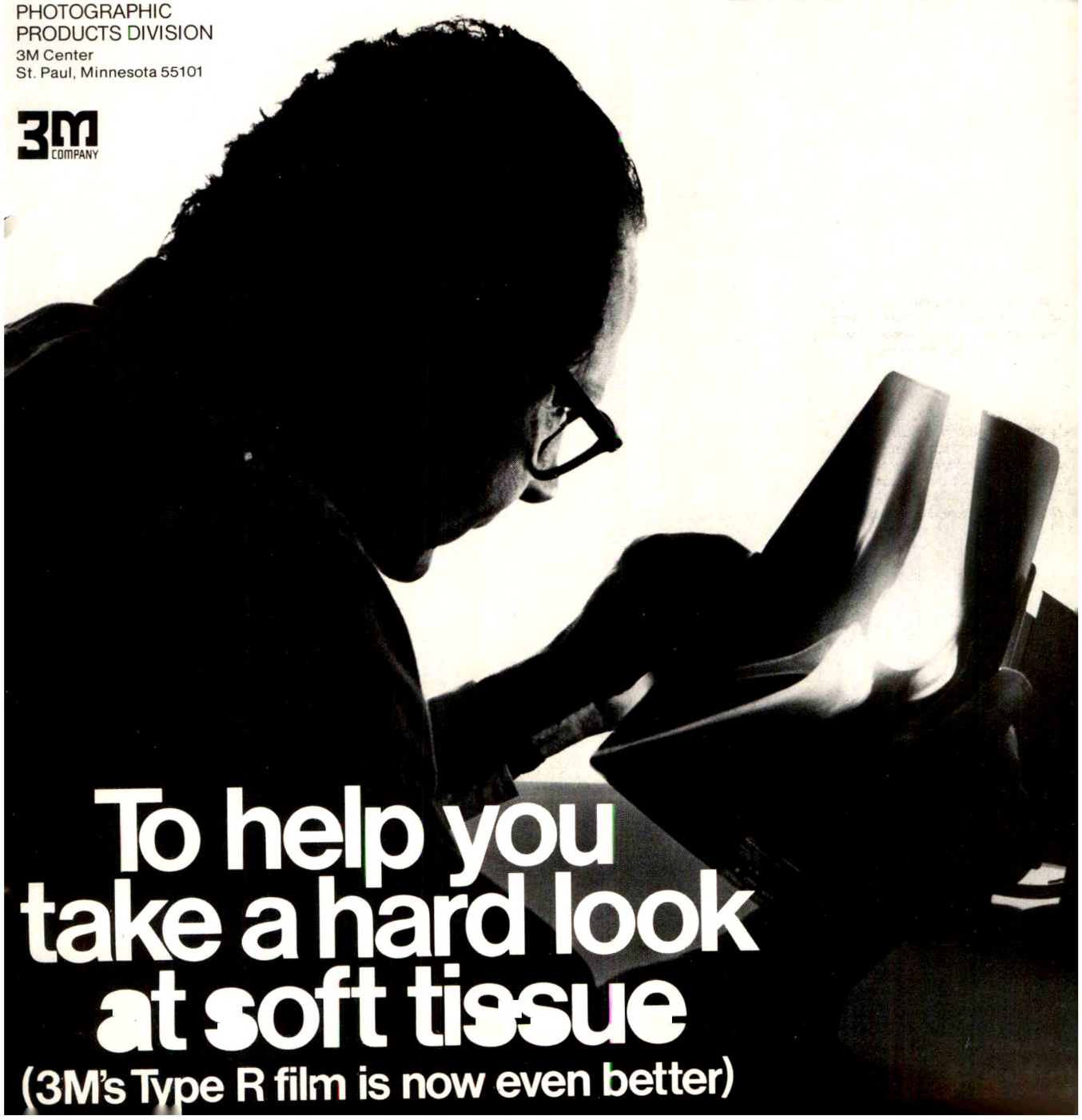

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THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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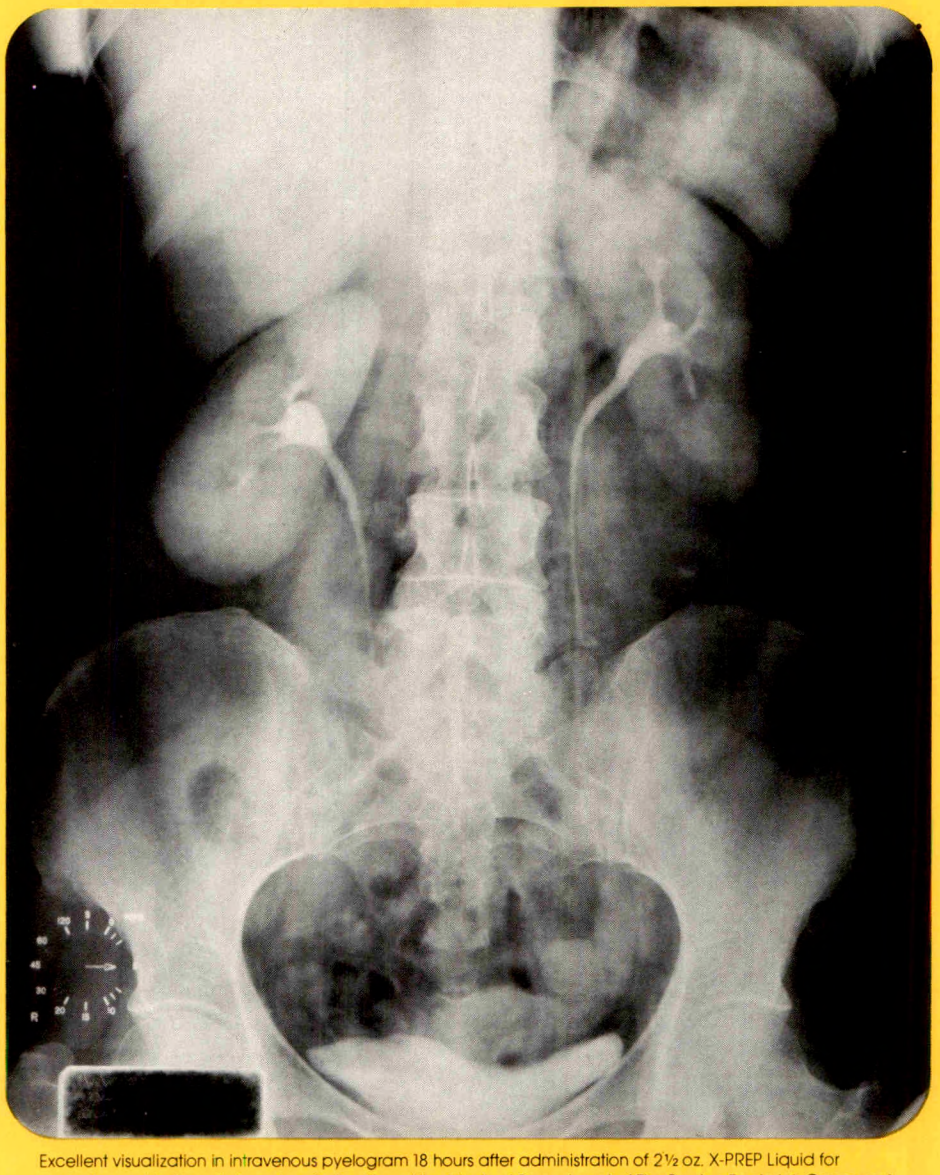

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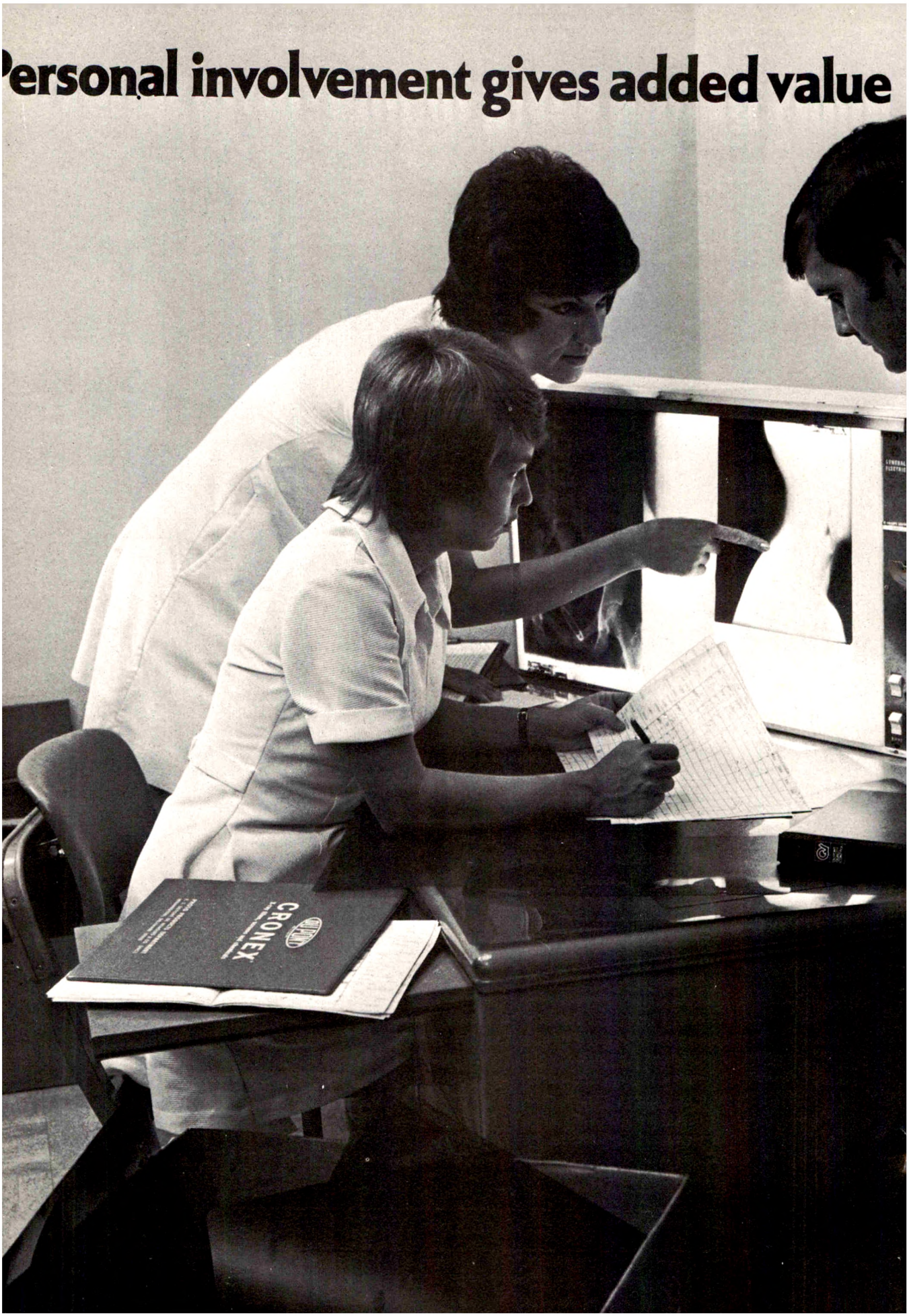

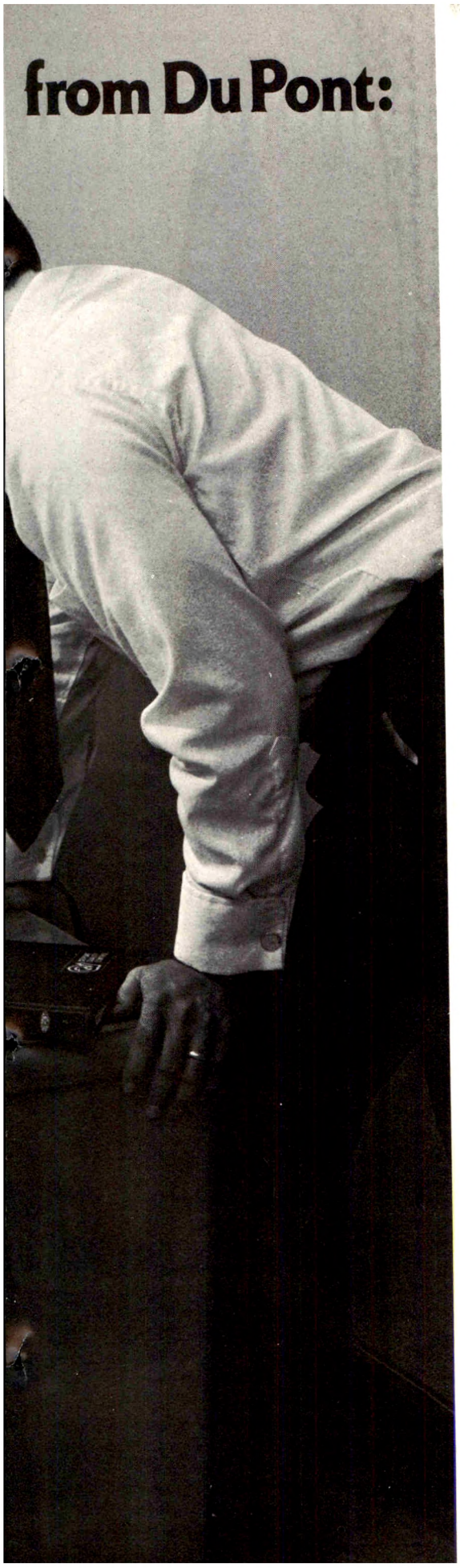

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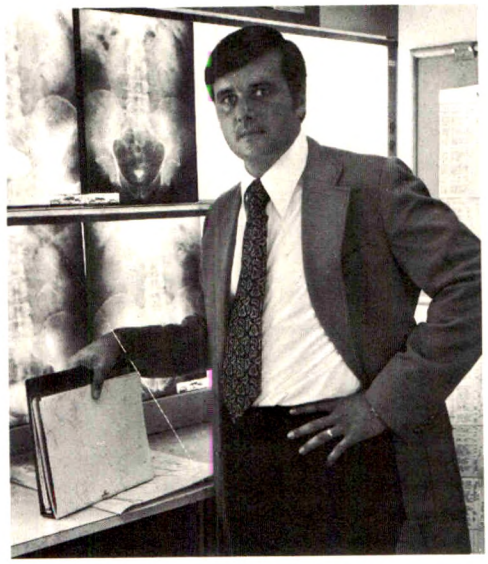

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All instructions are entered through the Computer Module keyboard or lighted push buttons. Instructions are given in response to questions asked by the Computer Module's built-in alpha-numeric printer and backlighted display panel. Inappropriate instructions are automatically detected, giving the operator a chance to make corrections before assays are run. Completed instruction sequences may be listed or edited before starting.

Made easy.

from SKI gives final answers in medically useful units–automatically.

with final assay results on permanent paper tapes. But the system is flexible. Automatic operation may be interrupted at any point for a stat procedure or to enter instructions for additional tests.

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The SKI Automated Gamma System's compact, modular design takes up little precious laboratory space. Almost any table or benchtop will do.

Now, virtually any laboratory can enjoy the speed and flexibility of a fully automated gamma counting system.

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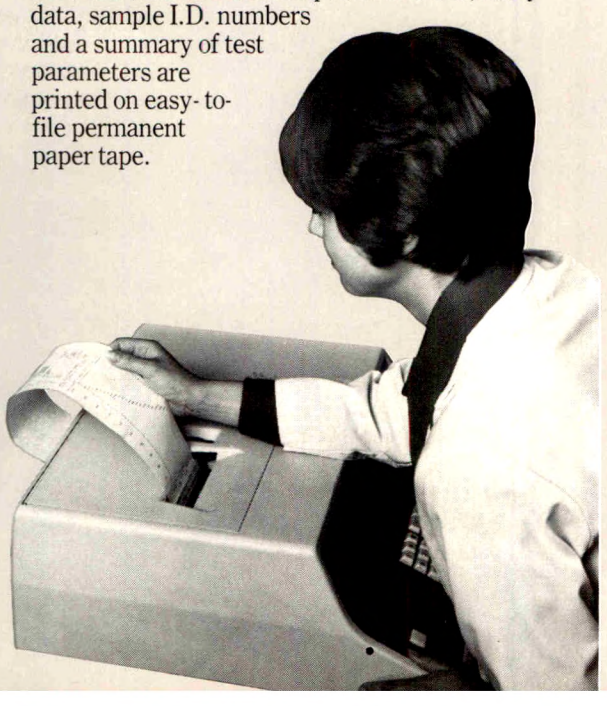
3. Examine the results.

The SKI Automated Gamma System gives results with or without automatic background subtraction in any of four different forms: (1) concentration derived from an automatically computed standard curve, (2) counts per minute at preset count, (3) counts per minute at preset time, or (4) ratio between standard and sample. In all cases, assay data, sample I.D. numbers and a summary of test parameters are printed on easy-to-file permanent paper tape.



☐ Please send details about The Gamma System.
☐ Please have a representative call for an appointment.
☐ We'd like a demonstration of The Gamma System at no expense or obligation to us.

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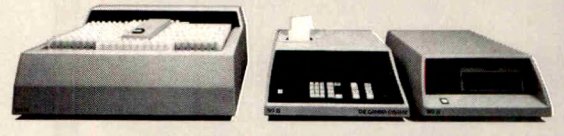

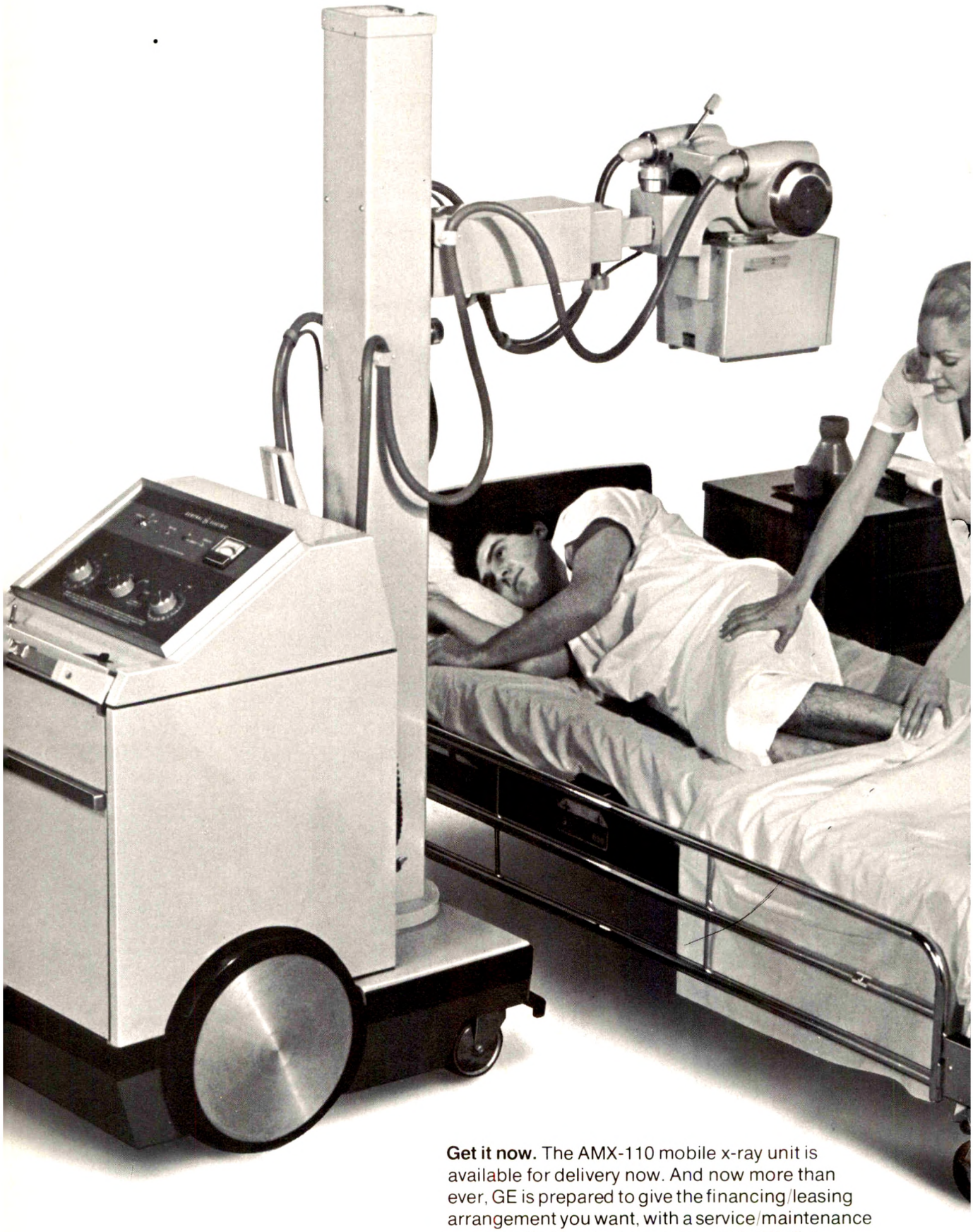

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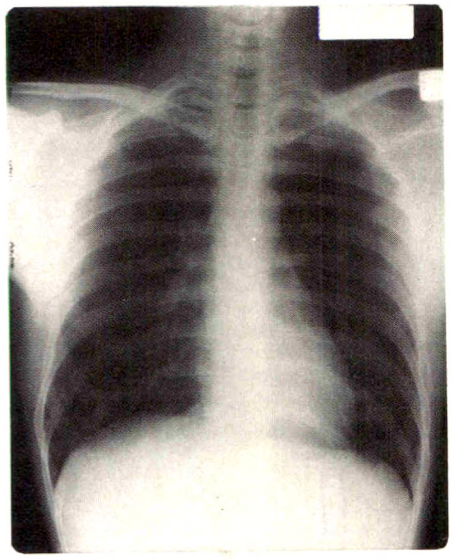
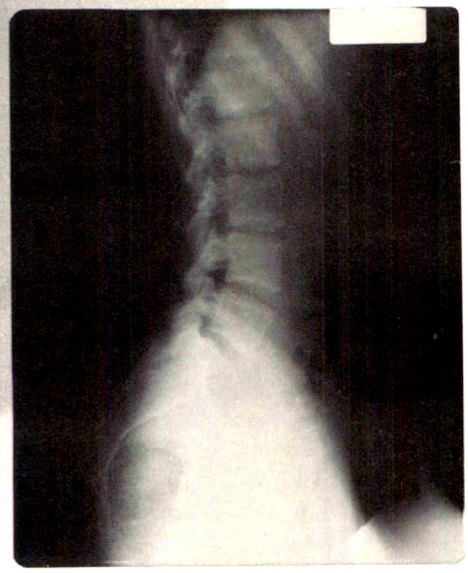


AP Chest, 1.0 mAs, 85 kVp. Non-grid, technic par speed screens, 40 inch distance.



LAT Lumbar spine, 250 mAs, 95 kVp. 12:1, 103 line grid cassette equipped with par speed screens, 40 inch distance.

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The Mass Film Viewer that saves you time and money

The Panoramascope Model 200D A belt driven unit mounted on a mobile base with additional illuminator for film comparisons.

This radiologist can do a day's diagnostic film reading in ⅓ less time than conventional viewing by using a Panoramascope, capable of displaying 200 14 x17 radiographs. He can retrieve any study he desires simply by pushing a button and bringing it into view. No film shuffling, mounting or misplaced jackets.

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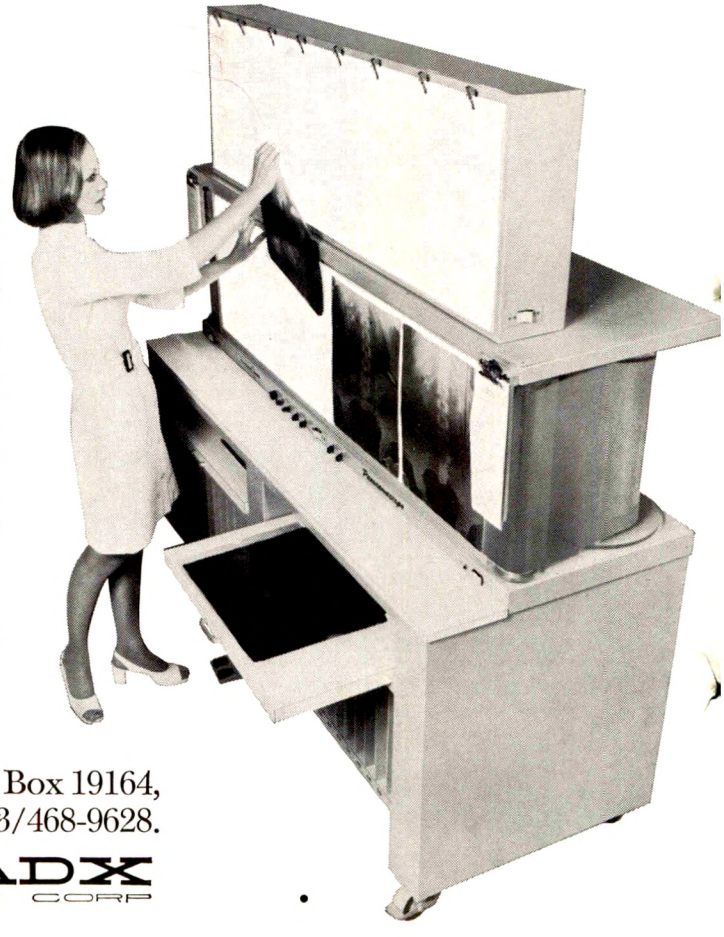

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RENOGRAFIN®-76

Meglumine Diatrizoate 66% and Sodium Diatrizoate 10% Injection

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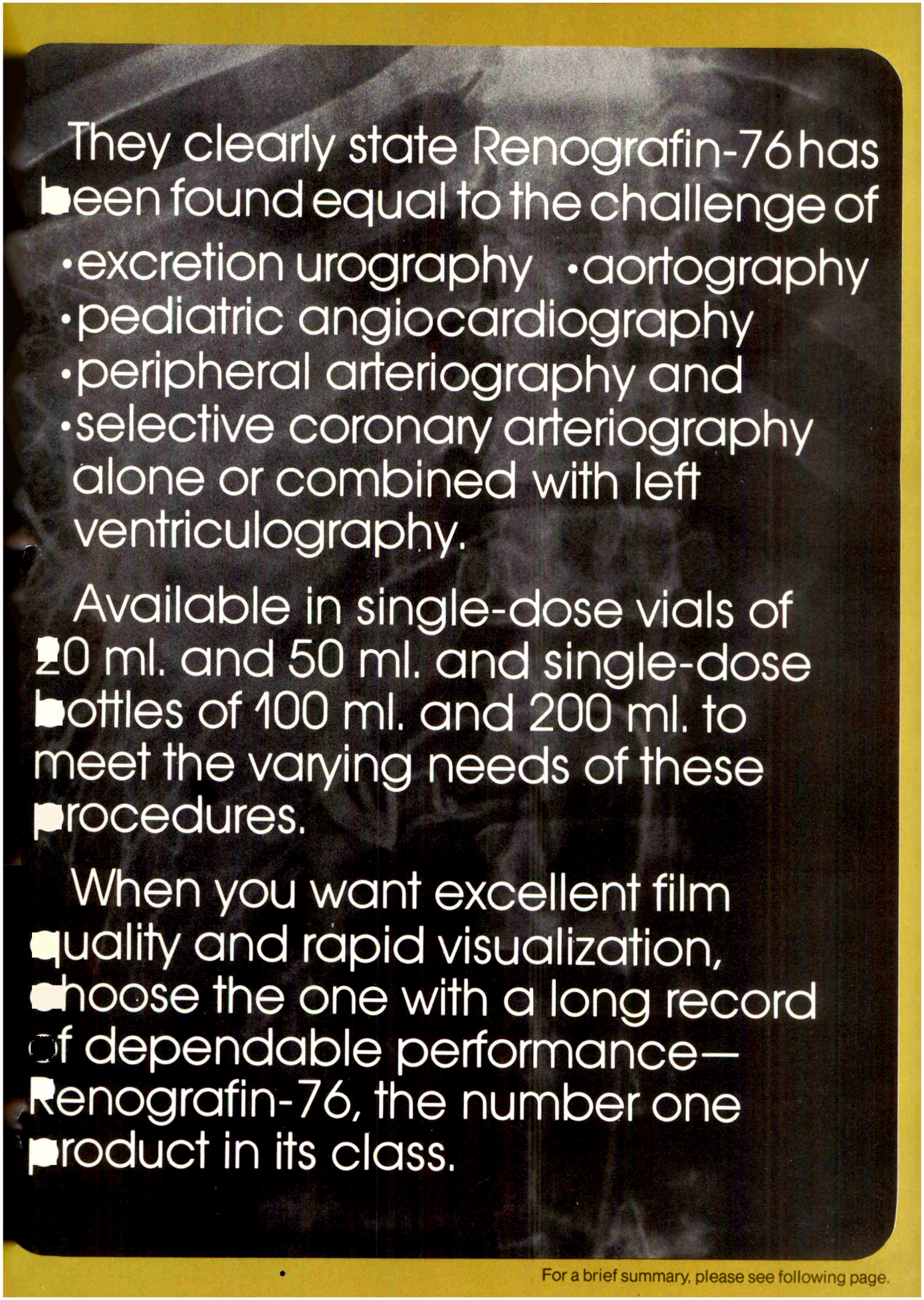
contrast media™

*Based on an approximation of the number of procedures performed using Renografin-76 from January, 1971, through June, 1974.



For a brief summary, please see following page.

RENOGRAFIN®-76

(Meglumine Diatrizoate 66% and Sodium Diatrizoate 10% Injection)

Renografin-76 is supplied as a sterile, aqueous solution providing 66% meglumine diatrizoate and 10% sodium diatrizoate with 0.32% sodium citrate as a buffer and 0.04% disodium edetate as a sequestering agent. The solution contains approximately 37% (370 mg./ml.) bound iodine and *approximately 4.48 mg. (0.19 mEq.) sodium per ml.*

CONTRAINDICATIONS: In patients with a hypersensitivity to salts of diatrizoic acid. Urography contraindicated in patients with anuria.

WARNINGS: A definite risk exists with the use of contrast agents in excretion urography in patients with multiple myeloma. There has been anuria with progressive uremia, renal failure and death. This risk of the procedure in these patients is not a contraindication; however, partial dehydration in preparation for study is not recommended since it may predispose for precipitation of myeloma protein in renal tubules. No therapy, including dialysis, has been successful in reversing this effect. Myeloma should be considered in persons over 40 before undertaking urographic procedures.

In cases of known or suspected pheochromocytoma, if the physician feels that the possible benefits outweigh the considered risks, radiopaque materials should be administered with extreme caution; however, an absolute minimum of material should be injected, the blood pressure should be assessed throughout the procedure, and measures for treating a hypertensive crisis should be available.

Contrast media may promote sickling in homozygous individuals when injected I.V. or intra-arterially. Although a history of sensitivity to iodine *per se* or to other contrast media is not an absolute contraindication, administration of diatrizoate requires extreme caution in such cases. Renografin-76 should be used in pregnant patients only when the physician deems its use essential to the welfare of the patient since safe use during pregnancy has not been established. Perform thyroid function tests prior to administration of Renografin-76 since iodine-containing contrast agents may alter the test results.

Weigh the inherent risks against necessity for performing *angiocardiography* in cyanotic infants and patients with chronic pulmonary emphysema. In *pediatric angiocardiography*, a dose of 10 to 20 ml. may be particularly hazardous in infants weighing less than 7 kg.; this risk is probably significantly increased if these infants have preexisting right heart "strain," right heart failure, and effectively decreased or obliterated pulmonary vascular beds. Perform *urography* with extreme caution in persons with severe concomitant hepatic and renal disease. Perform *selective coronary arteriography* only in selected patients and those in whom expected benefits outweigh the procedural risk.

PRECAUTIONS: Diagnostic procedures involving use of contrast agents should be performed under the direction of personnel with prerequisite training and a thorough knowledge of the particular procedure. Appropriate facilities should be available for coping with situations which may arise as a result of the procedure and for emergency treatment of severe reactions to the contrast agent itself; competent personnel and emergency facilities should be available for at least 30 to 60 minutes after I.V. administration since delayed reactions have been known to occur. These severe life-threatening reactions suggest

hypersensitivity to the contrast agent. A personal or family history of asthma or allergy or a history of a previous reaction to a contrast agent warrants special attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 ml. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated.

Renal toxicity has been reported in a few patients with liver dysfunction who were given oral cholecystographic agents followed by urographic agents; therefore, if known or suspected hepatic or biliary disorder exists, administration of Renografin-76 should be postponed following the ingestion of cholecystographic agents. Consider the functional ability of the kidneys before injecting Renografin-76. Use cautiously in severely debilitated patients and in those with marked hypertension. Bear in mind the possibility of thrombosis when using percutaneous techniques.

In *excretion urography*, adequate visualization may be difficult or impossible in uremic patients or others with severely impaired renal function (see Contraindications). In *aortography* repeated intra-aortic injections may be hazardous; this also applies to *pediatric angiocardiography* particularly in infants weighing less than 7 kg. (see Warnings). In *peripheral arteriography*, hypotension or moderate decreases in blood pressure seem to occur frequently with intra-arterial (brachial) injections; this is transient and usually requires no treatment. Monitor blood pressure during the immediate 10 minutes after injection. It is recommended that *selective coronary arteriography* not be performed for about 4 weeks after diagnosis of myocardial infarction; mandatory prerequisites to this procedure are experienced personnel, ECG monitoring apparatus, and adequate facilities for immediate resuscitation and cardioversion.

ADVERSE REACTIONS: Nausea, vomiting, flushing, or a generalized feeling of warmth are the reactions seen most frequently with intravascular injection. Symptoms which may occur are chills, fever, sweating, headache, dizziness, pallor, weakness, severe retching and choking, wheezing, a rise or fall in blood pressure, facial or conjunctival petechiae, urticaria, pruritus, rash, and other eruptions, edema, cramps, tremors, itching, sneezing, lacrimation, etc. Antihistaminic agents may be of benefit; rarely, such reactions may be severe enough to require discontinuation of dosage. There have been a few reports of a burning or stinging sensation or numbness, of venospasm or venous pain, and of partial collapse of the injected vein. Neutropenia or thrombophlebitis may occur. Severe reactions which may require emergency measures (see Precautions) are a possibility and include cardiovascular reaction characterized by peripheral vasodilatation with hypotension and reflex tachycardia, dyspnea, agitation, confusion, and cyanosis progressing to unconsciousness. An allergic-like reaction ranging from rhinitis or angioneurotic edema to laryngeal or bronchial spasm or anaphylactoid shock may occur. Temporary renal shutdown or other nephropathy may occur.

Adverse reactions as a consequence of *excretion urography* include cardiac arrest, ventricular fibrillation, anaphylaxis with severe asthmatic reaction, and flushing due to generalized vasodilatation. Risks of *aortography* procedures include injury to aorta and neighboring organs, pleural puncture, renal damage (including infarction and acute tubular necrosis with oliguria and anuria), accidental selective filling of right renal artery during translumbar procedure in presence of preexistent renal disease, retroperitoneal hemorrhage from translumbar approach, spinal cord injury and pathology associated with syndrome of transverse myelitis, generalized petechiae, and death following hypotension, arrhythmia, and anaphylactoid reactions. In *pediatric angiocardiography*, arrhythmia and death have occurred. During *peripheral arteriography*, hemorrhage from puncture site, thrombosis of the vessel, and brachial plexus palsy (following axillary artery injection) have occurred. During *selective coronary arteriography* and *selective coronary arteriography with left ventriculography*, transient ECG changes (most patients); transient arrhythmias (infrequent); ventricular fibrillation (from manipulation of catheter or administration of medium); hypotension; chest pain; myocardial infarction; transient elevation of creatinine phosphokinase (occurred in about 30% of patients tested); fatalities have been reported; hemorrhage, thrombosis, pseudoaneurysms at puncture site, dislodgment of arteriosclerotic plaques, dissection of coronary vessels, and transient sinus arrest have occurred due to the procedure.

For full prescribing information, consult package insert.

HOW SUPPLIED: Available in 20 ml. and 50 ml. single-dose vials and in 100 ml. and 200 ml. single-dose bottles.

SQUIBB®

contrast media™

The Reason • is Clear

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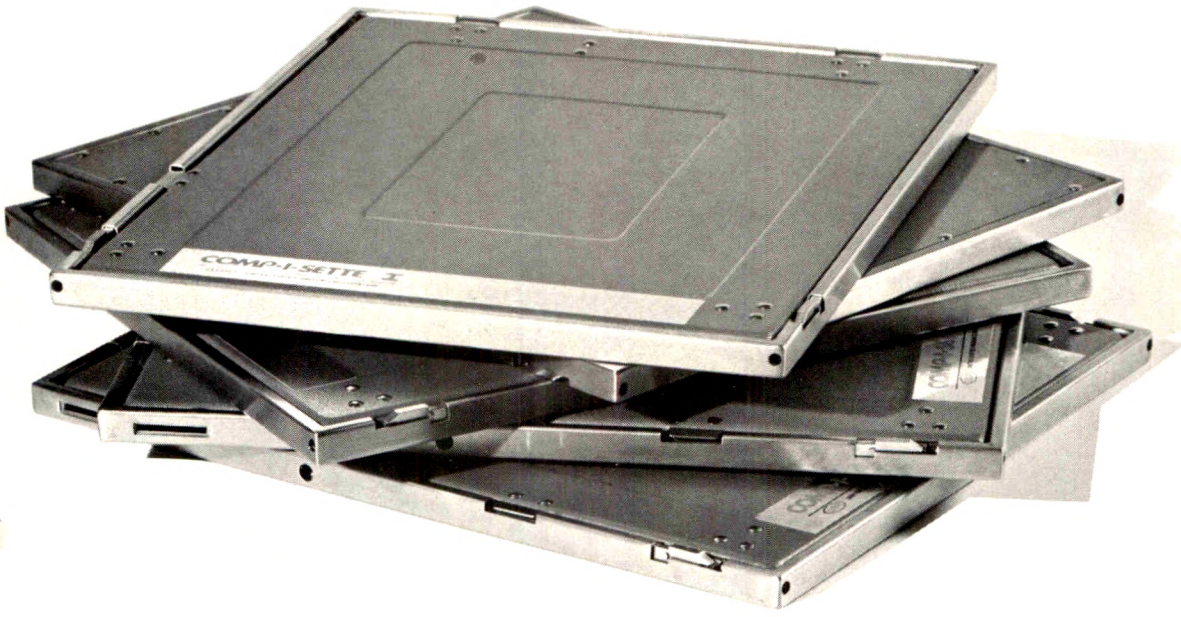
H625-006

Tough all over



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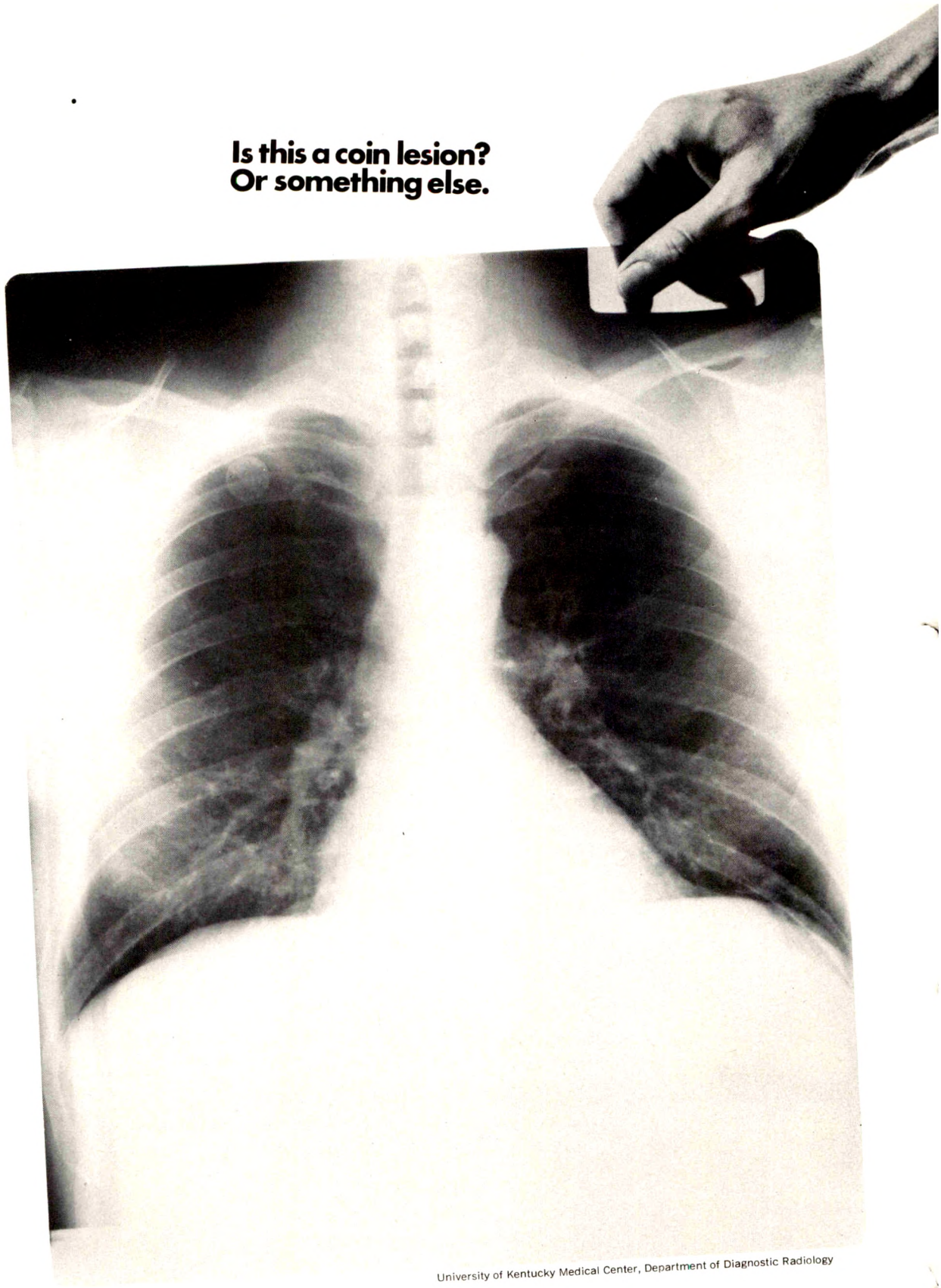

University of Kentucky Medical Center, Department of Diagnostic Radiology

(See GAF® page xxxi)

A perfect list of titles for your library

■ **Basic Radiation Biology, 2nd ed.**

By DONALD J. PIZZARELLO, Ph.D., New York University Medical Center, New York; and RICHARD L. WITCOFSKI, Ph.D. The Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, North Carolina. *143 pp. (7 x 10), 42 illus., 1975, $11.75.*

■ **Textbook of Radiotherapy, 2nd ed.**

By GILBERT H. FLETCHER, M.D., The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston. (45 Contributors and Collaborators). *816 pp. (7 x 10), 427 illus., 1973, $39.50.*

■ **Cardiac Catheterization and Angiography**

Edited by WILLIAM GROSSMAN, M.D., Peter Bent Brigham Hospital, Boston, Massachusetts. (24 Contributors). *339 pp. (7 x 10), 152 illus., 1974, $24.50.*

■ **Echocardiography**

By HARVEY FEIGENBAUM, M.D., Indiana University School of Medicine, Indianapolis. With the assistance of SONIA CHANG, B.S. *239 pp. (7 x 10), 193 illus., 1972, $11.00.*

■ **Cancer Medicine**

Edited and with contributions by JAMES F. HOLLAND, M.D., Mt. Sinai School of Medicine of the City University of New York, New York City; and EMIL FREI, III, M.D., Harvard Medical School, Boston, Massachusetts. (158 Contributors). *2018 pp. (7 x 10), 589 figs., numerous charts and tables, 1973, $90.00.*

■ **Anatomy: A Regional Atlas of the Human Body**

By CARMINE D. CLEMENTE, Ph.D., University of California at Los Angeles School of Medicine. *320 pp. (8½ x 11), 595 figs. on 300 plates (mostly in color), 1975, $24.50.*

■ **Gray's Anatomy of the Human Body, 29th American ed.**

By HENRY GRAY, F.R.S. Revised and edited by CHARLES MAYO GOSS, M.D., University of South Alabama College of Medicine, Mobile. *1466 pp. (7 x 10), 1178 illus. (mostly in color), 1973, $28.50.*

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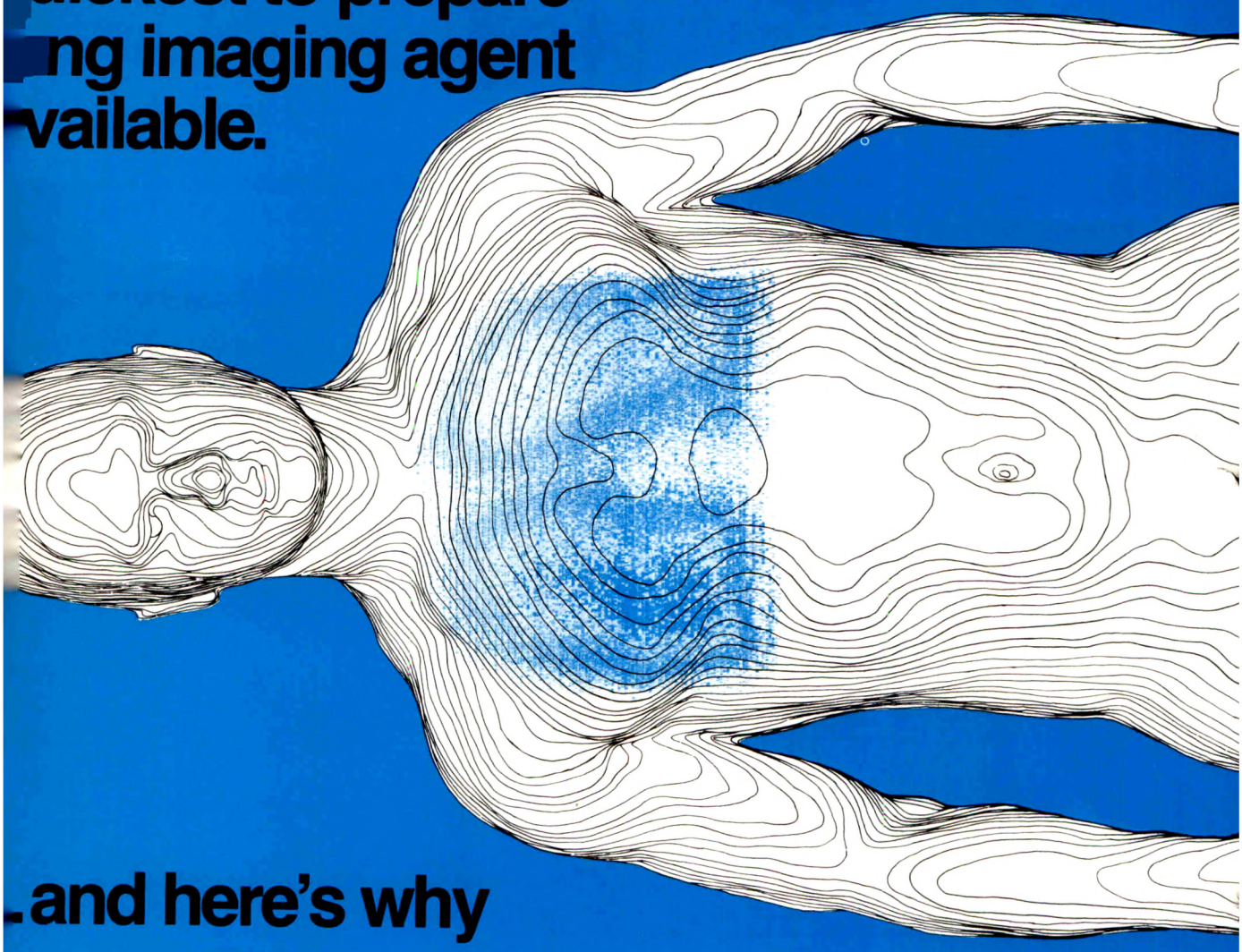

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Won't agglomerate in the vial, loses virtually labeling for 8 hours (if stored between 2°C. and C.).

Ideal for the busy lab. Recommended amount 99mTc for reconstitution high enough to allow merous scans from a single vial.

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IDICATIONS: For use in perfusion lung imaging as an adjunct to other diagnos- c procedures.

ONTRAINDICATIONS: At present there are no known contraindications to the se of this product.

ARNINGS: Radiopharmaceuticals should not be administered to patients who re pregnant, or during lactation, unless the benefits to be gained outweigh the otential hazards.

Ideally, examinations using radiopharmaceuticals, especially those elective in nature, of a woman of childbearing capability, should be performed during the first few (approximately 10) days following the onset of menses.

Since ^{99m}Tc is excreted in milk during lactation, formula-feedings should be substituted for breast-feedings.

Radiopharmaceuticals should be used only by physicians who are qualified by specific training in the safe use and handling of radionuclides produced by nuclear reactor or particle accelerator and whose experience ing have been approved by the appropriate government agency authorized license the use of radionuclides.

Note: Macrotec (Aggregated Albumin [Human]) is not radioactive. Howev after ^{99m}Tc is added, adequate shielding of the resultant preparation should b maintained.

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Aseptic technique is essential in the preparation of Technetated (Tc-99 Aggregated Albumin (Human).

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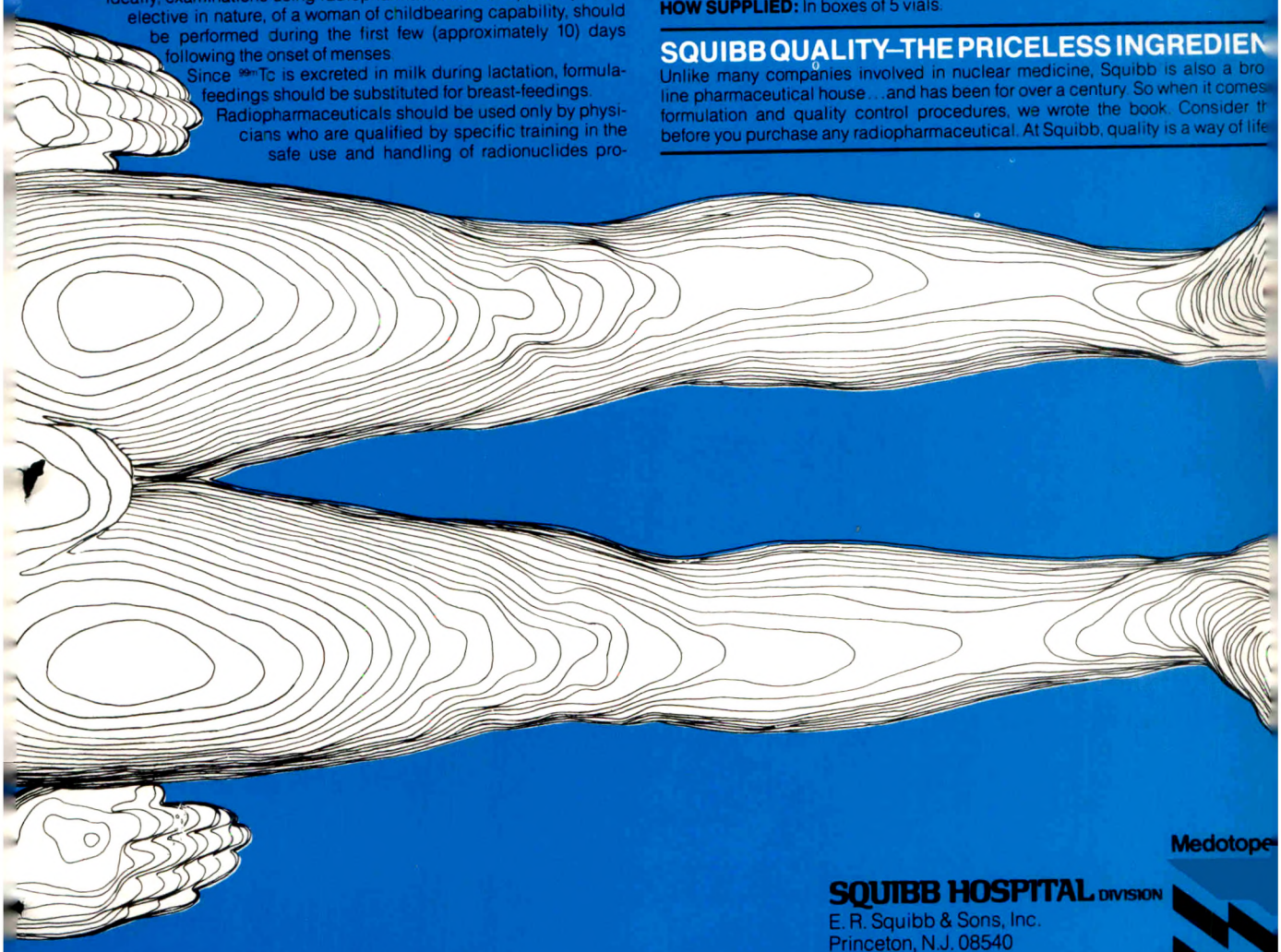

Medotope

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H605-507

99m-LABELED LUNG IMAGING AGENTS*

3. Gently agitate vial for few seconds.

4. *Allow to stand for 15 minutes* at room temperature.

5. Visually inspect vial for presence of large aggregates. *If present, do not use.*

6. *Agitate* to effect homogenous suspension of the aggregated albumin.

**Recommended maximu activity: 60 mCi.

4. Remove vial from shield (with forceps) and place in center of operating *ultrasonic bath* containing 3/4" of water. Bath should be protected by *lead glass or bricks.*
Ultrasound for 5 minutes.

**Recommended maximu activity: 30 mCi.

3. Withdraw (very slowly) 1.5-2.0 ml. of aggregate from ampul with *syringe.*

4. Inject (very slowly) syringe contents into *mixing vial.*

5. Wrap mixing vial in *absorbent paper disc* and place in lead shield.

6. Add 0.5-2.0 ml. of 99mTc** in saline into shielded mixing vial. Shake vigorously for at least 30 seconds. *Incubate* at room temperature *for 30 minutes.*

7. Shake contents vigorously just before removing aliquot intended for patient use.

**Recommended maximu activity: 25 mCi/ml.

**Recommended maximum activity: 50 mCi.

*Based on manufacturers' product information. **NOTE: See manufacturers' package inserts before the preparation of any of these produc

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Dynamax 70

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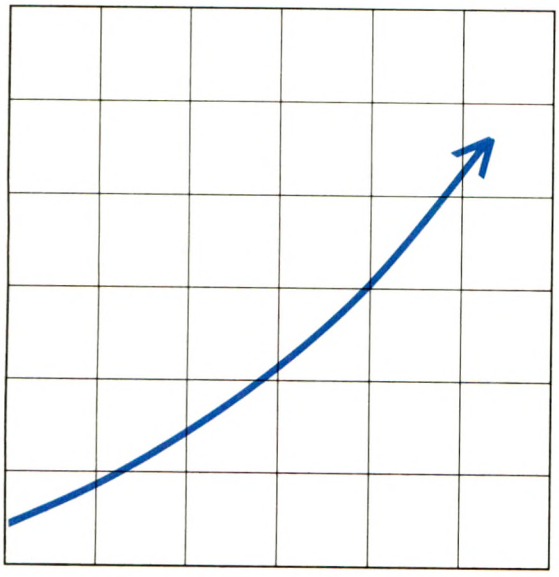
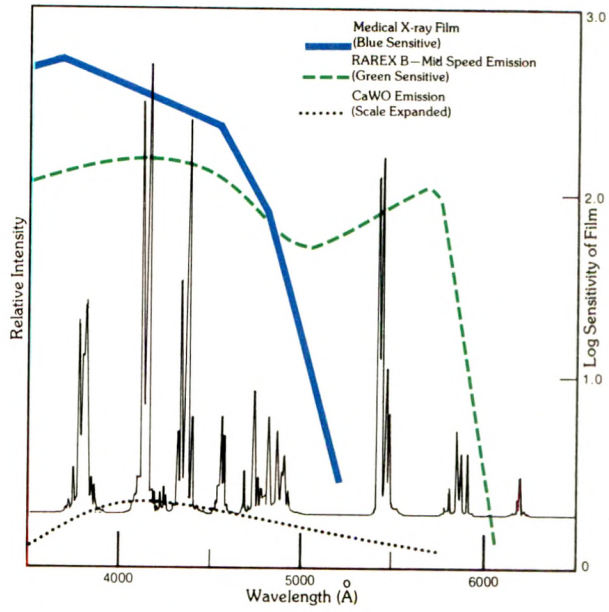


RAREX Sales vs. Months



Relative Intensity vs. Wave Length

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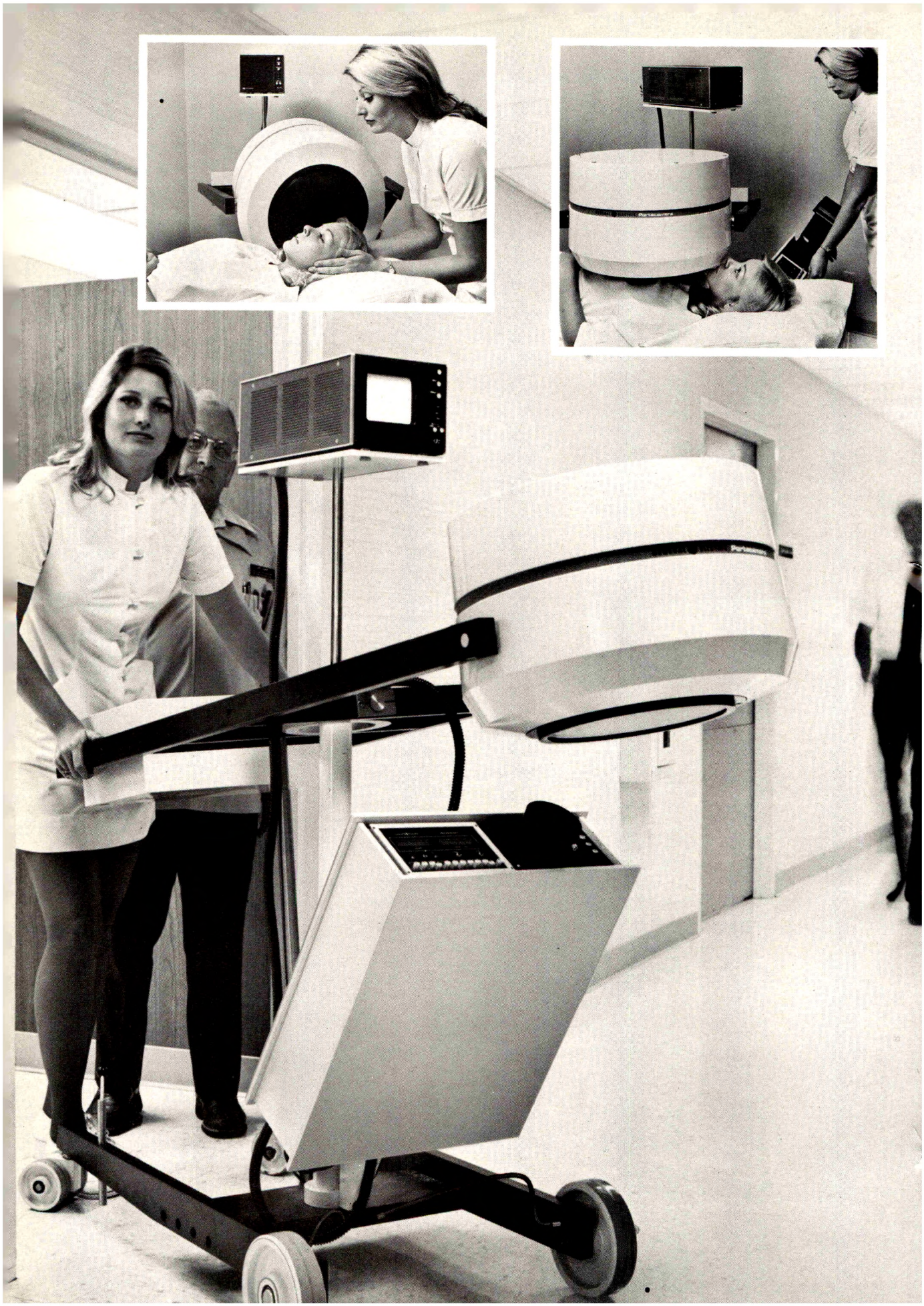

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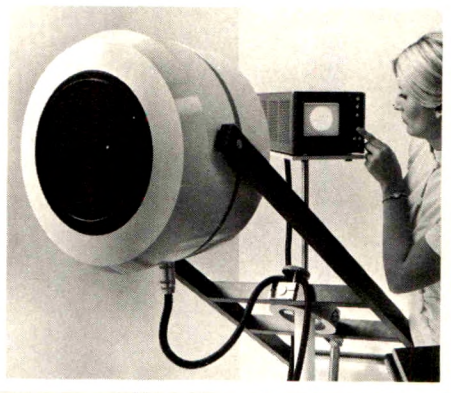

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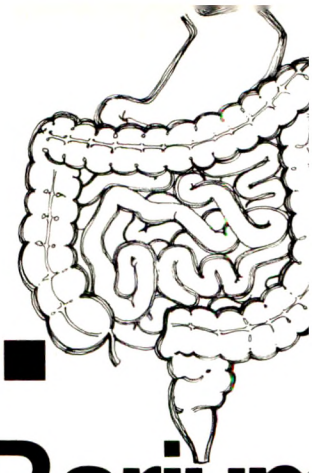

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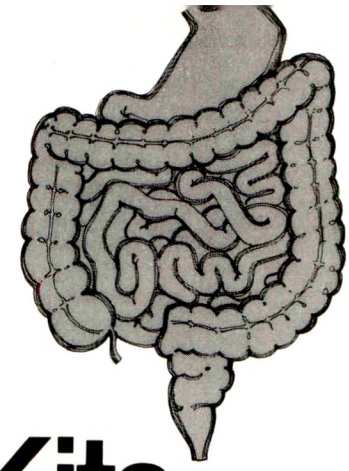

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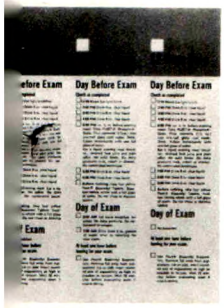

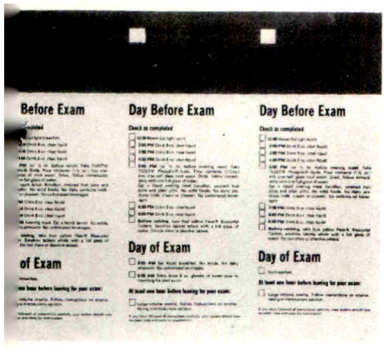

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1. *Detection of Colon Lesions*, First Standardization Conference, Amer. College of Radiology, (Chicago, Ill., 1973), pp. 203-204.
2. *Ibid*, p. 76.

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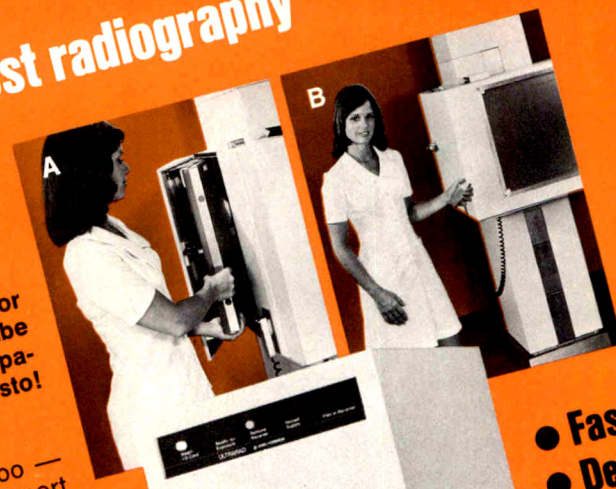

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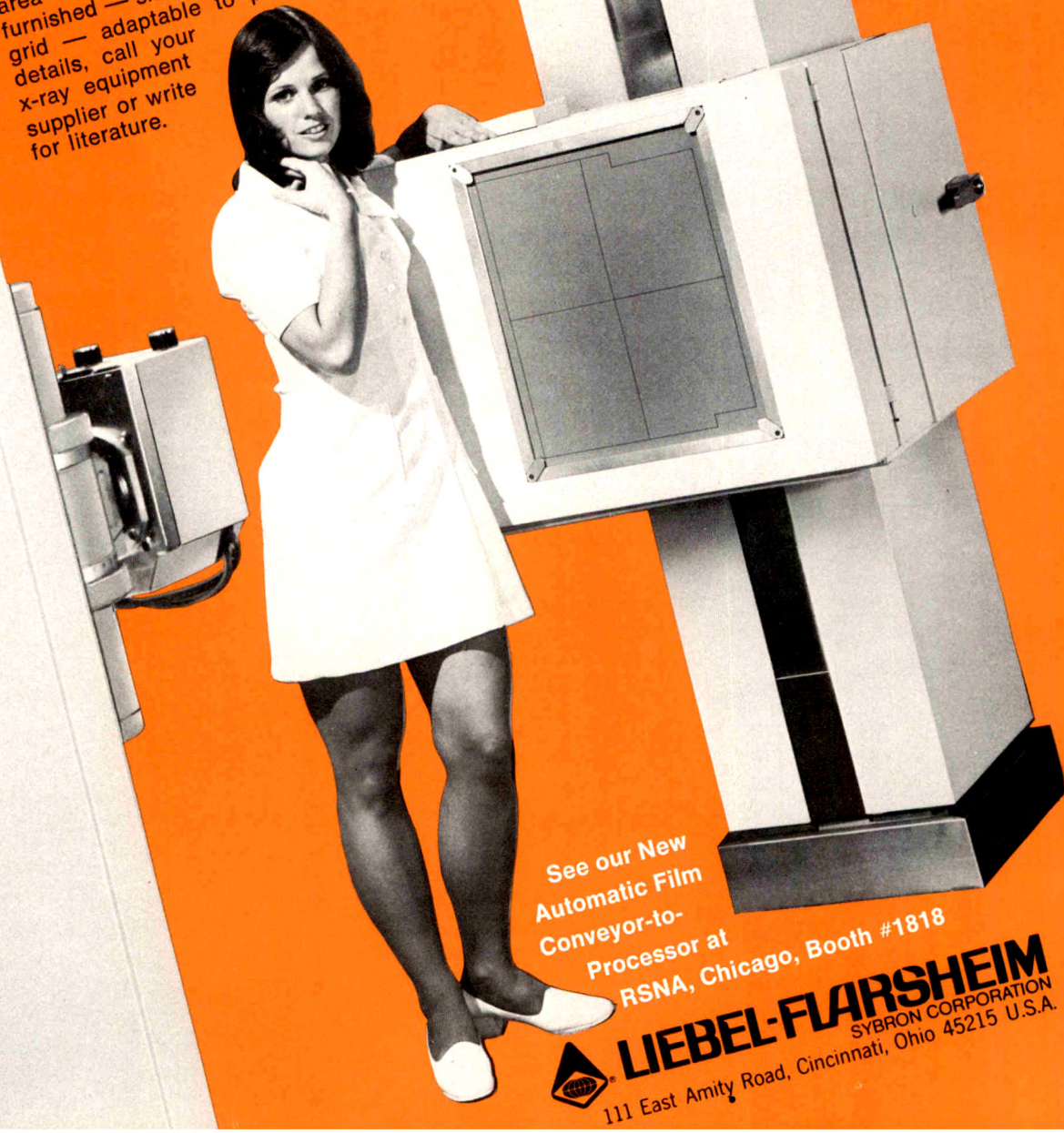

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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 125 OCTOBER, 1975 No. 2

THE SPECIFICITY OF RENAL VEIN THROMBOSIS*

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PHILADELPHIA, PENNSYLVANIA

ABSTRACT:

Clinical and roentgenographic features of renal vein thrombosis are discussed. The authors' experience with 17 cases is presented.

The roentgenologist with a high degree of suspicion can, by correlation of clinical and urographic findings, make the specific diagnosis of renal vein thrombosis in a high percentage of cases.

ORIGINALLY described by Rayer in 1840,[21] renal vein thrombosis is generally considered an uncommon event. In the past, most cases were first recognized at autopsy. However, increased clinical understanding and awareness of the condition in conjunction with improved roentgenographic techniques should allow antemortem diagnosis.

Our experience with 17 cases of renal vein thrombosis is presented. Correlation of the spectra of clinical and intravenous pyelographic presentations reveals a composite picture of the condition. Once suspected, the diagnosis can be confirmed angiographically. Prompt and appropriate therapy may then be undertaken.

BACKGROUND

Harrison *et al.*[11] described 4 fundamental varieties of renal vein obstruction: (1) thrombosis of the inferior vena cava and involvement of the renal veins secondary to extension of thrombi from pelvic and leg veins; (2) compression or invasion of the inferior vena cava and renal veins by tumor; (3) renal vein thrombosis secondary to primary renal disease; and (4) primary renal vein thrombosis. Except in the infant, primary renal vein thrombosis is thought to be rare, and this is probably related to the rapidity of blood flow in the adult. In the infant the flow rate normally tends to be low and this may pe compromised further by problems such as dehydration secondary to gastroenteritis.[11,17]

The response of the kidney is determined both by the completeness of occlusion of the vein and by the temporal balance between the occlusion of the vein and the develop-

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.
From the Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

TABLE I

ACUTE RENAL VEIN THROMBOSIS: CLINICAL DATA SUMMARY

Patient	Age	Sex	Primary Condition	Clinical Evidence	Thrombophlebitis	Pulmonary Embolus	Outcome
1.	58	M	Pancreatic adenocarcinoma	Flank pain, leg edema	Migratory, IVC	X	Death, renal failure
2.	28	F	SLE	Flank pain, leg edema, palpable kidney	None	None	Alive, right nephrectomy
3.	61	M	Diabetes mellitus, hypertension	Flank pain, leg edema	IVC, leg	X	Death, pulmonary embolus
4.	64	M	Colon carcinoma	Flank pain, leg edema, palpable kidney	IVC	X	Death, renal failure
5.	50	M	Pancreatic or colon carcinoma	Flank pain, leg edema	IVC, leg	X	Death, renal failure
6.	62	M	Abdominal aortic aneurysm	Abdominal pain, pulsatile mass	None	None	Death, renal failure

SLE = Systemic lupus erythematosus.
IVC = Inferior vena cava.

ment of collateral venous drainage. These factors in turn determine the clinical and roentgenographic presentations.[7,11,13,20,26,28] In the acute condition, sudden complete occlusion of the renal vein occurs. The affected kidney enlarges with consequent severe lumbar pain, and hematuria, proteinuria, and oliguria appear. Death from renal failure usually occurs if the condition is bilateral.[11,13,26,28] However, if the occlusion is gradual or only partial, collateral venous drainage will be developed. The effects are correspondingly less serious and adequate renal function may be preserved. This latter is the most common situation; a nephrotic syndrome frequently develops.[8,11,17,19,20]

CLINICAL AND UROGRAPHIC SPECTRA

Acute renal vein thrombosis occurs with sudden complete occlusion of the vein. Six of our 17 patients presented in this fashion. Table I summarizes the clinical data of these patients.

The patients were all acutely ill. Sudden persistent flank or abdominal pain occurred in all 6 patients.

Physical examination revealed marked costovertebral angle tenderness. Occasionally, the involved kidney was palpable (33 per cent). Leg edema was an almost constant finding, being present in 5 of 6 patients (83 per cent). Of particular note was the presence of thrombophlebitis and pulmonary embolism during the course of the illness; this occurred in 4 patients (67 per cent). Urinalysis in all 6 patients revealed gross hematuria and proteinuria. Of these 6 patients, only 1 is alive, and this patient was subjected to right nephrectomy; 4 patients died in renal failure, and 1 patient died from pulmonary embolus. This represents an 83 per cent mortality.

Previous authors have commented on the variable spectrum of intravenous pyelographic changes.[3,4,13,26,27,28] They have described a variability of renal size, nephrogram density and excretory function. Generally, there is absent to poor visualization of the collecting system which frequently is distorted and stretched, mimicking polycystic kidney disease. Compression of the renal pelvis and proximal ureter by the edematous, boggy renal mass may be seen.[3,4,13,23,26,28] A summary of the intravenous pyelographic statistics in our 6 cases with acute renal vein thrombosis is presented in Table II.

The affected kidney was increased in size in most cases (Fig. 1); width was increased to a greater degree than length. In 50 per cent of the patients, the nephrogram was decreased as expected; however, in the other 50 per cent, it was increased

Table II

ACUTE RENAL VEIN THROMBOSIS: INTRAVENOUS PYELOGRAM DATA SUMMARY

(6 Cases: 4 Intravenous Pyelograms)

Renal Size	Length		Width			
Range						
Rt.	11.5–14.5		6.0–8.7			
Lt.	13.5–16.7		6.5–8.4			
Average						
Rt.	13.4		7.1			
Lt.	14.5		7.4			
Nephrogram	Decreased	2	Increased	2		
Pelvocalyceal System	Stretched	2	Not Seen	1	Dilated from obstruction	1
Ureters	Displaced	1	Notched	1	Not seen	2

(Fig. 2, *A* and *B*). We were able to visualize all but 1 pelvocalyceal system; 50 per cent of these were distorted and stretched (Fig. 2*B*; and 3).

Additional roentgenographic findings may be helpful: evidence of pulmonary embolism occurred in 50 per cent of our patients. Abdominal roentgenograms may reveal a mass such as an aneurysm or neoplasm in the region of the renal vein.

Chronic renal vein thrombosis occurs with gradual or incomplete occlusion of the renal venous drainage. A nephrotic syndrome is a frequent sequela.

McCarthy *et al.*[17] showed pain to be present in 13 of 40 patients (34 per cent). In 11 patients (29 per cent) there was thrombosis of the inferior vena cava, and in 14 cases (36 per cent), thromboembolic occlusion of a pulmonary artery.

Eleven of our 17 patients presented as chronic renal vein thrombosis. Table III summarizes the clinical data.

Seven of 11 patients were male; the average age was 56 years. Pain was a prominent feature in only 3 cases (27 per cent);

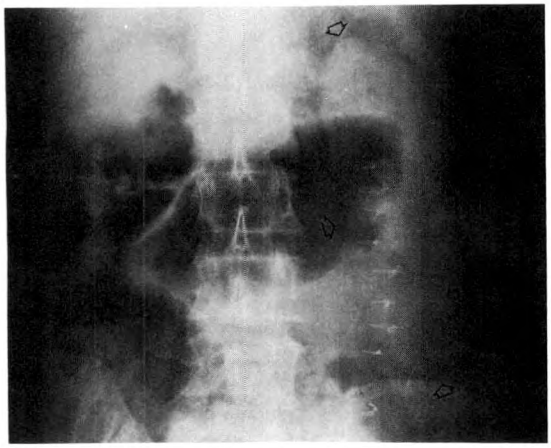

Fig. 1. A 64 year old male with marked left flank pain. Note increase in length and width of left kidney (open arrows).

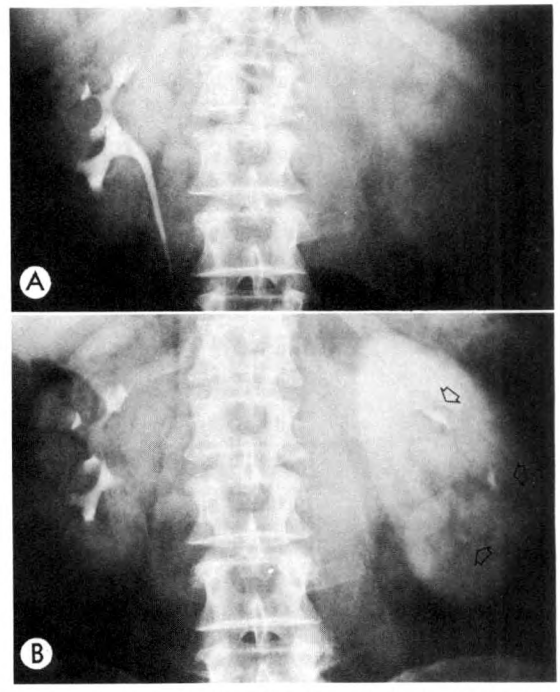

Fig. 2. A 63 year old male with left flank pain. Note increasingly dense nephrogram between (*A*) 15 minute film and (*B*) 5 hour film. Pelvocalyceal system stretched but not dilated (open arrows).

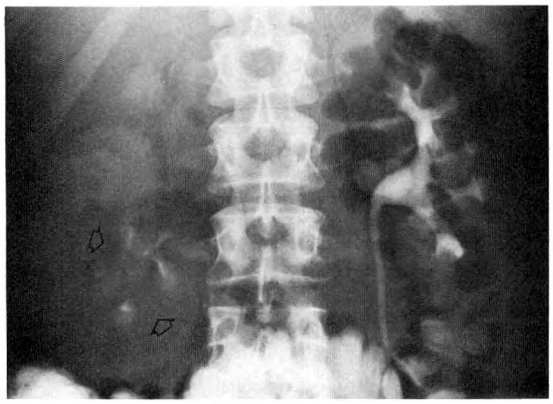

FIG. 3. A 28 year old female with right flank pain. Note stretched, distorted pelvocalyceal system.

however, edema and a palpable mass were both present in 7 cases (64 per cent). As in the patients with acute renal vein thrombosis, those with chronic renal vein thrombosis demonstrated a high incidence of pulmonary embolism—4 cases (36 per cent). This is consistent with the high clinical incidence of pleuritic chest pain, pleural effusion, and congestive heart failure in our patients. Primary renal disease was the apparent underlying etiology in 3 cases (27 per cent), trauma in 2 cases (19 per cent), and carcinomatosis with a positive coagulogram in 1 case (9 per cent). In the remaining 5 cases (45 per cent), compression of the renal venous drainage was caused by tumor (hypernephroma in 3, carcinoid in 1, and abdominal aortic aneurysm in 1). Of note is the fact that in only 1 of these latter patients (carcinoid) was the nephrotic syndrome present.

On physical examination the most frequent abnormality was leg edema. Dilated abdominal wall veins with upward blood flow indicative of inferior vena caval obstruction was seen in only 1 patient.

Proteinuria was the only consistently found laboratory abnormality. The urinalysis, however, may also demonstrate occasional red blood cells, white blood cells, granular or hyaline casts, and oval fat bodies.

Creatinine and blood urea nitrogen levels ranged from normal to moderately elevated.

As in the acute condition, various authors have described a variety of intravenous pyelographic appearances in chronic renal vein thrombosis.[3,4,13,27,26,28]

Table IV summarizes urographic data of the 10 of 11 patients with available studies.

Renal size was generally increased; the width, as in the acute stage, was increased to a greater degree than length (Fig. 4; and 5). The nephrogram was normal in 5 cases (50 per cent), decreased in 4 cases (40 per cent), and increased in only 1 case (10

TABLE III

CHRONIC RENAL VEIN THROMBOSIS: CLINICAL DATA SUMMARY

Patient	Age Sex	Primary Condition	Clinical Evidence	Thrombophlebitis	Nephrotic Syndrome	Tumor Invasion or Compression	Pulmonary Embolus
1.	15M	SLE	Pain, mass, edema	X (IVC occlusion)	X	O	X
2.	55F	Hypernephroma	Mass	O	O	X	O
3.	70F	Hypernephroma	Pain, mass	O	O	X	O
4.	42M	Trauma	Edema, mass	O	X	O	O
5.	52M	Trauma	Edema	X (IVC occlusion)	X	O	X
6.	72M	Diabetes mellitus	Edema	O	X	O	O
7.	51M	Carcinoid	Edema	X (IVC occlusion)	X	X	X
8.	48M	Hypertension	Pain, edema, mass	X	X	O	X
9.	75M	Hypernephroma	Mass	O	O	X	O
10.	68F	Positive coagulogram; carcinoma, uncertain primary	Edema	X	X	O	O
11.	63F	Abdominal aortic aneurysm	Mass	O	O	X	O

SLE = Systemic lupus erythematosus.
IVC = Inferior vena cava.

TABLE IV

CHRONIC RENAL VEIN THROMBOSIS: INTRAVENOUS PYELOGRAM DATA SUMMARY

(11 Cases, 10 Intravenous Pyelograms: 1 Report Only)

Renal Size		Length		Width	
Range					
Rt.		11.0–17.5		6.0–10.0	
Lt.		12.5–17.3		6.0–10.0	
Average					
Rt.		13.8		7.5	
Lt.		14.5		7.9	
Nephrogram	Normal 5	Decreased 4	Increased 1		
Pelvocalyceal System	Normal 2	Stretched 6	Not seen 1	Dilated secondary to	
Ureters	Normal 1	Not seen 3	Notched 6	obstruction	1

per cent). The pelvocalyceal system was normal in only 2 cases (20 per cent), and distorted and stretched in 6 cases (60 per cent) (Fig. 4; and 5). In 6 of 7 cases (86 per cent) visualized ureters were notched (Fig. 5; and 6).

Additional roentgenographic findings which may be helpful include the chest roentgenogram for evidence of pulmonary emboli or metastatic disease (55 per cent). A mass in the region of the kidney or renal vein may be evident on abdominal roentgenograms.

ADDITIONAL ROENTGENOGRAPHIC TECHNIQUES

Retrograde pyelography generally is indicated only in the face of an enlarged kidney without visualization of the collecting system on drip infusion intravenous pyelography. Weiner *et al.*[27] recently reviewed the findings in renal vein thrombosis using retrograde pyelography. Of note was the presence of an irregular pelvic outline in association with an irregular nodular-linear mucosal pattern. Others have noted the splayed collecting system, narrowing of the ureteropelvic junction, and parenchymal back diffusion of injected contrast material—all findings secondary to marked renal edema.[18,28]

Renal arteriography for several reasons is to be preferred as the initial confirmatory angiographic study.[3,26] With this study, one may evaluate, without the danger of dislodging a clot, the state of the renal paren-

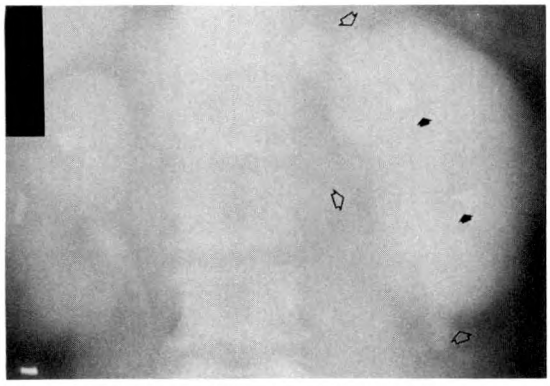

FIG. 4. A 40 year old male with the nephrotic syndrome. Note large, widened left kidney (open arrows). The renal outline is fuzzy. The pelvocalyceal system is distorted and stretched (closed arrows).

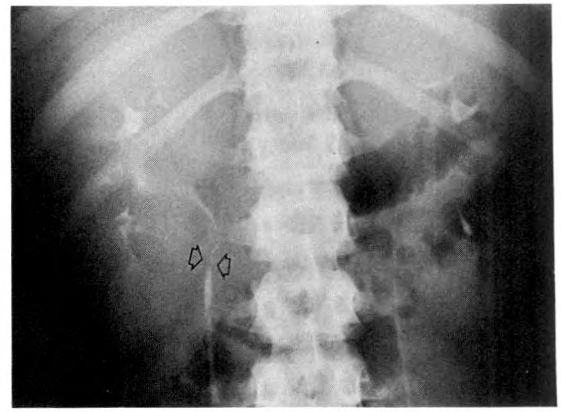

FIG. 5. A 15 year old male with the nephrotic syndrome. Note large kidneys with stretched pelvocalyceal systems. Ureteral notching is marked on the right (open arrows).

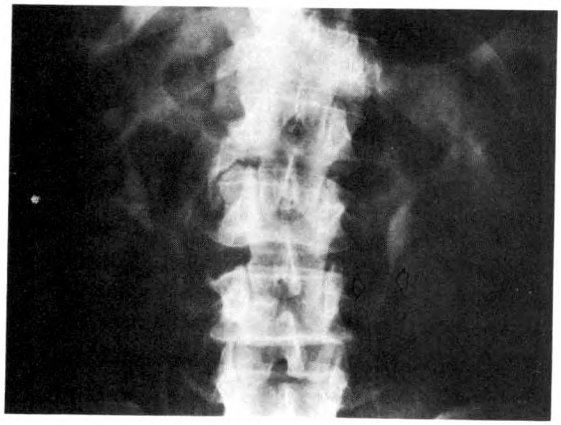

FIG. 6. A 53 year old male with the nephrotic syndrome. Note mildly stretched pelvocalyceal system and notched ureter on the left (open arrows).

chyma and renal blood flow. Also, with delayed filming, one may visualize the renal vein and possibly the site of obstruction. Collateral renal venous drainage may be seen. Arterial changes include decreased caliber of the arteries, decreased rate of blood flow, and deviation and stretching of the interlobar arteries.[13,26,28] The nephrographic phase may reveal loss of the normal distinction between the cortical and medullary blush.[3] In the chronic stage, these findings in the arterial and nephrographic stages may not be seen. Notching of the ureter secondary to collaterals may be documented.[2,3]

The more definitive, although somewhat hazardous, technique in doubtful cases is inferior vena cavography, with or without selective renal phlebography. Janower[14] emphasizes the role of femoral vein injection using an 18 gauge needle as a confirmatory measure. This not only reduces the hazards that occur with catheter employment but also allows clarification of the diagnosis with use of only basic equipment without specialized skills (Fig. 7). In both the acute and chronic case, renal vein thrombosis may be demonstrated by visualization of clot. The lack of wash-out in the renal veins is a suggestive finding. In selective renal phlebography, prolonged wash-out time associated with decreased renal vein branch filling is consistent with thrombosis of the smaller venous radicles.[9,14]

DISCUSSION

Renal vein thrombosis has a relatively grave prognosis. Treatment, although not clearly defined, can be undertaken only after having established a definitive diagnosis. Of particular interest is the relative infrequency with which the premortem diagnosis is definitely established.

The clinical presentation of renal vein thrombosis has been described in the literature as a spectrum. Yet Pollak *et al.*[20] concluded that in a significant proportion of patients, the syndrome is characteristic enough to permit a clinical diagnosis. Despite positive urographic and ancillary roentgenographic features in most cases,

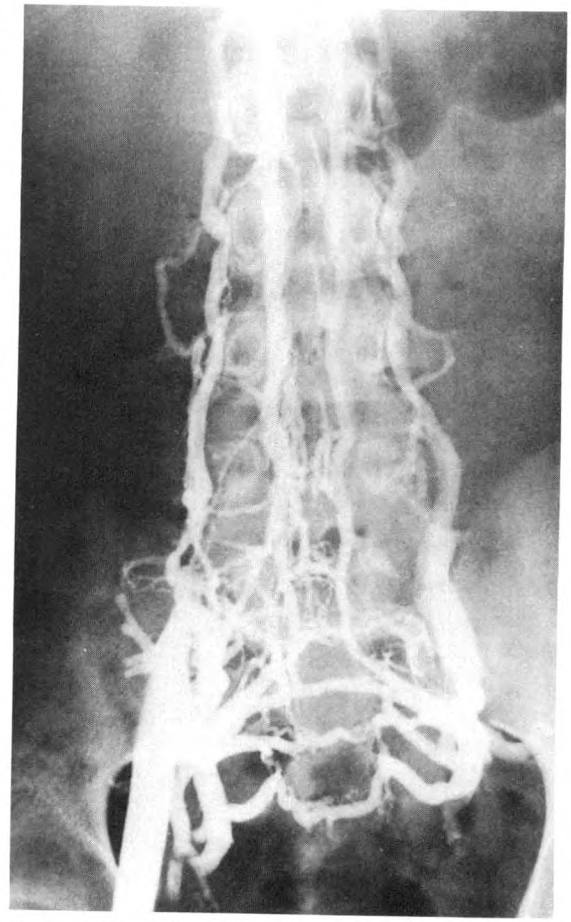

FIG. 7. Right femoral vein injection shows venous obstruction with extensive collaterals.

few authors have appreciated a characteristic roentgenographic picture of this entity. An analysis of our experience with 17 cases reveals on the contrary a significant degree of diagnostic specificity if particular attention is paid to the historical data, physical findings, and laboratory data in conjunction with the urographic observations.

The acute episode of renal vein thrombosis presents a rather classical picture. The outstanding features are: (1) pain in the lower back or flank usually radiating to the groin area; (2) frequently a palpable mass; (3) associated edema in the lower extremities; (4) hematuria; (5) proteinuria; and (6) frequently extrarenal thromboembolic episodes. Urographically, the kidney appears congested, with increased dimensions. There may be no renal contrast medium excretion on the involved side (or sides). More commonly, a nephrogram is seen and, although faintly visualized, the collecting system is stretched.

At this point, utilizing available clinical and roentgenographic information considerably narrows the diagnosis. Obstructive uropathy secondary to a urinary tract calculus is a frequent consideration but should not produce proteinuria, peripheral edema, or thromboembolic episodes if these are present. A stretched pelvocalyceal system, when visible, is also inconsistent with this diagnosis. Other diagnostic considerations such as renal artery occlusion or perinephric abscess are also unlikely to present with a clinical and roentgenographic appearance which might be confused with renal vein thrombosis. When this disease is suspected, inferior vena cavography or arteriography will easily confirm the diagnosis.

In chronic renal vein thrombosis associated with the nephrotic syndrome, the diagnosis is usually less clear-cut and the differential diagnosis includes essentially all the causes of the nephrotic syndrome. Typically the patient presents with a previous history of flank pain or thromboembolic episodes. Physical evidence of inferior vena caval obstruction, such as collateral abdominal veins or lower trunk and leg edema, may be observed. The presence of an underlying disorder associated with increased incidence of renal vein thrombosis is supportive evidence (Table v).

Table V

REPORTED CONDITIONS ASSOCIATED WITH RENAL VEIN THROMBOSIS

1.	Infectious
	Pyelonephritis
	Ascending phlebitis
	Ileocolitis
	Phycomycosis (particularly with lymphoma, leukemia)
2.	Neoplasm
	Hypernephroma
	Transitional cell carcinoma, renal pelvis
	Primary carcinoma elsewhere (bronchogenic carcinoma, breast carcinoma, hepatoma)
3.	Circulatory Disorder
	MI; CHF
	Cyanotic and polycythemic conditions
	Sickle cell anemia
4.	Systemic Disorders
	Amyloidosis
	Diabetic glomerulosclerosis
	Renal hypertension
5.	Trauma
6.	Surgery
	Splenorenal shunt
	Transplant
	Indwelling catheter in IVC
7.	Other
	Idiopathic
	Papillary necrosis
	Post partum
	Consumptive coagulopathy
	Glomerulonephritis, membranous

MI = Myocardial infarct.
CHF = Congestive heart failure.
IVC = Inferior vena cava.

Urographically, the involved kidney may demonstrate the following: (1) slight increase in size; (2) normal or slightly decreased concentration of contrast material by the kidney; (3) a stretched collecting system; and (4) ureteral notching. It is important to emphasize that these findings may be minimal.

It is not feasible in the present framework to list all possible causes of the nephrotic syndrome and its corresponding

clinical and urographic picture to differentiate it from that seen in chronic renal vein thrombosis. However, using a modification of the etiologic scheme of the nephrotic syndrome as proposed by Schreiner,[24] one can eliminate several categories rather simply. For example, historical data should reveal exposure to nephrotoxins, allergens, or harmful drugs. The clinical history and laboratory data should expose an infectious etiology of the nephrotic syndrome. Various metabolic diseases presenting with a nephrotic syndrome, such as diabetic glomerulosclerosis, will generally have associated abnormal laboratory values, such as an abnormal glucose tolerance test. Urography will commonly exhibit small shrunken kidneys bilaterally.

Amyloidosis may be a very difficult diagnosis to exclude. The physical examination rarely reveals evidence of amyloid deposition elsewhere. Rectal biopsy is a safe confirmatory procedure. Symmetrical urographic changes are likely and ureteral notching is not seen.

Various other systemic diseases noted by Schreiner[24] may also cause the nephrotic syndrome. By virtue of the fact that they are systemic diseases, evidence of the pathologic process should be clinically present elsewhere. Sickle cell anemia, for example, will present in a young Black with a positive sickle cell preparation. Essential hypertension can be suspected on the basis of severe hypertension and other evidence of systemic vascular disease, such as eyeground changes.

Glomerular disease, one of the most frequent causes of the nephrotic syndrome, must also be excluded. Recent sore throat with fever or a positive ASO titer is consistent with glomerulonephritis. Hematuria, the hallmark of glomerulonephritis, is a prominent finding in renal vein occlusion only during the acute stage. The urographic findings of acute renal vein thrombosis are not present in glomerulonephritis.

In those patients with chronic renal vein thrombosis not associated with a nephrotic syndrome, tumor is the most likely etiologic factor. Hypernephroma or renal pelvic carcinoma, the most frequent neoplasms to invade the renal vein, should be recognized roentgenographically. Compression or invasion of the renal vein or vena cava by tumor may be suspected on the urogram with ureteral notching.

As is the case in many other diseases, the clinical and urographic picture of renal vein thrombosis is sufficiently characteristic to establish the diagnosis. A high index of suspicion on the part of the clinician or the radiologist will lead to the diagnosis in most instances. Confirmatory studies, particularly modified inferior vena cavography, may be undertaken immediately. Arteriography will yield further confirmatory information.

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REFERENCES

1. Abeshouse, B. S. Thrombosis and thrombophlebitis of renal vein. *Urol. & Cutan. Review*, 1945, *49*, 661–675.
2. Chait, A., Matasar, K. W., Fabian, C. E., and Mellins, H. Z. Vascular impressions on ureters. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *111*, 729–749.
3. Chait, A., Stoane, L., Moskowitz, H., and Mellins, H. Z. Renal vein thrombosis. *Radiology*, 1968, *90*, 886–896.
4. Coel, M. N., and Talner, L. B. Obstructive nephrograms due to renal vein thrombosis. *Radiology*, 1971, *101*, 573–574.
5. Cohn, L. H., Lee, J., Hopper, J., and Najarian, J. S. Treatment of bilateral renal vein thrombosis and nephrotic syndrome. *Surgery*, 1968, *64*, 387–396.
6. Cox, J. S. J., John, H. T., Bankole, M. A., and Warren, R. Collateral circulation after renal vein occlusion. *Surgery*, 1962, *52*, 875–882.
7. Crummy, A. B., and Hipona, F. A. Roentgen diagnosis of renal vein thrombosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *93*, 898–903.
8. Derow, H. A., Schlesinger, M. J., and Savitz, H. A. Chronic progressive occlusion of inferior vena cava and renal and portal veins with clinical picture of nephrotic syndrome. *Arch. Int. Med.*, 1939, *63*, 626.
9. Eisen, S., Friedenberg, M. J., and Klahr, S.

Bilateral ureteral notching and selective renal phlebography in nephrotic syndrome due to renal vein thrombosis. *J. Urol.*, 1965, *93*, 343–346.

10. Fein, R. L., Chait, A., and Levitan, A. Renal vein thrombectomy for treatment of renal vein thrombosis associated with nephrotic syndrome. *J. Urol.*, 1968, *99*, 1–13.
11. Harrison, C. V., Milne, M. D., and Steiner, R. E. Clinical aspects of renal vein thrombosis. *Quart. J. Med.*, 1956, *25*, 285–298.
12. Hasson, J., Berkman, J. I., Parker, J. G., and Rifkin, H. Clincopathologic study of chronic renal vein thrombosis in adults. *Ann. Int. Med.*, 1957, *47*, 493–517.
13. Hipona, F. A., and Crummy, A. B. Roentgen diagnosis of renal vein thrombosis: clinical aspects. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 122–131.
14. Janower, M. L. Nephrotic syndrome secondary to renal vein thrombosis: value of inferior vena cavography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *95*, 330–334.
15. Koehler, P. R., Bowles, W. T., and McAlister, W. H. Renal arteriography in experimental renal vein occlusion. *Radiology*, 1966, *86*, 851–855.
16. March, T. L., and Halpern, M. Renal vein thrombosis demonstrated by selective renal phlebography. *Radiology*, 1963, *81*, 958–962.
17. McCarthy, L. J., Titus, J. L., and Daugherty, G. W. Bilateral renal vein thrombosis and nephrotic syndrome in adults. *Ann. Int. Med.*, 1963, *58*, 837–857.
18. Melick, W. F., and Witt, A. E. Thrombosis of renal vein. *J. Urol.*, 1944, *51*, 587–596.
19. Morris, J. F., Ginn, H. E., and Thompson, D. D., Unilateral renal vein thrombosis associated with nephrotic syndrome. *Am. J. Med.*, 1963, *34*, 867–874.
20. Pollak, V. E., Kark, R. M., Pirani, C. L., Shafter, H. A., and Muehrcke, R. C. Renal vein thrombosis and nephrotic syndrome. *Am. J. Med.*, 1956, *21*, 496–520.
21. Rayer, P. F. O. Traite des maladies des reins. J. B. Balliére, Paris, 1840.
22. Renert, W. A., Rudin, L. J., and Casarella, W. J. Renal vein thrombosis in carcinoma of renal pelvis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *114*, 735–740.
23. Scanlon, G. T. Radiographic changes in renal vein thrombosis. *Radiology*, 1963, *80*, 208–211.
24. Schreiner, G. E. Nephrotic syndrome. In: Disease of the Kidney. Edited by M. B. Strauss and L. G. Welt. Little, Brown & Company, Boston, 1971.
25. Sturgill, B. C., and Rowe, C. T. Renal vein thrombosis and nephrotic syndrome. *A.M.A. Arch. Int. Med.*, 1967, *120*, 708–711.
26. Wegner, G. P., Crummy, A. B., Flaherty, T. T., and Hipona, F. A. Renal vein thrombosis: roentgenographic diagnosis. *J.A.M.A.*, 1969, *209*, 1661–1667.
27. Weiner, P. L., Lim, M. S., Knudson, D. H., and Semerdjian, H. S. Retrograde pyelography in renal vein thrombosis. *Radiology*, 1974, *111*, 77–79.
28. Zheutlin, N., Hughes, D., and O'Loughlin, B. J. Radiographic findings in renal vein thrombosis. *Radiology*, 1959, *73*, 884–889.

CONTROL OF RENAL HEMORRHAGE BY SELECTIVE ARTERIAL EMBOLIZATION*

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ABSTRACT:

An attempt was made to control renal hemorrhage by selective embolization of the renal artery in 7 patients. In 2 patients the bleeding site was single, but in 2 others, several sites of arterial extravasation were present. Two patients had arteriovenous fistulas, and 1 had an unsalvageable, macerated kidney. Six of the 7 patients (86 per cent) were treated successfully. Nephrectomy was avoided in 5. No patient developed hypertension during the follow-up of 4 months to 2 years. Our results in these patients indicate that selective arterial embolization should have an important place in the management of patients with renal trauma.

ANGIOGRAPHY is the most accurate method for evaluating patients with renal trauma.[8,10,13] Selective renal angiography allows diagnosis of injury to the renal pedicle, arteriovenous fistula, parenchymal vascular disruption, infarction, and perinephric hematoma. Recently, renal arterial embolization has been used to control post-traumatic renal hemorrhage in humans[3,11,12] and in experimental animals.[4] Including our preliminary reports of 2 patients,[3,11] we have attempted embolic control of renal hemorrhage in 7 patients and have been successful in 6. In the evaluation and treatment of these patients we have encountered a variety of problems, including multiple renal bleeding sites, large arteriovenous fistulas, and an unsalvageable kidney.

REPORT OF CASES

Patients with a single site of hemorrhage.

CASE I. A 15 year old boy was admitted with gross hematuria caused by blunt abdominal injury. An intravenous urogram showed decreased function of the left kidney. Blood clots were present in the left renal pelvis. The vital signs were stable and he was treated with bed rest and observation. His hematuria improved initially, but then became more severe. A renal angiogram 9 days after hospitalization revealed a 2 cm. false aneurysm in the midportion of the left kidney (Fig. 1*A*). The catheter was selectively positioned into the bleeding segmental artery and 0.5 cc. of autogenous clot was injected. A repeat study showed the bleeding artery to be completely occluded (Fig. 1*B*). The patient had slight flank pain for 2 hours and his hematuria gradually cleared over 2 days. An angiogram 5 days after embolization revealed that the previously occluded branch was again patent except for a small residual thrombus. Another angiogram 3 months later showed very slight narrowing of the previously embolized segmental artery but normal interlobar arteries and nephrogram (Fig. 1*C*). The false aneurysm remained thrombosed, and there has been no recurrence of the hematuria. He has remained normotensive during 2 years of follow-up.

CASE II. A 35 year old man developed gross hematuria after falling from a roof. An intravenous urogram revealed poor visualization of the right upper calyces. His blood pressure was 108/70 mm. Hg initially. During the following week of observation his hematocrit fell in spite of several transfusions, and the hematuria persisted. A renal angiogram demonstrated a fracture in the lower pole of the right kidney with a pseudoaneurysm in direct communication with a calyx. Selective injection of

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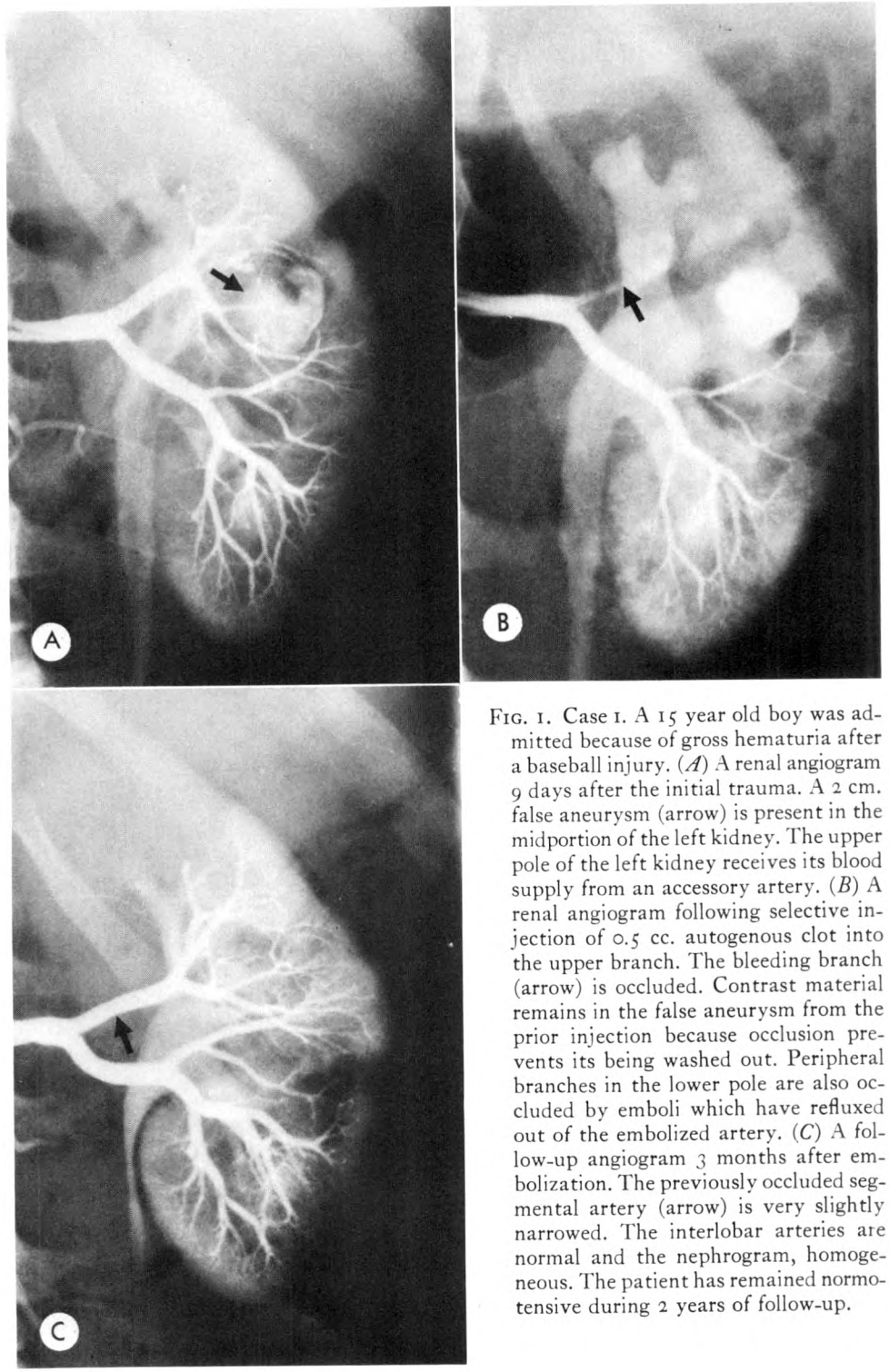

Fig. 1. Case 1. A 15 year old boy was admitted because of gross hematuria after a baseball injury. (*A*) A renal angiogram 9 days after the initial trauma. A 2 cm. false aneurysm (arrow) is present in the midportion of the left kidney. The upper pole of the left kidney receives its blood supply from an accessory artery. (*B*) A renal angiogram following selective injection of 0.5 cc. autogenous clot into the upper branch. The bleeding branch (arrow) is occluded. Contrast material remains in the false aneurysm from the prior injection because occlusion prevents its being washed out. Peripheral branches in the lower pole are also occluded by emboli which have refluxed out of the embolized artery. (*C*) A follow-up angiogram 3 months after embolization. The previously occluded segmental artery (arrow) is very slightly narrowed. The interlobar arteries are normal and the nephrogram, homogeneous. The patient has remained normotensive during 2 years of follow-up.

several aliquots of 0.1 cc. autogenous clot mixed with oxycel (Parke, Davis) failed to control the bleeding. The patient became restless and further embolization with a larger amount of clot was not attempted. He had a nephrectomy 2 days later.

Patients with multiple bleeding sites.

Case III. An 18 year old man had right flank pain and gross hematuria after a motorcycle accident. His blood pressure on admission to a local hospital was 100/60 mm. Hg. The intravenous urogram showed poor visualization of the right upper calyces. The hematuria persisted and his hematocrit level fell from 47 to 33 gm. per cent over 8 days of observation. His blood pressure rose to 170/100 mm. Hg. After transfer to the University of Michigan Medical Center, a repeat intravenous urogram revealed extravasation of contrast medium from the right kidney. A renal angiogram showed 3 distinct areas of extravasated contrast medium (Fig. 2*A*). Two of these bleeding sites originated from a ventral branch and were successfully controlled with 0.5 cc. of Amicar-mixed blood clot (Fig. 2*B*). Because of stenosis and torsion of the origin of the dorsal segmental branch, however, the third, small bleeding point could not be embolized. His hematuria cleared in 3 days and his blood pressure stabilized at 130/80 mm. Hg. He remained asymptomatic and was normotensive (120–130/80–90 mm. Hg). A repeat renal angiogram 3 months later showed that the previously embolized segmental artery was patent but had decreased in diameter (Fig. 2*C*). The lower pole of the left kidney was also smaller, probably because of infarction. Renin assays revealed no difference between kidneys (right renal vein 4.6 ng./ml./hr., left renal vein 4.2 ng./ml./hr., and the inferior vena cava 2.7 ng./ml./hr.).

Case IV. A 16 year old boy received a shotgun wound to the right chest and abdomen. The positive findings on admission included a 20 per cent right pneumothorax, gross hematuria, and multiple buckshot pellets in the right upper quadrant of the abdomen and right upper extremity. His blood pressure was 120/80 mm. Hg. Abdominal exploration revealed nonbleeding lacerations of the right lobe of the liver and 30–40 pellet injuries to the right kidney, none of which was bleeding. Postoperatively, he continued to have hematuria and required several transfusions. A renal angiogram on the fourth postoperative day demonstrated several bleeding sites in the lower pole of the right kidney. Several injections of approximately 1.5 cc. of autogenous clot were made into the bleeding branches. The repeat angiogram revealed successful control of the bleeding, as well as areas of decreased and absent concentration in the nephrogram secondary to the emboli. An angiogram 1 week later showed areas of infarction but no active bleeding. A pseudoaneurysm and an arteriovenous fistula had developed in the mid portion of the kidney. No further treatment was given. His hematuria gradually cleared in 10 days after arterial embolization. He did well clinically and was discharged. A follow-up renal angiogram 5 months later revealed that the false aneurysm and arteriovenous fistula were still present but had decreased significantly in size. The right kidney had decreased 0.5 cm. in its greatest length. The results of renin assays at this time were: right renal vein 10.96 ng./ml./hr.; left renal vein 5.68 ng./ml./hr.; upper vena cava 6.29 ng./ml./hr.; and lower vena cava 6.0 ng./ml./hr. The right renal vein renin value was 93 per cent higher than the left. His blood pressure was 130/70 mm. Hg. He was discharged to be followed as an out-patient.

Patients with arteriovenous fistulas.

Case V. A 25 year old pregnant woman was admitted because of toxemia. She had a blood pressure of 200/120 mm. Hg, leg edema, uremia, proteinuria, and positive LE preparations. She responded poorly to treatment. A percutaneous renal biopsy of the lower pole of the right kidney revealed severe lupus nephritis. Following the biopsy she developed hematuria which continued for 8 days. Several units of blood were required because of unstable vital signs. A renal angiogram 8 days after biopsy revealed 2 arteriovenous fistulas in the lower pole of the right kidney. After selective catheterization of the artery supplying both fistulas, 1.0 cc. autogenous clot was injected. A repeat angiogram revealed occlusion of both arteriovenous fistulas. The hematuria ceased abruptly. The patient had slight flank pain for 2 hours. A follow-up angiogram 5 days later showed that all the occluded branches were again patent. No residual thrombus or arteriovenous fistula was seen. The patient's hypertension persisted and she died a month later of cerebral involvement and terminal pneumonia. An autopsy showed a small infarct in the lower pole of the

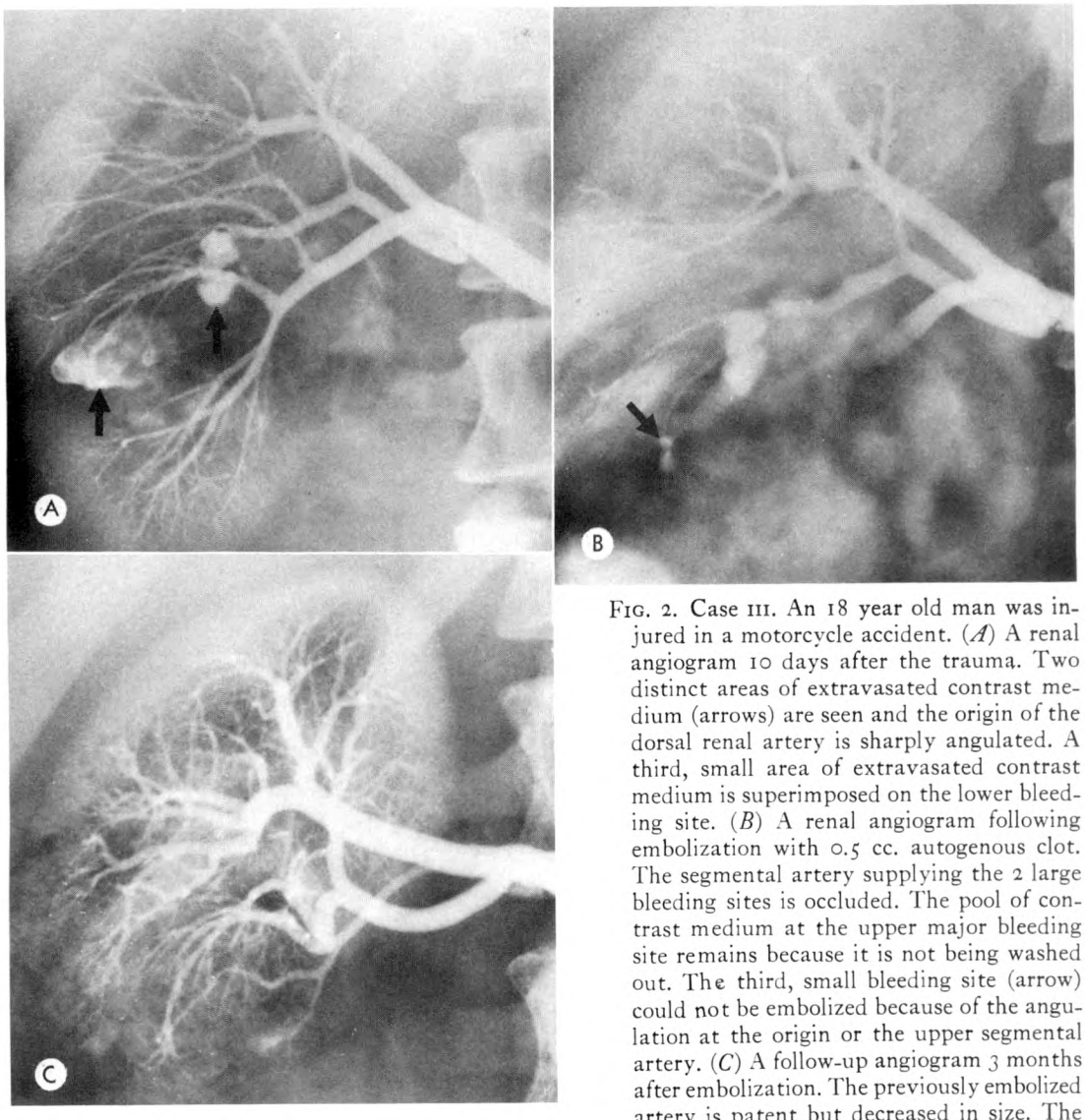

Fig. 2. Case III. An 18 year old man was injured in a motorcycle accident. (*A*) A renal angiogram 10 days after the trauma. Two distinct areas of extravasated contrast medium (arrows) are seen and the origin of the dorsal renal artery is sharply angulated. A third, small area of extravasated contrast medium is superimposed on the lower bleeding site. (*B*) A renal angiogram following embolization with 0.5 cc. autogenous clot. The segmental artery supplying the 2 large bleeding sites is occluded. The pool of contrast medium at the upper major bleeding site remains because it is not being washed out. The third, small bleeding site (arrow) could not be embolized because of the angulation at the origin or the upper segmental artery. (*C*) A follow-up angiogram 3 months after embolization. The previously embolized artery is patent but decreased in size. The nephrogram phase revealed renal infarction in the lower pole. The embolized bleeding sites remained occluded and the small, non-embolized bleeding site had closed spontaneously. The patient has remained asymptomatic and normotensive despite the renal infarction from embolization.

right kidney but no residual thrombus or arteriovenous fistulas.

Case VI. A 33 year old man was knifed in the right posterior chest. In addition to a 20 per cent right pneumothorax, successfully treated with a chest tube, he had gross hematuria. The intravenous urogram revealed decreased function of the right kidney with clot in the renal pelvis and upper ureter. The inferior pole of the right kidney was displaced laterally by a retroperitoneal hematoma. An exploratory celiotomy showed a large right perinephric and periduodenal hematoma, but no injury to the liver, bile ducts, pancreas, or intestine. He was treated conservatively, but his hematuria persisted. A renal angiogram demonstrated a 2 cm. pseudoaneurysm in the upper pole of the right kidney associated with a large arteriovenous fistula (Fig. 3*A*). An injection of 0.5 cc. Amicar clot passed through the fistula. A 5×5×20 mm. Gelfoam embolus was then injected and a repeat angiogram demonstrated nearly complete occlusion of the artery (Fig. 3*B*). A

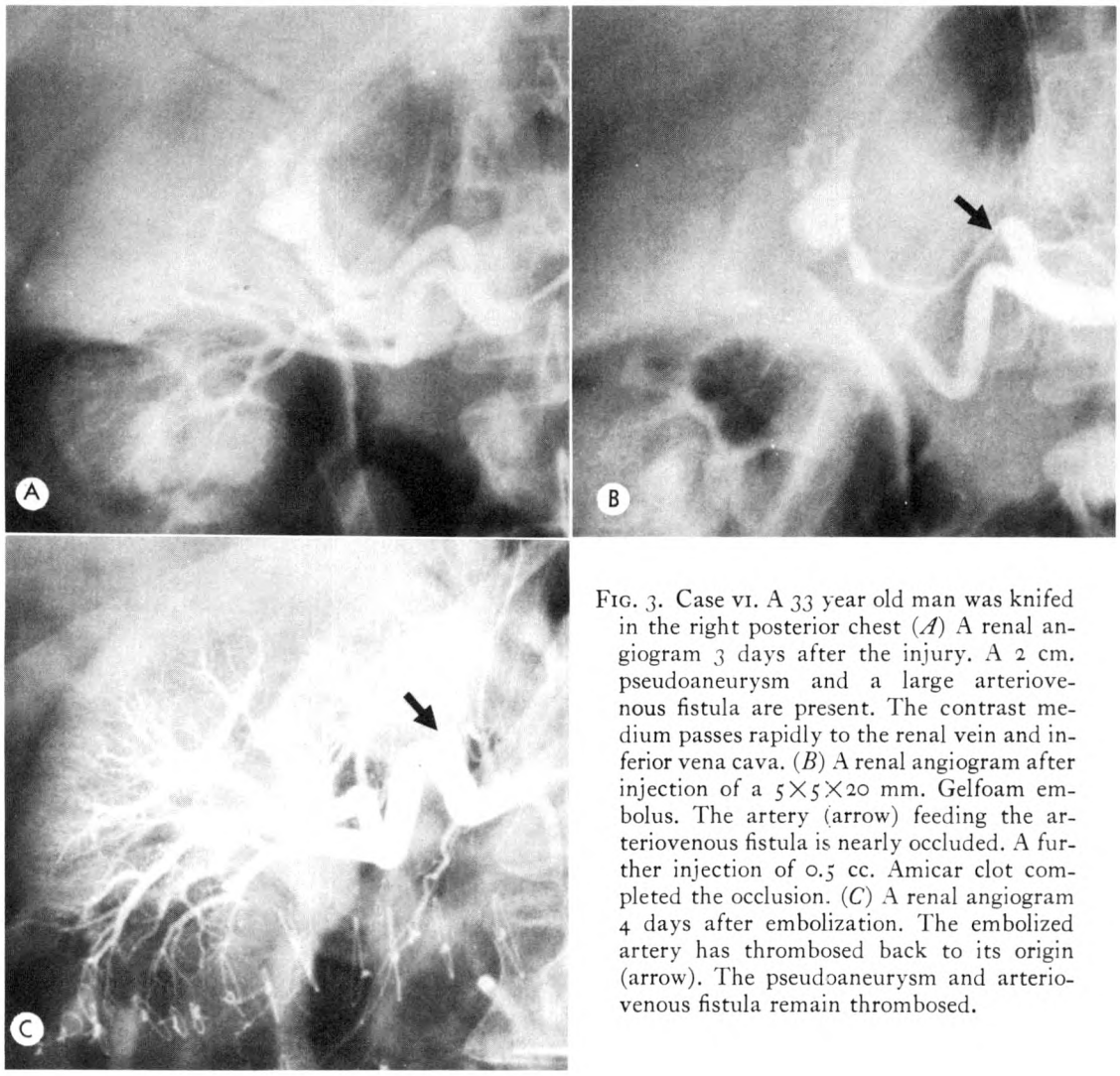

Fig. 3. Case vi. A 33 year old man was knifed in the right posterior chest (*A*) A renal angiogram 3 days after the injury. A 2 cm. pseudoaneurysm and a large arteriovenous fistula are present. The contrast medium passes rapidly to the renal vein and inferior vena cava. (*B*) A renal angiogram after injection of a 5×5×20 mm. Gelfoam embolus. The artery (arrow) feeding the arteriovenous fistula is nearly occluded. A further injection of 0.5 cc. Amicar clot completed the occlusion. (*C*) A renal angiogram 4 days after embolization. The embolized artery has thrombosed back to its origin (arrow). The pseudoaneurysm and arteriovenous fistula remain thrombosed.

further 0.5 cc. Amicar clot completed the occlusion. The color of the urine changed immediately from red to light pink and became clear over 2 days. A renal angiogram 4 days later showed thrombosis of the embolized artery at its origin, with obliteration of the pseudoaneurysm and arteriovenous fistula (Fig. 3*C*). The patient did not experience any pain during the embolization. No further treatment was given; he became asymptomatic, was discharged 1 week later, and has remained normotensive during 4 months of follow-up.

Patient with an unsalvageable kidney.

Case vii. An 18 year old woman who injured her left back in an automobile accident was admitted in shock. Her blood pressure was stabilized with 4 units of blood. An intravenous urogram demonstrated transection of the left kidney with perinephric extravasation of contrast medium. Only the upper pole functioned. The left calyces and pelvis were not seen. Because of transection of the renal pelvis, however, she had no hematuria. An emergency angiogram revealed transection of the left kidney with multiple sites of extravasated contrast medium in the upper pole. During the angiography, her vital signs became unstable. Selective arterial embolization was used to control the renal hemorrhage. An injection of 0.3 cc. autogenous clot was made into the only remaining upper pole renal artery and a repeat angiogram showed successful control of bleeding. Her blood pressure and hematocrit level became

stable for the next 48 hours, but she then developed a temperature of 101 degrees, presumable due to retroperitoneal leakage of urine. A nephrectomy was done 4 days after embolization. The transected kidney and the perinephric hematoma had formed a solid mass, and the surgeons considered the nephrectomy much easier than one performed in the face of active bleeding. The pathological examination revealed a severe renal crush injury, but residual thrombi were not demonstrated in any of the arteries.

DISCUSSION

The treatment of renal trauma has generally been conservative. However, Scott *et al.*[13] have recommended an increased use of angiography and an aggressive operative approach in patients with penetrating renal injuries. Angiography is advocated not only because of the high incidence of false negative results at intravenous urography, but also because of its high accuracy in detecting injury to the renal pedicle and major parenchymal wounds. Moreover, as shown by our patients, the catheter can usually be advanced into a bleeding segmental artery and the bleeding controlled by selective embolization. Even if the kidney is so severely damaged that it is not salvageable, embolization can be used to achieve immediate hemostasis, allowing an emergency nephrectomy to be transformed to an elective procedure.

We prefer autogenous blood clot as the embolic material for renal artery embolization because of its relatively short duration of occlusion. Since renal arteries are end arteries, the ideal embolic occlusion should last long enough to control the bleeding but not long enough to cause infarction. It has been shown experimentally that most autogenous blood clot emboli lyse from normal segmental and interlobar renal arteries in a few hours,[2,4] although they seem to persist longer in areas of injury.

When several bleeding sites are present, control of all the bleeding sites is more difficult. However, since laparotomy for persistent bleeding in these patients generally results in a nephrectomy, aggressive attempts at transcatheter hemostasis are justified.

In Case III the patient's hematuria cleared and his hypertension dramatically abated even though only 2 of the 3 active bleeding sites were controlled. In Case IV, several bleeding sites were controlled by using a relatively large amount of autogenous clot. Although the kidney was smaller on a follow-up angiogram, the hematuria was controlled and the patient continued to be normotensive on follow-up. Thus, both patients with multiple bleeding sites were successfully treated by selective embolization.

Both patients with arteriovenous fistula were treated successfully. The small arteriovenous fistula in Case V was occluded with a single injection of autogenous clot. The large arteriovenous fistula with a false aneurysm in Case VI was more of a problem. Because of the large communication, the fragments of blood clot were swept through the fistula. Therefore, a long piece of Gelfoam was used to occlude the fistula. Gelfoam can be cut into the desirable size and shape, soaked and softened in solution, then squeezed through a catheter in one piece.

Case VII presented a different problem. In addition to renal transection, the lack of hematuria in this patient indicated severance of the renal pelvis or ureter. Thus, the kidney was probably unsalvageable. Since a transfusion of 4 units of blood within a period of several hours failed to control her vital signs, the active bleeding demonstrated by renal angiography was judged life threatening. In such a situation, immediate hemostasis by transcatheter embolization is simple and rapid, and allows the nephrectomy to be performed electively.

An important finding in this group of patients is that none developed hypertension. Five of the 7 had had normal blood pressures before and after renal artery embolization, and have remained normotensive for 4 months to 2 years of follow-up. One patient (Case V) was hypertensive before the embolization and her blood pres-

sure remained elevated after embolization until death. The other patient (Case III), who was initially normotensive, developed hypertension a week after renal trauma, but returned to normal 5 days after embolic control of the renal bleeding. Also in support of this encouraging finding, Kelley *et al.*[9] and Chuang and associates[4] have noted that experimental renal arterial embolization in dogs does not cause hypertension for periods up to 2 months. Also, Almgård *et al.*,[1] noted that their patients remained normotensive following embolization of renal adenocarcinomas. Since the work of Goldblatt *et al.*,[6] much research has been done in the field of renovascular hypertension. Although questions remain unanswered, there is general agreement that embolic infarction of a renal segment or the entire kidney need not produce hypertension. A recent study by Goldin *et al.*[7] has shown in experimental animals that complete renal infarction with selectively injected barium failed to cause hypertension. However, the development of collateral circulation which results in ischemic but viable tissue generally causes hypertension.[14]

The renin values in the traumatized and embolized kidneys are interesting. In Case II, no significant difference of the renin level was noted 3 months after the trauma and embolization. In Case IV, the renin level was higher in the embolized kidney than in the non-traumatized kidney but the patient remained normotensive. The elevated renin level could result from renal trauma itself, the arterial embolization, or the residual arteriovenous fistula. Since most of our patients remained normotensive and asymptomatic, the renal vein renin assay was not done as part of the follow-up study. A determination of renin levels will be part of subsequent follow-up studies.

The failure of treatment in Case II was, in retrospect, probably due to an inadequate quantity or size of emboli. The study was done early in the series, and only a small quantity of clot was used. Consequently, the patient lost his kidney. A larger clot probably would have resulted in successful control of the bleeding. We now believe that a more aggressive approach to obtain hemostasis by arterial embolization is justified.

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REFERENCES

1. Almgård, L. E., Fernström, I., Haverling, M., and Lundqvist, A. Treatment of renal adenocarcinoma by embolic occlusion of renal circulation. *Brit. J. Urol.*, 1973, *45*, 474–479.
2. Bookstein, J. J., Chlosta, E. M., Foley, D., and Walter, J. F. Transcatheter hemostasis of gastrointestinal bleeding using modified autogenous clot. *Radiology*, 1974, *113*, 277–285.
3. Bookstein, J. J., and Goldstein, H. M. Successful management of postbiopsy arteriovenous fistula with selective arterial embolization. *Radiology*, 1973, *109*, 535–536.
4. Chuang, V. P., Reuter, S. R., and Schmidt, R. W. Control of experimental traumatic renal hemorrhage by embolization with autogenous blood clot. Presented at the 60th Annual Meeting of RSNA, Chicago, December, 1974.
5. Dowse, J. L. A., and Kihn, R. B. Renal injuries: diagnosis, management and sequelae in 67 cases. *Brit. J. Surg.*, 1963, *50*, 353–361.
6. Goldblatt, H., Lynch, J., Hanzal, R. F., and Summerville, W. W. Production of persistent hypertension in dogs. *Am. J. Path.*, 1933, *9*, 942–945.
7. Goldin, A. R., Naude, J. H., and Hickman, R. Experimental percutaneous renal infarction. *Brit. J. Urol.*, 1974, *46*, 127–131.
8. Halpern, M. Angiography in renal trauma. *Surg. Clin. North America*, 1968, *48*, 1221–1233.
9. Kelley, K. M., Craven, J. D., and Jorgens, J. Experimental thromboembolism of renal artery. *Invest. Radiol.*, 1974, *9*, 327.
10. Morrow, J. W., and Mendez, R. Renal trauma. *J. Urol.*, 1970, *104*, 649–653.
11. Reuter, S. R., and Chuang, V. P. Control of abdominal bleeding with autogenous embolized material. *Radiologe*, 1974, *14*, 86–91.
12. Rizk, G. K., Atallah, N. K., and Bridi, G. I. Renal arteriovenous fistula treated by catheter embolization. *Brit. J. Radiol.*, 1973, *46*, 222–224.
13. Scott, R., Jr., Carlton, C. E., Jr., and Goldman, M. Penetrating injuries of kidney: analysis of 181 patients. *J. Urol.*, 1969, *101*, 247–253.
14. Yuile, C. L. Obstructive lesions of main renal artery in relation to hypertension. *Am. J. M. Sc.*, 1944, *207*, 394–404.

Something else.

What you see in the radiograph on page xviii is just a coin placed on the patient's posterior right hemithorax. It does bear a resemblance to the coin lesion produced by a benign solitary lesion, perhaps a hamartoma or tuberculoma.

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CIRCUMAORTIC RENAL COLLAR*

A POTENTIALLY HAZARDOUS ANOMALY OF THE LEFT RENAL VEIN

By HAROLD A. MITTY, M.D.
NEW YORK, NEW YORK

ABSTRACT:

The circumaortic renal collar is a potentially hazardous anomaly of the left renal vein. Failure to recognize the dorsal component during retroperitoneal surgery may lead to hemorrhage, nephrectomy or death.

The angiographer can make a significant contribution in documenting this anomaly in patients who are candidates for surgery in this area.

THE left renal vein usually passes anterior to the aorta to join the vena cava. Rarely, a portion of the venous drainage passes through an additional vein posterior to the aorta. The resulting venous ring has been described as a circumaortic renal collar (Fig. 1).[2-6] Failure to recognize this anomaly at surgery may lead to disruption of these veins with accompanying serious hemorrhage.[1,2,7] It is particularly important for the radiologist to recognize the flow pattern associated with this anomaly during fluoroscopic test injections so that the characteristic venographic pattern may be documented.

It is the purpose of this paper to describe our experience with three patients with this anomaly.

REPORT OF CASES

CASE 1. A 45 year old female was admitted for the evaluation of hypertension. Physical examination was within normal limits with the exception of a blood pressure of 160/95 mm. Hg. Routine laboratory studies and excretory urography were normal. Aortography revealed a mild degree of medial fibroplasia in the right renal artery. This was confirmed by selective arteriography (Fig. 2*A*). Venous catheterization was performed in order to obtain blood samples for renin assay. During the course of left renal vein catheterization, an abnormal flow pattern was noted fluoroscopically after a test injection. A portion of the contrast material flowed in a vein in an inferior and then medial direction to enter the vena cava at L-3. Some of the contrast material flowed in a normal fashion to enter the vena cava at L-1. Roentgenograms revealed the typical pattern of a circumaortic renal collar (Fig. 2*B*). Renal venous renin as-

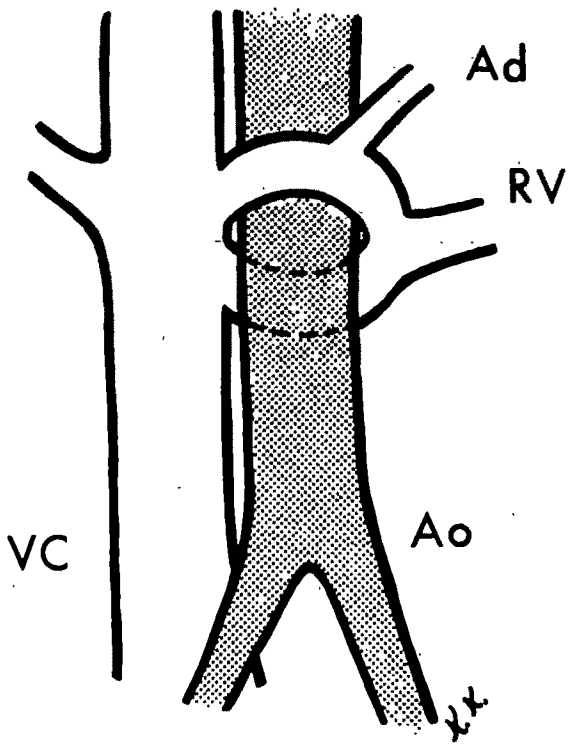

FIG. 1. Diagram of circumaortic renal collar. The renal vein has a component which is posterior to the aorta as well as the anterior component. Note that the posterior vein inserts in the vena cava at a lower level than the anterior renal vein. VC = vena cava. Ao = aorta. RV = renal vein. Ad = adrenal vein.

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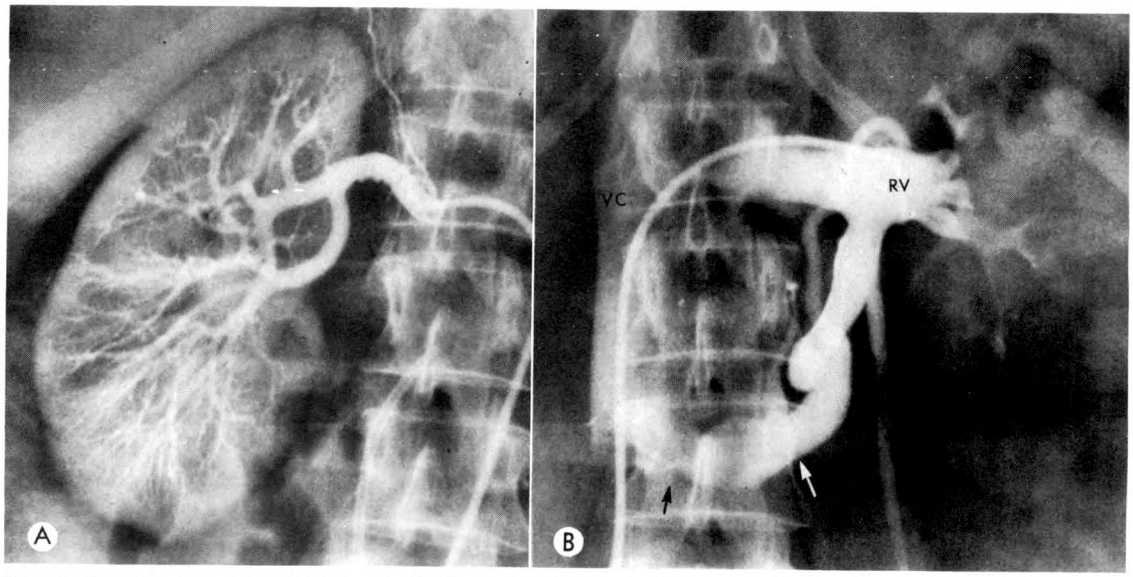

FIG. 2. Case 1. A 45 year old female with hypertension. (*A*) Selective right renal arteriogram. There is irregular narrowing of the midportion of the main renal artery consistent with medial fibroplasia. (*B*) Selective left renal venogram. The catheter is in the anterior renal vein. The postaortic component of this circumaortic renal collar passes inferiorly before it courses medially behind the aorta to insert in the vena cava (arrows).

says demonstrated elevated levels on the right.

Comment. This patient was a candidate for surgical repair or bypass grafting of the abnormal right renal artery. This type of surgery often requires cross-clamping of the aorta. It is extremely important for the surgeon to know that there is a posterior or retroaortic component to the left renal venous drainage to prevent tearing this hidden vein during surgery.

CASE II. A 50 year old woman was admitted for the evaluation of obesity and hirsutism. Laboratory studies of steroid excretion were consistent with Cushing's syndrome. Excretory urography revealed a left suprarenal mass. Adrenal venography demonstrated a normal right adrenal gland. During the course of attempted catheterization of the left adrenal vein, difficulty was encountered in advancing the catheter in the renal vein. Extravasation of contrast material occurred during a test injection. For this reason, the catheter was withdrawn to the orifice of the left renal vein. A test injection at this site demonstrated the characteristic pattern of a circumaortic renal collar (Fig. 3). The patient returned to her room and maintained normal vital signs and a stable hematocrit. Subsequent selective arteriography demonstrated the left suprarenal mass to be a pancreatic neoplasm associated with ectopic ACTH production. This was confirmed at surgery.

Comment. The anterior portion of the circumaortic renal collar in this patient was approximately 1/2 the diameter of a normal renal vein. Thus, the distal limb of the preformed left adrenal catheter was too long for safe manipulation in the renal vein. Since the left adrenal vein joins the ventral renal vein in such cases, adrenal venography must be performed with special care. The surgeon took care during the resection of the pancreatic adenoma and exploration of the left adrenal not to interrupt the left venous collar.

CASE III. A 58 year old man was admitted for evaluation of hypertension. Physical examination was normal with the exception of a blood pressure of 175/100 mm. Hg. Excretory urography was normal. Aortography revealed some atherosclerotic changes in the abdominal aorta. Although no renal artery stenosis was observed, venous blood samples were obtained for renin assay. During fluoroscopic test injections, an abnormal venous flow pattern was noted on the left. The characteristic appearance of a cir-

cumaortic renal collar was documented by anteroposterior and lateral left renal venography (Fig. 4).

Comment. This patient was not a surgical candidate during this admission. He was, however, informed of the nature of the left renal vein anomaly as a precaution should he require future abdominal or renal surgery.

DISCUSSION

The embryology of the vena cava and the renal veins has been well studied.[1,3,4,6] By the eighth week of life the posterior cardinal system, which remains only as the iliac bifurcation in the adult, has been replaced by four longitudinal channels. These are the paired supracardinal veins that are posterior to the aorta and the paired subcardinal veins that are anterior to the aorta. There are rich anastomotic communications between the supracardinal-subcardinal, intersupracardinal, and intersubcardinal channels which form a plexus or collar of veins. Usually, the ventral portion of the circumaortic plexus persists as the normal left renal vein. If the dorsal portion of this plexus persists, then the left renal vein is posterior to the aorta. If both the dorsal and ventral portions persist, there will be a circumaortic venous collar in the adult.

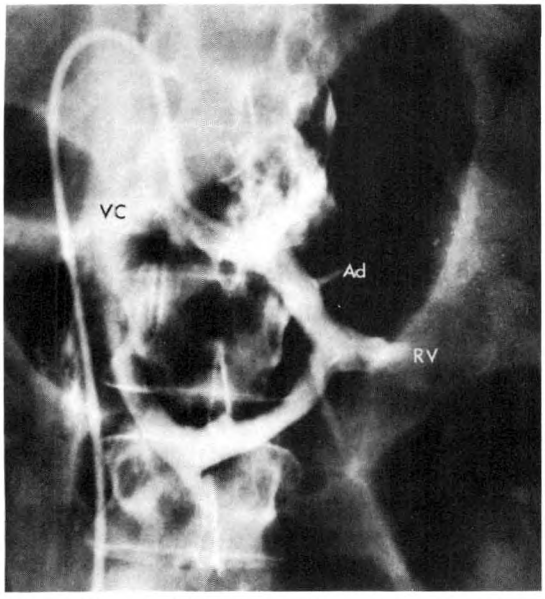

Fig. 3. Case II. Selective renal venogram. A 50 year old female with obesity, hirsutism and a left abdominal mass. Extravasation occurred during an attempted adrenal venogram. The renal venogram demonstrates that the veins of the circumaortic renal collar are each approximately half the size of a single normal main renal vein.

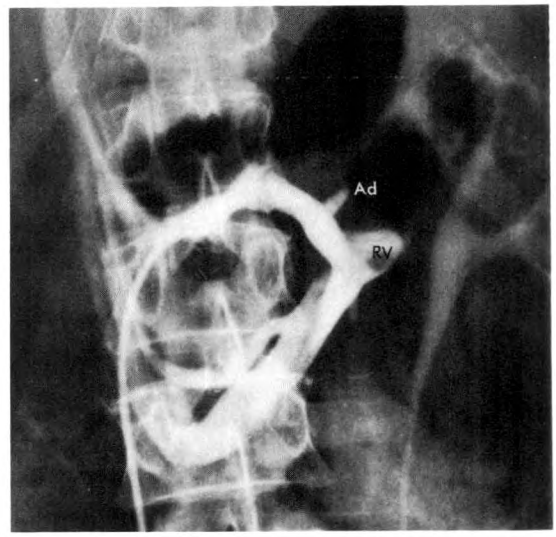

Fig. 4. Case III. A 58 year old patient admitted with hypertension. This selective renal venogram again demonstrates the characteristic pattern of a circumaortic renal collar. The posterior component has two branches in this patient and is larger than the anterior component.

The frequency of these venous anomalies in the adult varies from series to series. Davis and Lundberg[2] reported in 270 autopsies a retroaortic left renal vein in 1.8 per cent and a circumaortic renal collar in 1.5 per cent. Reis and Esenther[6] reported a 2.4 per cent incidence of venous collar. Recently, Brener *et al.*[1] reported 2 cases of circumaortic renal collar in a series of 31 major venous anomalies of the renal vein and vena cava. The more common anomaly in this series was a single retroaortic left renal vein which was present in 20 patients.

It is important for the surgeon to be aware of the presence of a retroaortic left renal vein during retroperitoneal surgery. The circumaortic renal collar is particularly dangerous since the surgeon may be lulled into a false sense of security when he identifies the ventral portion of this anom-

aly. He may then attempt to clamp the aorta or mobilize the kidney and tear the retroaortic component of the anomaly. Cases of severe hemorrhage leading to nephrectomy and death have been reported as a result of trauma to this venous ring.[1,2,7] Surgery for abdominal aneurysm poses a particular problem since the left renal vein is used as a landmark below which the aorta is clamped. Since the posterior portion of this venous ring passes inferior as well as posterior, it is at particular risk during aortic cross-clamping. The left kidney is usually preferred for transplant surgery since the left renal vein is longer. With a circumaortic venous ring this advantage is lost due to fixation of the vein at the aortic margin. It becomes obvious that the presence of this anomaly is of vital importance to the vascular surgeon and urologist.

The angiographer has an opportunity to recognize and document this anomaly during selective venous catheterization. Fluoroscopic recognition of a splitting of the stream of contrast material into the normal ventral left renal vein and the persistent dorsal component is not difficult since the dorsal vein courses inferiorly as well as medially, as it approaches the vena cava. This fluoroscopic observation should be documented with films as an aid to the surgeon. If surgery is not to be performed, then the nature of the anomaly should be in the patient's hospital record in case future surgery should be necessary.

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REFERENCES

1. Brener, B. J., Darling, R. C., Frederick, P. L., and Linton, R. R. Major venous anomalies complicating abdominal aortic surgery. *A.M.A. Arch. Surg.*, 1974, *108*, 159–165.
2. Davis, C. J., and Lundberg, G. D. Retroaortic left renal vein: relatively frequent anomaly. *Am. J. Clin. Path.*, 1968, *50*, 700–703.
3. Davis, R. A., Milloy, F. J., and Anson, B. J. Lumbar, renal and associated parietal and visceral veins based upon study of 100 specimens. *Surg., Gynec. & Obst.*, 1958, *100*, 1–22.
4. Edwards, E. A. Clinical anatomy of lesser variations of inferior vena cava: proposal for classifying anomalies of this vessel. *Angiology*, 1951, *2*, 85–99.
5. Pick, J. W., and Anson, B. J. Renal vascular pedicle: anatomical study of 430 body halves. *J. Urol.*, 1940, *44*, 411–434.
6. Reis, R. H., and Esenther, G. Variations in pattern of renal vessels and their relation to type of posterior vena cava in man. *Am. J. Anat.*, 1959, *104*, 295–318.
7. Thomas, T. V. Surgical implications of retroaortic left renal vein. *A.M.A. Arch. Surg.*, 1970, *100*, 738–740.

Vol. 125, No. 2

SUBMUCOSAL HEMORRHAGE OF THE RENAL COLLECTING SYSTEM*

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ABSTRACT:

Two cases of submucosal hemorrhage in the renal pelvis and ureter are presented. The roentgenographic appearance was identical to that of pyeloureteritis cystica but without evidence of urinary tract infection and, in 1 case, the submucosal impression resolved rapidly. When nodular, submucosal filling defects are demonstrated in the renal collecting system in the clinical setting of anticoagulant therapy or trauma, a diagnosis of submucosal hemorrhage should be considered.

THE differential diagnosis of roentgenographically seen multiple small, submucosal impressions on the renal collecting system includes: pyeloureteritis cystica; uroepithelial tumor; vascular impressions; and submucosal edema.[9] We encountered 2 patients with submucosal hemorrhage of the upper urinary tract that was urographically identical to pyeloureteritis cystica.

REPORT OF CASES

CASE 1. A 27 year old man had been on coumarin therapy for 2 years for pulmonary emboli. He was seen at our hospital because he had been having pain in his left flank for 1 week without fever, chills, frequency, or dysuria. He reported no prior trauma or history of urinary tract infection; an excretory urogram obtained 2 years before showed normal findings. On physical examination he was afebrile and had left flank tenderness. Urinalysis revealed 40 to 50 red blood cells, without clots, and 1 to 2 white blood cells per high power field. Gram stain and urine culture were negative. Prothrombin time was 32.4 seconds with a control of 11.8 seconds. An excretory urogram was performed (Fig. 1, *A* and *B*) which showed multiple, radiolucent, nodular impressions on the left infundibula, pelvis, and proximal ureter. The patient's anticoagulation therapy was discontinued. His clotting studies returned to normal, his flank pain resolved, and repeat urinalysis was normal. An excretory urogram 2 weeks later showed complete resolution of the pelvic impressions and only 1 remaining small defect in the proximal ureter (Fig. 1*C*).

CASE II. A 22 year old man was admitted to the hospital emergency room after being stabbed in the left upper quadrant of the abdomen with a 3 inch knife. His medical history was not contributive. Gross hematuria was observed. An emergency exploratory laparotomy disclosed 3 perforations of the distal jejunum, a laceration in the posterior peritoneum overlying the lower pole of the left kidney, and a large retroperitoneal hematoma, which was not drained. The patient's hematuria progressively decreased. Urine cultures and serial urinalyses showed no evidence of infection. On the tenth hospital day, an excretory urogram was obtained (Fig. 2); it showed submucosal impressions on the lower pole collecting system of the left kidney underlying the stab site. The patient was discharged on the fifteenth hospital day. We were unable to obtain a follow-up urogram.

DISCUSSION

Previous authors[3,6,7] have categorized the urographic manifestations of upper urinary tract hemorrhage by location. Hence, hematomas have been designated retroperitoneal, perirenal, subcapsular, intraparenchymal, and intraluminal. Smith *et al.*[8] described 4 patients on anticoagulant therapy who had urographic findings of submucosal hemorrhage in the upper uri-

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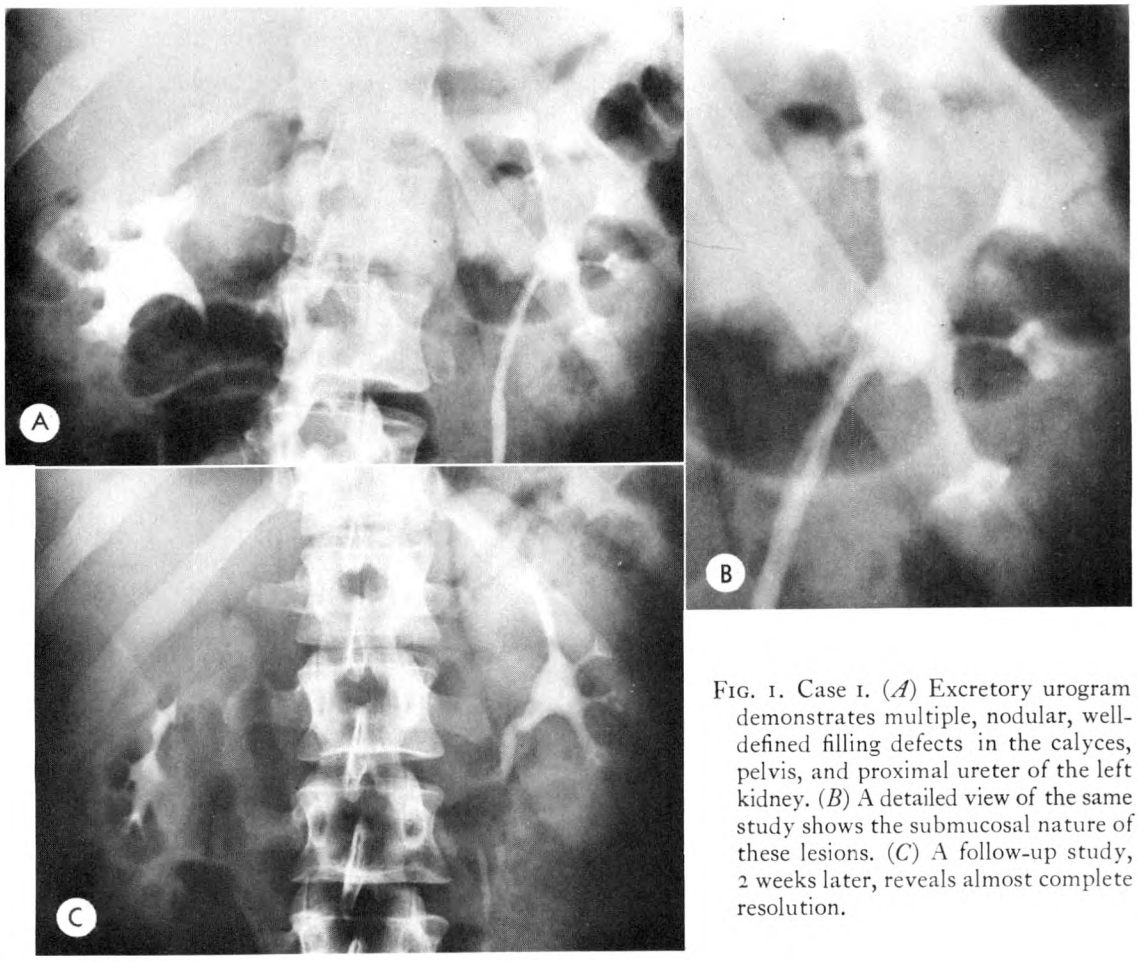

FIG. 1. Case 1. (*A*) Excretory urogram demonstrates multiple, nodular, well-defined filling defects in the calyces, pelvis, and proximal ureter of the left kidney. (*B*) A detailed view of the same study shows the submucosal nature of these lesions. (*C*) A follow-up study, 2 weeks later, reveals almost complete resolution.

nary tract. Theirs was the only description of this entity that we have found in the literature.

The urographic findings in our patients simulated the features of pyeloureteritis cystica. The defects in pyeloureteritis cystica consist of submucosal cysts formed as a result of chronic inflammation from urinary tract infection.[4] Neither of our patients had a history of urinary tract infection, any clinical or laboratory evidence of urinary tract infection, or urographic evidence of pyelonephritis. In fact, 1 patient showed normal urographic findings 2 years earlier and submucosal defects (Fig. 1) which resolved without therapy. Some patients with pyeloureteritis cystica have had hematuria and a hemorrhagic diathesis.[9] Perhaps submucosal blood was present to contribute to the urographic observations in those patients.

The clinical course of our patients also excludes the possibility of uroepithelial neoplasm. Although zones of edema could produce the same urographic picture, in both our patients the edema would be a response to the presence of blood or to its breakdown products.

Bleeding in the upper urinary tract is a common complication of anticoagulant therapy.[1,2] However, there has been only 1 report[8] of submucosal hemorrhage in the upper urinary tract. The apparent rarity of this observation is particularly surprising in view of the well known precedent of submucosal gastrointestinal bleeding in patients on anticoagulation therapy.[5] Whether these few patients reflect the true frequency

of this urologic complication is not known.

Submucosal accumulation of blood in the upper urinary tract is not limited to patients taking anticoagulants. Case II indicates that this finding can also result from a penetrating wound.

This paper supports the recently suggested correlation between urographically seen submucosal impressions and submucosal or intramural hemorrhage of the upper urinary tract.[8] This correlation has not yet been confirmed surgically or histologically.

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REFERENCES

1. Douglas, A. S. Thrombotic diseases. *Seminars Hematol.*, 1971, *8*, 95–139.
2. Goodman, L. S., and Gilman, A. Editors. The Pharmacological Basis of Therapeutics. Macmillian, New York, 1970, p. 1455.
3. Kaufman, S. A., and McLellan, P. Urinary tract complications of anticoagulation therapy: "pseudotumour" of kidney. *Brit. J. Radiol.*, 1968, *41*, 180–185.
4. Köhler, R. Pyelo-ureteritis cystica. *Acta radiol.* (Diag.), 1966, *4*, 123–128.
5. Messinger, N. H., and Beneventano, T. C. Submucosal hemorrhage of small intestine: complication of anticoagulant therapy. *Am. J. Gastroenterol.*, 1972, *57*, 74–78.
6. Navani, S., Bosniac, M. A., Shapiro, J. H., and Kaufman, S. Varied radiographic manifestations of urinary tract bleeding. *J. Urol.*, 1968, *100*, 339–343.
7. Pollack, H. M., and Popky, G. L. Roentgenographic manifestations of spontaneous renal hemorrhage. *Radiology*, 1974, *110*, 1–6.

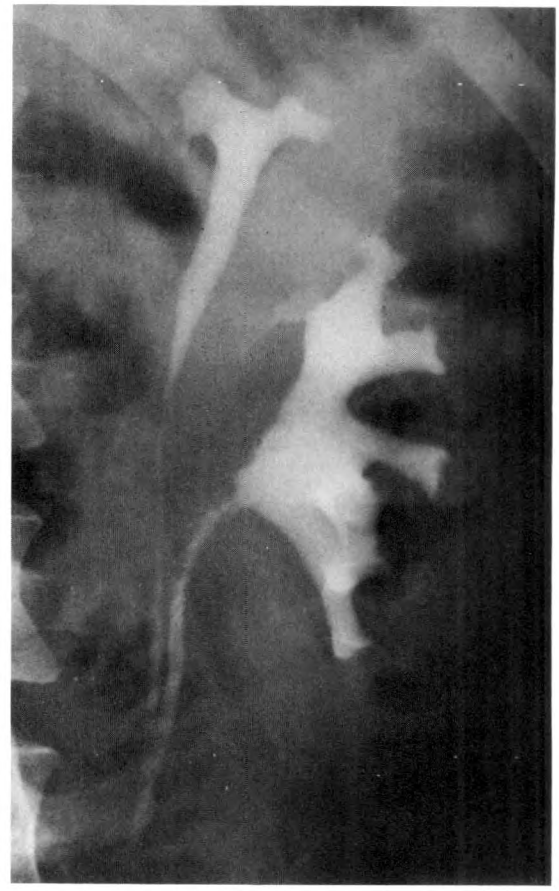

Fig. 2. Case II. A detailed view of the excretory urogram of the left kidney shows a partial duplication of the collecting system with submucosal impressions on the lower pelvis and proximal ureter.

8. Smith, W. L., Weinstein, A. S., and Wiot, J. F. Defects of renal collecting systems in patients receiving anticoagulants. *Radiology*, 1974, *113*, 649–651.
9. Wright, F. M. Mucosal edema of ureter and renal pelvis. *Radiology*, 1969, *93*, 1309–1312.

VESICOURETERAL REFLUX IN ADULTS*

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ABSTRACT:

Vesicoureteral reflux was detected in 200 adults seen in a 4 year period. In group 1 (no history of urinary tract infection and negative urine cultures), all patients had abnormalities on excretory urograms that were suggestive of reflux. In patients with a history of recurrent urinary tract infection (groups 2 and 3), the excretory urograms showed an abnormality in 79 per cent of those with negative cultures and in 83 per cent of those with positive cultures. Thus, reflux should be suspected in patients with a history of recurrent urinary tract infection even if infection cannot be documented with urine culture. Urographic evidence of renal parenchymal scarring, ureteral dilatation, or mucosal striations should also suggest the possibility of vesicoureteral reflux even when the patient may have no urinary tract symptoms. Voiding cystourethrography should be performed in all adults with these findings to exclude the possibility of vesicoureteral reflux.

DESPITE numerous articles in the literature concerning vesicoureteral reflux in children, it was not until recently that the importance of vesicoureteral reflux in adults was emphasized.[4,9,18] This emphasis can be attributed to an increased awareness of the condition in adults as well as to an increased utilization of voiding cystourethrography in evaluating adults with recurrent urinary tract infections.[4,5,9,11,14–16,18–21]

MATERIAL AND METHOD

From 1969 through 1973, vesicoureteral reflux was documented in 200 adults ranging in age from 16 to 70 years. Patients were included in the study if the initial diagnosis of reflux was established at age 16 years or older. The diagnosis of vesicoureteral reflux was made by utilizing standard fluoroscopic and spot film techniques during voiding cystourethrography. Voiding cystourethrography utilizing fluoroscopy has been shown to be the technique of choice in establishing the diagnosis of reflux.[25]

Clinical and laboratory data, voiding cystourethrograms, and excretory urograms were reviewed, and the 200 patients were divided into 4 clinical groups (Table 1): (1) 18 patients with no symptoms of urinary tract infection and in whom urine

TABLE I

VESICOURETERAL REFLUX IN 200 ADULTS: CLINICAL GROUPS

	No. of Patients
Group 1	18
No history of urinary tract infection	
Negative urine culture	
Group 2	112
History of recurrent urinary tract infection	
Negative urine culture	
Group 3	60
History of recurrent urinary tract infection	
Positive urine culture	
Group 4	10
Iatrogenic reflux	
Previous genitourinary or pelvic surgery	
Total	200

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.
From the Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

cultures were repeatedly negative; (2) 112 patients with recurrent urinary tract infection but in whom urine cultures were repeatedly negative; (3) 60 patients with recurrent urinary tract infection and positive urine cultures (cultures were considered positive if bacterial counts were 100,000 colonies/ml. or greater; *Escherichia coli*, the most common organism, was detected on urine culture in 60 per cent of patients in group 3); and (4) 10 patients with iatrogenic reflux due to previous pelvic or genitourinary surgery.

OBSERVATIONS AND DISCUSSION

CLINICAL FINDINGS

There were 157 females and 43 males, for a female-to-male ratio of 3.7:1. The majority of patients (157) was between 16 and 45 years old (Table II) and in this group, 75 per cent were female. In the 60 to 70 year old range (20 patients), the female-to-male ratio was equal. The change in sex ratio in this group is attributed to the larger number of males being evaluated because of bladder outlet obstruction secondary to prostatic disease. The association between reflux and prostatic hyperplasia has been described by Bumpus[8] who noted vesicoureteral reflux in 4.7 per cent of men with prostatic hyperplasia. Of the 10 men between 60 and 70 years old, 5 had benign prostatic hyperplasia, bladder outlet obstruction, and vesicoureteral reflux. In group 1, the sex ratio was reversed, with males outnumbering females 10:8. The patients in this group are similar to a group of patients with sterile reflux described by Hutch and Smith.[18]

TABLE II

AGE (AT DIAGNOSIS) DISTRIBUTION OF 200 ADULTS WITH VESICOURETERAL REFLUX

Age (yr.)	No. of Patients
16–20	14
21–25	33
26–30	30
31–35	31
36–40	25
41–45	24
46–50	15
51–55	4
56–60	4
61–65	10
66–70	10

A history of onset of urinary tract infection during childhood was noted in 13 per cent (26 of 200) of patients, and all of these patients had renal parenchymal scarring detected on excretory urograms. In 87 per cent of the patients there was no history of urinary tract infection until adulthood, and renal parenchymal scarring was noted in 49 per cent.

It is not possible to determine when reflux began in these patients or whether continuous or intermittent reflux was present for variable lengths of time. However, the association of renal parenchymal scarring in patients with a history of urinary tract symptoms beginning in childhood suggests that significant vesicoureteral re-

TABLE III

PRIMARY INDICATIONS FOR EXCRETORY UROGRAPHY IN ADULTS WITH VESICOURETERAL REFLUX

Indication	Group 1	Group 2	Group 3	Group 4	Total
Symptoms of urinary tract infection	0	95	46	10	151
Hypertension	12	4	0	0	16
Calculi	0	7	6	0	13
Difficulty in voiding	0	4	4	0	8
Neurogenic bladder	0	0	4	0	4
Abdominal pain	4	0	0	0	4
Other complaints	2	2	0	0	4
Total patients	18	112	60	10	200

TABLE IV

EXCRETORY UROGRAPHIC FINDINGS IN 261 URETERS IN ADULTS WITH REFLUX

Finding	Per Cent
Renal parenchymal scarring	62
Localized	40
Generalized	60
Ureteral dilatation	20
Mucosal striations	10
Renal calculi	9
Complete duplication	6

TABLE V

EXCRETORY UROGRAPHIC FINDINGS IN ADULTS WITH VESICOURETERAL REFLUX, BY GROUP

Finding	Per Cent		
	Group 1	Group 2	Group 3
Normal	0	21	17
Abnormal	100	79	83
Parenchymal scarring	65	51	65
Ureteral dilatation	27	13	33
Mucosal striations	11	7	18
Calculi	11	5	23
Duplication	16	7	7

flux was probably present for several years before the reflux was demonstrated roentgenographically. In patients with urinary tract symptoms beginning in adulthood, the association of renal parenchymal scarring in only 49 per cent suggests that reflux was present for a shorter period.

In 32 of the 157 women, the initial diagnosis of reflux was established after a pregnancy. All of these patients were in group 2 or 3 and had had symptoms of pyelonephritis during their pregnancy. According to Hutch and associates,[12] dilatation of the collecting system and ureters to the level of the pelvic brim, especially on the right, oc-

FIG. 1. Tomogram of left kidney, showing parenchymal scar on lateral border with blunting of underlying calyx.

FIG. 2. Tomogram of left kidney, showing marked parenchymal atrophy involving upper pole and lateral scarring. Calyceal system is poorly visualized and there is compensatory hypertrophy of the lower pole.

curs during almost all pregnancies and, in a few (2 per cent), is associated with infection and pyelonephritis. The exact cause of reflux during pregnancy is uncertain, but Hutch *et al.*[12] and Heidrick *et al.*[10] stated that these patients may have a short intravesical ureter and may have had intermittent reflux during childhood which becomes more severe during pregnancy. In our series, 25 per cent of patients had had previous symptoms of urinary infections, but in the remaining 75 per cent urinary tract infection and reflux were initially diagnosed after the first pregnancy.

The primary indications for initial referral for excretory urography in the group of 200 patients are presented in Table III. By definition, none of the patients in group 1 was referred for urinary tract infection. The excretory urogram was abnormal and suggestive of reflux, and the radiologist suggested that voiding cystourethrography be performed. As expected, the majority of patients (82 per cent) in groups 2 and 3 was referred for excretory urography because of symptoms of urinary tract infection. Thirty per cent (52 of 172) of the patients in groups 2 and 3 were referred directly for voiding cystourethrography because of urinary tract symptoms; in the remaining 70 per cent, voiding cystourethrography was ultimately performed because of abnormalities seen on excretory urograms.

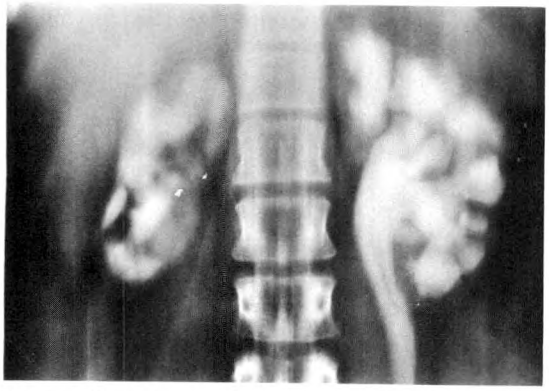

Fig. 3. Tomogram of kidneys, showing diffuse bilateral parenchymal loss (with dilatation of collecting systems and renal asymmetry).

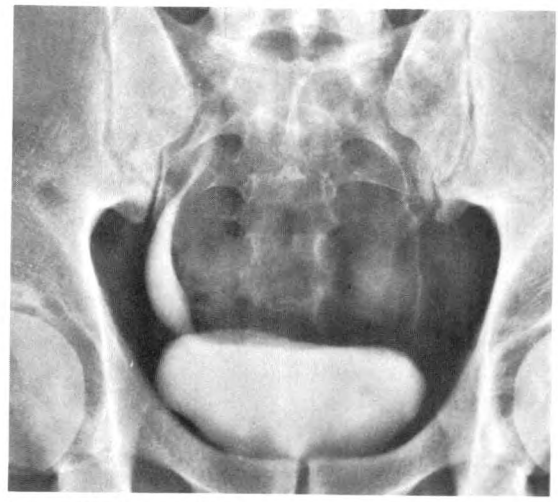

Fig. 4. Bladder and lower ureters, showing dilatation of lower right ureter.

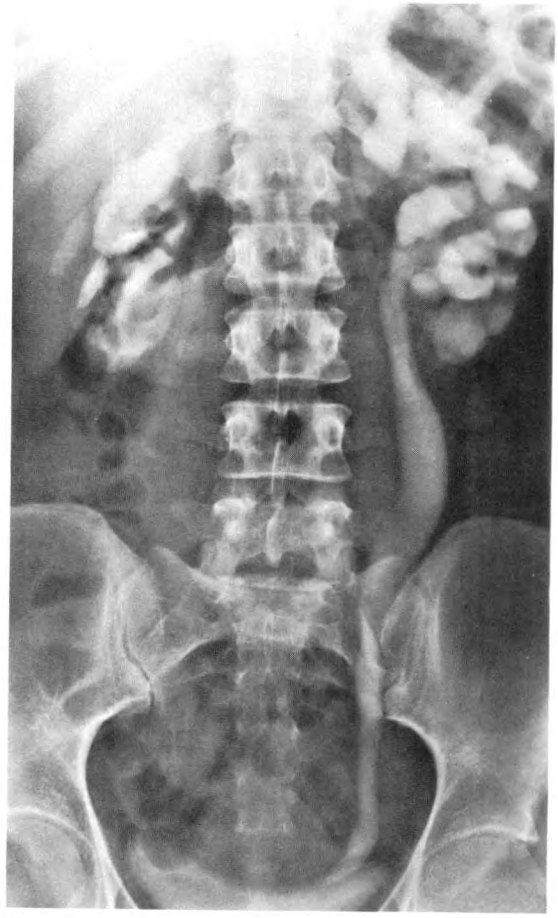

Fig. 5. Dilatation of entire left ureter and collecting system, with diffuse parenchymal atrophy in involved kidney.

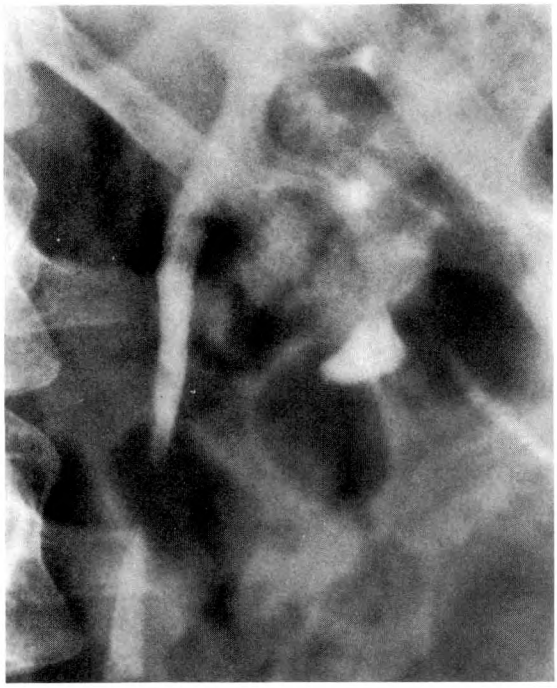

FIG. 6. Mucosal striations in the upper ureter and pelvis, more marked in the renal pelvis.

All 10 patients in group 4 (iatrogenic reflux due to previous surgery) were referred for excretory urography and voiding cystourethrography because of symptoms of urinary tract infection. Reflux was detected in 4 patients after ureterocele repair and in 5 patients after radical pelvic surgery for carcinoma of the cervix. In 1 patient, reflux developed after surgical removal of a bladder papilloma near the ureterovesical junction.

TABLE VI

ASSOCIATION OF RENAL PARENCHYMAL SCARRING WITH VESICOURETERAL REFLUX

Reflux	No. of Ureters	Scarring	
		No.	Per Cent
No dilatation			
Partial	68	31	46
Complete	140	75	54
Dilatation			
Partial	8	6	75
Complete	45	35	78

ROENTGENOGRAPHIC FINDINGS

Reflux was detected in 261 ureters. It was bilateral in 41 patients. In patients with unilateral reflux there was no significant difference between the incidences of reflux in the right and left ureters.

Renal Parenchymal Scarring. The most common renal abnormality associated with reflux was renal parenchymal scarring and blunting of the underlying calyces; this was detected in 62 per cent of the kidneys on excretory urograms (Table IV). Parenchymal scarring was more frequent in groups 1 and 3 than in group 2 (Table V). Parenchymal scarring was localized, involving only a small area of parenchyma, and was associated with blunting of a single calyx in 40 per cent of refluxing units (Fig. 1). In the

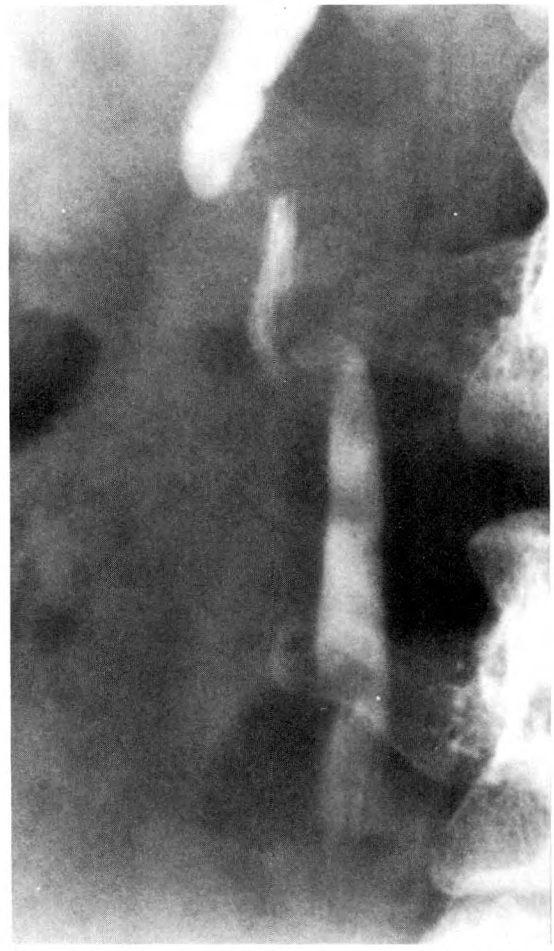

FIG. 7. Prominent mucosal striations in upper ureter and, to a lesser degree, in the dilated midureter.

remaining cases, parenchymal scarring was more extensive (Fig. 2; and 3) with involvement of an entire pole of the kidney, blunting of multiple calyces, or diffuse irregular scarring of the entire kidney with distortion of the entire calyceal system.

Renal parenchymal scarring in patients with vesicoureteral reflux has been referred to in numerous articles and the association is well documented.[3,4,6,7,9,14,15,17–22,24] In the study by Duffy,[9] reflux was detected in 75 per cent of patients with roentgenographic evidence of pyelonephritis and in 50 per cent of patients with a history of recurrent urinary tract infection. Our experience also suggests that adults with renal parenchymal scarring or a history of recurrent urinary tract infection should be evaluated by voiding cystourethrography.

Ureteral dilatation (Fig. 4; and 5) was noted with complete reflux in 45 ureters and with partial reflux in 8 (Table VI). Renal parenchymal scarring was present in 77 per cent of the kidneys when there was associated reflux with dilatation and was not dependent on whether the reflux was partial or complete. The correlation between reflux (partial or complete) and renal parenchymal scarring was more significant when there was associated dilatation of the ureter.

Mucosal Striations. Mucosal striations, detected in 26 (10 per cent) of the refluxing ureters, involved the upper ureter or renal pelvis in 20 cases and lower ureter in 6. Reflux was complete in all instances (Fig. 6; and 7). Mucosal striations have been previously described in 33 to 67 per cent of patients with reflux.[23]

Fig. 8. Autopsy specimen of kidneys, ureters, and bladder after removal of some of the contrast medium. Mucosal striations are noted in both upper ureters, more evident on the right.

In an attempt to clarify the etiology of mucosal striations and to determine the association of striations with dilatation and infection, we reviewed the clinical data and roentgenograms of 66 patients in

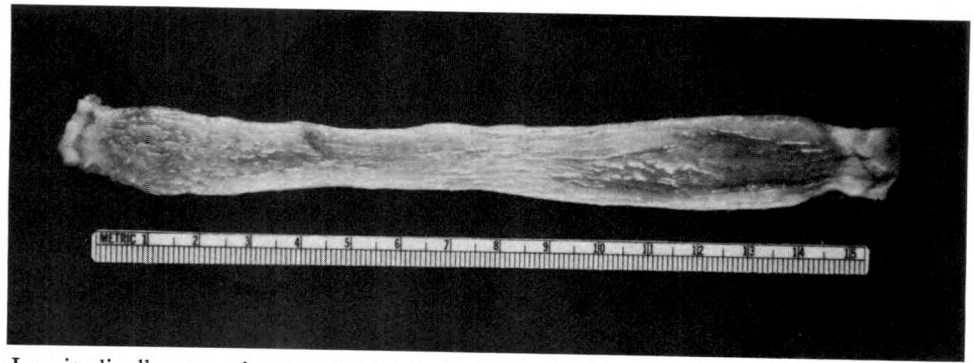

Fig. 9. Longitudinally opened ureter removed surgically from patient with chronic pyelonephritis and reflux. There is marked thickening of the mucosa with longitudinal folds. Striations were present on the excretory urogram.

FIG. 10. Excretory urogram, showing complete duplication of right collecting system. Note focal parenchymal scar in lower pole. Reflux was present in lower pole segment on voiding cystourethrogram.

whom mucosal striations were noted incidentally on the excretory urogram. In 13 patients, mucosal striations were associated with dilatation of the collecting system; in none of these patients was there a history of urinary tract infection, and all urine cultures were negative. In 4 patients, mucosal striations were associated with documented urinary tract infections but there was no associated dilatation of the collecting system. In 4 patients, mucosal striations were present but were not associated with dilatation or urinary tract infection. In 45 patients (68 per cent), mucosal striations were associated with dilatation of the collecting system and documented urinary tract infections. Within this group there were 30 patients who had had voiding cystourethrograms performed because of a history of urinary tract infection, and in only 8 of these patients could reflux be demonstrated.

In normal ureters obtained at autopsy, when the ureter was distended with contrast medium, no mucosal striations were identified. However, when the contrast medium was removed in small increments, striations were easily identified in the incompletely filled ureter and collecting system (Fig. 8).

From the observations in these 66 patients and in the autopsy study, incomplete distention of the dilated pelvis or ureter is the most important factor underlying the demonstration of mucosal striations on excretory urograms, regardless of the cause of the dilatation. Because 41 of the 66 patients had documented urinary tract infections, the question of the presence of urinary tract infection should be pursued clinically whenever mucosal stria-

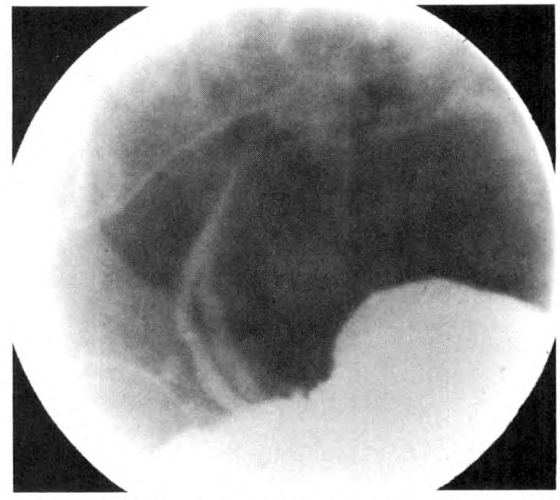

FIG. 11. Voiding cystourethrogram, showing partial reflux into both segments of duplicated right ureter.

tions are seen on excretory urograms (Fig. 9).

Other Roentgenographic Findings. The only other findings noted with any frequency were renal calculi in 22 patients and complete duplication in 15 patients. An increased incidence of renal calculi has been described in patients with reflux,[2] and, as would be expected, calculi were more frequent in patients with documented infections (group 3) (Table v). The association between complete duplication and reflux is well established,[1] and in our series the ureteral segment supplying the lower pole of the kidney was found to reflux in all 15 patients (Fig. 10). Reflux was detected in both segments in 1 case (Fig. 11).

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REFERENCES

1. AMAR, A. D. Reflux in duplicated ureters. *Brit. J. Urol.*, 1968, *40*, 385–401.
2. AMAR, A. D., HUTCH, J. A., and KATZ, I. Coexistence of urinary calculi and vesicoureteral reflux. *J.A.M.A.*, 1968, *206*, 2312–2313.
3. AMAR, A. D., and SINGER, B. Vesicoureteral reflux: 10-year study of 280 patients. *J. Urol.*, 1973, *109*, 999–1001.
4. AMAR, A. D., SINGER, B., LEWIS, R., and NOCKS, B. Vesicoureteral reflux in adults: twelve-year study of 122 patients. *Urology*, 1974, *3*, 184–189.
5. AMBROSE, S. S. Reflux pyelonephritis in adults secondary to congenital lesions of ureteral orifice. *J. Urol.*, 1969, *102*, 302–304.
6. BAKER, R., MAXTED, W., MAYLATH, J., and SHUMAN, I. Relation of age, sex and infection to reflux: data indicating high spontaneous cure rate in pediatric patients. *J. Urol.*, 1966, *95*, 27–32.
7. BRANNAN, W., OCHSNER, M. G., ROSENCRANTZ, D. R., WHITEHEAD, C. M., JR., and GOODIER, E. H. Experiences with vesicoureteral reflux. *J. Urol.*, 1973, *109*, 46–48.
8. BUMPUS, H. C., JR. Urinary reflux. *J. Urol.*, 1924, *12*, 341–346.
9. DUFFY, P. Primary vesico-ureteric reflux in adults. *M. J. Aust.*, 1972, *2*, 1056–1060.
10. HEIDRICK, W. P., MATTINGLY, R. F., and AMBERG, J. R. Vesicoureteral reflux in pregnancy. *Obst. & Gynec.*, 1967, *29*, 571–578.
11. HODSON, C. J., and EDWARDS, D. Chronic pyelonephritis and vesico-ureteric reflux. *Clin. Radiol.*, 1960, *11*, 219–231.
12. HUTCH, J. A., AYRES, R. D., and NOLL, L. E. Vesicoureteral reflux as cause of pyelonephritis of pregnancy. *Am. J. Obst. & Gynec.*, 1963, *87*, 478–485.
13. HUTCH, J. A., and SMITH, D. R. Sterile reflux: report of 24 cases. *Urol. Int.*, 1969, *24*, 460–465.
14. KERN, H. B., and MALAMENT, M. Vesico-ureteral reflux and adult male. *Brit. J. Urol.*, 1969, *41*, 295–306.
15. KLOTZ, P. G. Ureteral reflux in adults. *Canad. M. A. J.*, 1969, *100*, 4–12.
16. LEADBETTER, G. W., JR., DUXBURY, J. H., and DREYFUSS, J. R. Absence of vesicoureteral reflux in normal adult males. *J. Urol.*, 1960, *84*, 69–70.
17. LENAGHAN, D. Natural history of reflux and long-term effects of reflux on kidney. *Brit. J. Urol.*, 1974, *46*, 115. (Abst.)
18. LIPSKY, H., and CHISHOLM, G. D. Primary vesico-ureteric reflux in adults. *Brit. J. Urol.*, 1971, *43*, 277–283.
19. MCANINCH, J. W., and CAMPBELL, P. M. Primary vesicoureteral reflux in adult patients. *Urology*, 1973, *2*, 393–395.
20. MCGOVERN, J. H., and MARSHALL, V. F. Reflux and pyelonephritis in 35 adults. *J. Urol.*, 1969, *101*, 668–672.
21. SERVADIO, C., and SHACHNER, A. Observations of vesicoureteral reflux and chronic pyelonephritis in adults. *J. Urol.*, 1970, *103*, 722–726.
22. SHOPFNER, C. E. Vesicoureteral reflux: five-year re-evaluation. *Radiology*, 1970, *95*, 637–648.
23. SILBER, I., and MCALISTER, W. H. Longitudinal folds as indirect sign of vesicoureteral reflux. *J. Urol.*, 1970, *103*, 89–91.
24. TANAGHO, E. A., and HUTCH, J. A. Primary reflux. *J. Urol.*, 1965, *93*, 158–164.
25. VLAHAKIS, E., HARTMAN, G. W., and KELALIS, P. P. Comparison of voiding cystourethrography and expression cystourethrography. *J. Urol.*, 1971, *106*, 414–415.

TOMOGRAPHY OF PELVIC-ABDOMINAL MASSES DURING INTRAVENOUS UROGRAPHY*

AN ADDED DIMENSION

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ABSTRACT:

Experience with a wide variety of pelvic-abdominal masses indicates that good quality tomography during high dose urography permits separation of solid from cystic lesions in almost all cases, even with minimal mass opacification. Evaluation of surface contour, absence or presence of a wall and its features, opacification, calcification, and uterine status provides the framework for characterization of the mass. Multiple masses can be identified and evaluated separately. While titration of the above criteria permits an accurate assessment independent of the clinical diagnosis, the latter information is important in separating a probable abscess from other cystic lesions.

Since urography continues to be a pivotal preoperative roentgenologic investigation (ureteral status, pelvic kidney exclusion, etc.), the addition of tomography provides an added dimension to its usefulness. With the exception of the premenopausal female in whom radiation exposure is a consideration, tomography appears to preclude the routine necessity for subsequent ultrasound study which has been used to separate cystic from solid lesions.

ROENTGENOGRAPHIC demonstration of pelvic-abdominal masses during urography has, until recently, been limited to secondary signs and inferences. Contrast enhancement of pelvic and abdominal masses has been shown to be a useful technique,[1,3–6] and recently infusion tomography of the female pelvis has been described.[3] Since 1973, we have studied 25 proved pelvic-abdominal masses by tomography during intravenous urography; and we concur with Love *et al.*[3] that the addition of tomography to the urographic study provides an added dimension since it permits a more accurate assessment of such a mass, in addition to the usual evaluation of the urinary tract.

MATERIAL AND METHOD

Twenty-five masses in 22 patients are presented in which tomography of the pelvic-abdominal mass during urography provided preoperative definition and characterization of the mass. Most of these patients had a preoperative clinical diagnosis of "pelvic, adnexal, or abdominal mass."

TABLE I

PELVIC-ABDOMINAL TOMOGRAPHIC UROGRAPHY

Technique
1. Preliminary anteroposterior film of pelvic-abdominal area
   Determine size pelvic mass:
   8 cm. or less: 6 cm. tomogram
   10 cm. or more: 12 cm. tomogram
2. Preliminary anteroposterior tomogram of pelvic-abdominal area
3. Intravenous urogram (bolus or infusion)
4. Early tomography of mass
   a. 1–4 min. (bolus)
      3–5 min. (infusion)
   b. small mass: 4–11 cm. levels
      larger mass: 8–17 cm. levels
5. Standard urography of urinary tract

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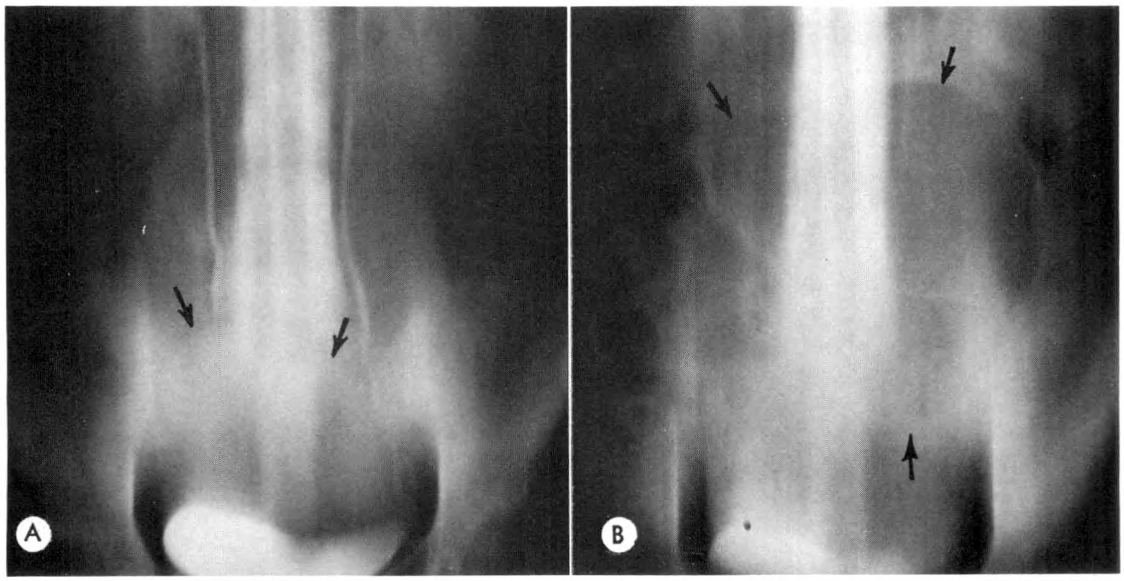

FIG. 1. (R.C., Case 11.) (*A*) A 14 cm. tomographic level showing enlarged posteriorly positioned opacified *leiomyoma* of uterus (arrows). (*B*) An 18 cm. tomographic level demonstrating a separate anteriorly positioned multiloculated thin-walled cystic ovarian mass (*dermoid*) (arrows).

No single method of delivery of contrast medium was adhered to; 17 patients received a 300 ml. infusion urogram, 1 received a 100 ml. bolus of contrast medium, and 4 received a 50 ml. bolus. While tomography was done at times ranging from 1 to 25 minutes, the present technique is described in Table 1. Both a preliminary plain film of the abdomen and a preliminary tomogram of the pelvic-abdominal area should be obtained to evaluate subsequent opacification of the mass. Following injection of contrast medium, early tomography is suggested to study the pelvis prior to significant bladder filling. The level of the tomogram generally depends on the size of the mass (Table 1). Standard urographic filming completes the examination.

These 22 patients with 25 masses (3 patients with 2 distinct masses) include 13 ovarian neoplasms, 9 uterine masses, 1 pelvic abscess, 1 ectopic pregnancy, and 1 mesenteric tumor (Table II). Of the 13 ovarian tumors, 6 were benign cystadenomas, 3 were malignant cystadenocarcinomas, 3 were cystic dermoids, and 1 was a malignant solid dysgerminoma. Of the 9 uterine masses, 7 were leiomyomas and 2 were dilated uteri (hydrometria). The 1 abscess was of tubo-ovarian origin, the ectopic pregnancy was ruptured and infected, and the abdominal mesenteric tumor was a fibrosarcoma.

ROENTGEN APPEARANCE

In our initial group of 22 patients, tomography of pelvic-abdominal masses has characterized and identified lesions with greater clarity and detail than urography alone. The following observations can be made: pelvic-abdominal tomographic urography in cystic ovarian masses (Fig. 1; 3; 5; and 7) showed a smooth opacified wall 1–40 mm. thick and a lucent center in 11 of 12 cases (92 per cent), and a separate normal uterus in those cases in which it was evaluated. The 1 solid malignant ovarian mass presented no wall or opacification, and a smooth surface.

In the 2 dilated uterine masses (hydrometria) both (2 of 2) showed the following: opacified wall of uniform thickness (1.5 cm.) with a smooth rounded surface and lucent center, without a separate uterine density (Fig. 2; and 3). Of the 7 uterine solid tumors (Fig. 1; 4; and 6), all (7 of 7)

TABLE II

TOMOGRAPHY OF PELVIC-ABDOMINAL MASSES DURING UROGRAPHY

Case	Age Sex	Clinical Diagnosis	Tomography Level; Dose Time	Tomographic Findings: Size Mass	Opacification: Central	Opacification: Wall	Features	Separate Uterus	Roentgen Diagnosis	Pathology
(1) M.W.	21 F	Pelvic-abdominal mass	15 cm. 300 ml. DIP 5 min.	20 cm.	−	−	Round smooth surface, no opacification	N.E.	Ovarian mass	Ovarian dysgerminoma (solid tumor)
(2) B.J.	43 F	Pelvic mass	10 cm. 50 ml. bolus 1 min.	8 cm.	−	+	1 mm. smooth wall and surface	+	Ovarian cyst	Ovarian cyst
(3) L.M.	34 F	Pelvic mass, PID	12 cm. 50 ml. bolus 15 min.	5 cm.	−	+	1 mm. smooth wall and surface	+	Ovarian cyst	Ovarian cyst
(4) J.R.	33 F	L adnexal mass	5 cm. 100 ml. bolus 12 min.	4 cm.	−	++	1 mm. smooth wall and surface	N.E.	Ovarian cyst	Ovarian cyst
(5) C.L.	67 F	Pelvic mass	17 cm. 300 ml. DIP 5 min.	13 cm.	−	+	1 mm. smooth wall and surface	+	Ovarian cyst	Ovarian cystadenoma (mucinous)
(6) V.P.	53 F	Pelvic mass	11 cm. 50 ml. bolus 25 min.	9 cm.	−	+	3–9 mm. smooth wall and surface	+	Ovarian cyst	Ovarian cystadenoma (mucinous)
(7) F.S.	50 F	Pelvic-abdominal mass	16 cm. 300 ml. DIP 5 min.	21 cm.	−	−	Round smooth surface	N.E.	Ovarian mass	Ovarian cystadenoma (serous)
(8) B.N.	74 F	Pelvic-abdominal mass	15 cm. 300 ml. DIP 5 min.	18 cm.	−	+	1 mm. smooth wall and surface	N.E.	Ovarian cyst	Ovarian cystadenocarcinoma
(9) E.K.	57 F	Pelvic-abdominal mass	17 cm. 300 ml. DIP 5 min.	26 cm.	−	+	3 mm. smooth wall and lobulated surface	+	Ovarian cyst	Ovarian cystadenocarcinoma
(10) M.G.	63 F	Ovarian tumor	15 cm. 300 ml. DIP 5 min.	16 cm.	−	++	Curvilinear Ca^{++} for 3 yr., 5-40 mm. wall	+	Ca^{++} ovarian cyst (dermoid)	1. L: ovarian dermoid cyst with squamous cell carcinoma 2. R: ovarian dermoid cyst with carcinoid
(11) R.C.	39 F	Pelvic-abdominal mass	14 and 18 cm. 300 ml. DIP 5 min.	19 cm.	−	++	Superior: multiloculated mass 2 mm. wall	−	1. Multilocular ovarian cyst	1. Ovarian dermoid cyst
				15 cm.	+	−	Inferior: uniform opacification and lobular surface	−	2. Leiomyoma uterus	2. Leiomyoma uterus
(12) J.M.	64 F	Ovarian cancer	15 cm. 300 ml. DIP 5 min.	23× 12 cm.	−	++	2 contiguous lesions, R: 13 cm. mass with 1.5 cm. smooth wall and surface	−	Multilocular ovarian cyst	1. R: hematometrium 2° to obstructing adenocarcinoma endometrium
					−	++	L: 10 cm. mass with 1 mm. smooth wall and surface			2. L: ovarian cystoadenocarcinoma
(13) M.M.	69 F	Recurrent tumor—carcinoma cervix	15 cm. 300 ml. DIP 5 min.	12 cm.	−	++	1.5 cm. smooth wall and surface	−	Hydrometrium	Pyometrium (sterile) 2° to fibrosis of cervical os
(14) N.H.	31 F	Pelvic mass, PID	10 cm. 50 ml. bolus 1 min.	9 cm.	−	+	3 mm. smooth wall and surface	N.E.	Ovarian cyst	Abscess, subacute
(15) D.D.	22 F	Pelvic mass, possible abscess or ectopic pregnancy	11 cm. 300 ml. DIP 8 min.	18 cm.	−	++	1–10 mm. multilocular smooth walled mass	−	Hemorrhagic ovarian cyst	Infected ruptured ectopic pregnancy

PID = Pelvic inflammatory disease.
DIP = Drip infusion pyelogram.
N.A. = Not applicable.
N.E. = Not evaluated.
R = Right side.
L = Left side.
2° = Secondary.

TABLE II (*continued*)

Case	Age Sex	Clinical Diagnosis	Tomographic Level; Dose Time	Tomographic Findings: Size Mass	Opacification: Central	Opacification: Wall	Features	Separate Uterus	Roentgen Diagnosis	Pathology
(16) C.D.	62 F	Ovarian cancer	17 cm. 300 ml. DIP 5 min.	22 cm.	+	−	Uniform opacification and slightly lobular surface	N.E.	Leiomyoma uterus	Leiomyoma uterus
(17) M.S.	46 F	Pelvic mass	10 cm. 300 ml. DIP 5 min.	7 cm.	+	−	Uniform opacification and lobular surface	−	Leiomyoma uterus	Leiomyoma uterus
(18) E.H.	41 F	Pelvic mass	17 cm. 300 ml. DIP 5 min.	25 cm.	+	−	Uniform opacification and lobular surface	−	Leiomyoma uterus	Leiomyoma uterus
(19) M.T.	48 F	Pelvic mass	17 cm. 300 ml. DIP 5 min.	23 cm.	++	−	Uniform opacification and lobular surface	−	Leiomyoma uterus	Leiomyoma uterus
(20) A.W.	82 F	Ovarian cancer	8 cm. 300 ml. DIP 5 min.	13 cm.	++	−	Uniform opacification, lobular surface, and fibroid calcification	−	Leiomyoma uterus	Leiomyoma uterus
(21) A.M.	56 F	Abdominal mass	15 cm. 300 ml. DIP 10 min.	30 cm.	++	−	Uniform opacification and slightly lobular surface	−	Leiomyoma uterus	Leiomyoma uterus
(22) S.H.	59 M	Abdominal pain	17 cm. 300 ml. DIP 5 min.	17× 12 cm.	++	−	Uniform opacification	N.A.	Solid mesenchymal tumor of jejunum	Fibrosarcoma of mesentery

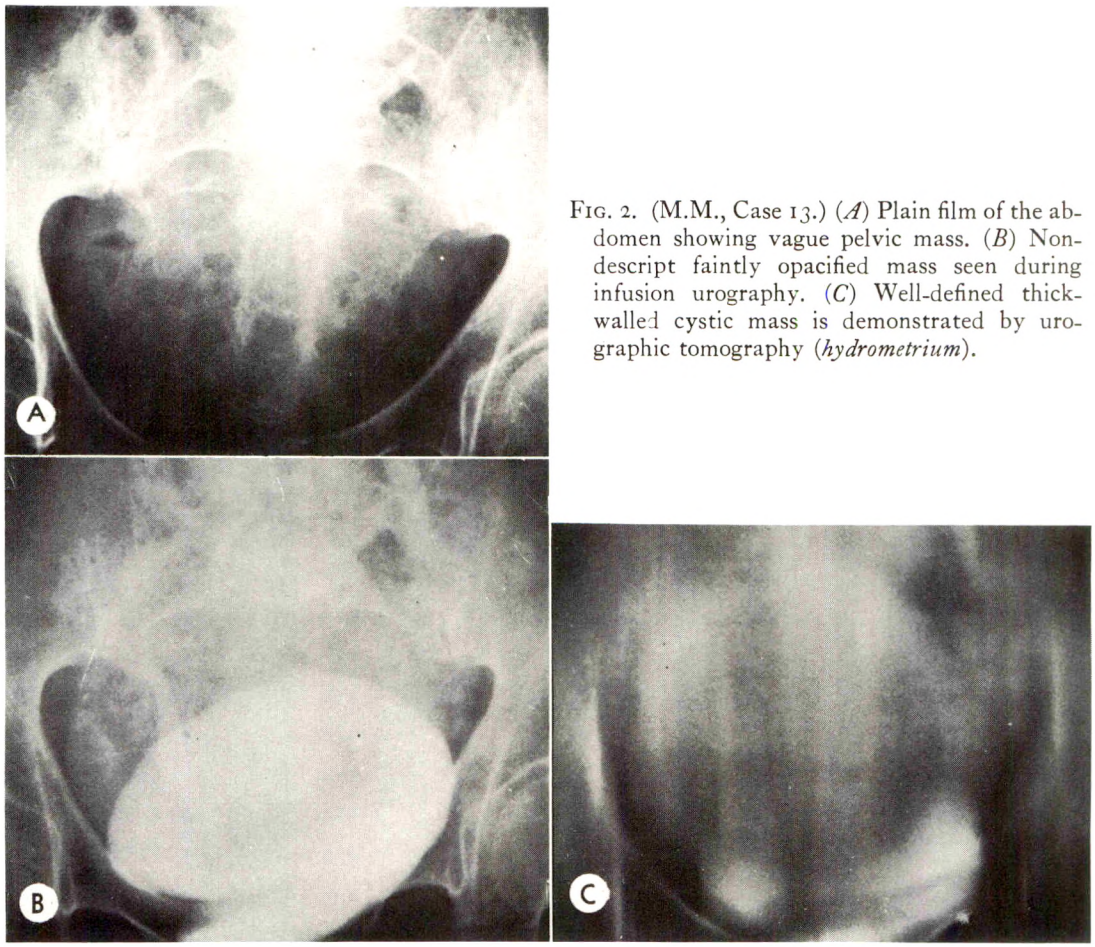

FIG. 2. (M.M., Case 13.) (*A*) Plain film of the abdomen showing vague pelvic mass. (*B*) Nondescript faintly opacified mass seen during infusion urography. (*C*) Well-defined thick-walled cystic mass is demonstrated by urographic tomography (*hydrometrium*).

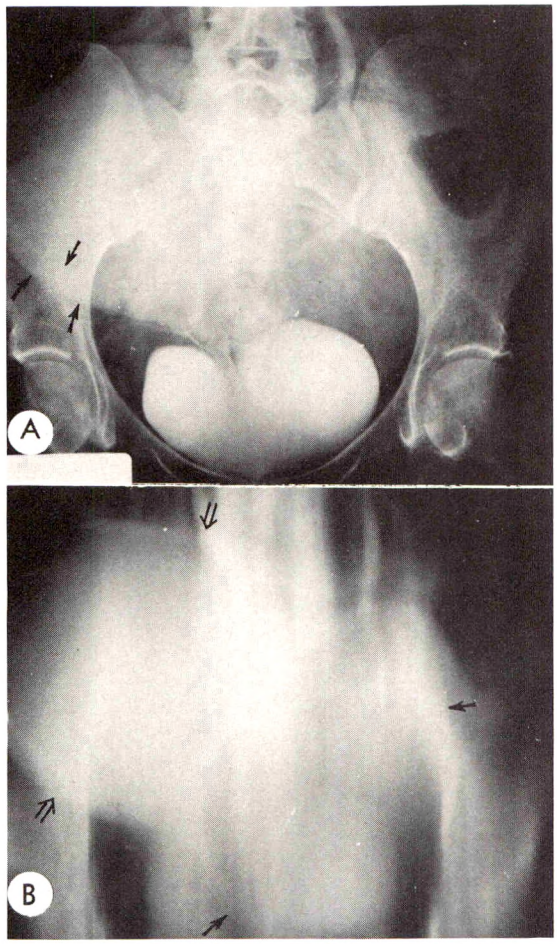

FIG. 3. (J.M., Case 12.) (*A*) Large oval pelvic mass with thick wall inferolateral on right (arrows) at infusion urography. (*B*) Urographic tomography demonstrates 2 adjacent cystic lesions: *right, hydrometrium* (double tailed arrows) and *left, ovarian cystadenocarcinoma* (solid arrows).

had uniform opacification, lobular surface, no wall and no separate uterine density.

The 1 tubo-parametrial mass (abscess, subacute) had an opacified wall 3 mm. thick, a smooth round surface, and lucent center (Fig. 8). The ruptured infected ectopic pregnancy had multiple smooth walls 1–10 mm. thick with a lobular surface. The 1 solid mesenteric neoplasm had a lobular surface, uniform opacification and no wall (Fig. 10).

Indications and contraindications to the examination are given in Table III and radiation exposure data are listed in Table IV.

DISCUSSION

Total body opacification is an accepted method for demonstrating abdominal and pelvic masses, first observed in children by O'Connor and Neuhauser in 1963.[5] Since then, in adults, other authors[1,2,6] have had variable success in outlining cystic lesions or opacifying solid structures during high dose urography.

Birnholz,[1] from this department, first reported the "adventitious hysterogram" as being useful in the differential diagnosis of pelvic masses, but noted "no . . . opacification was present in 12 cases of ovarian

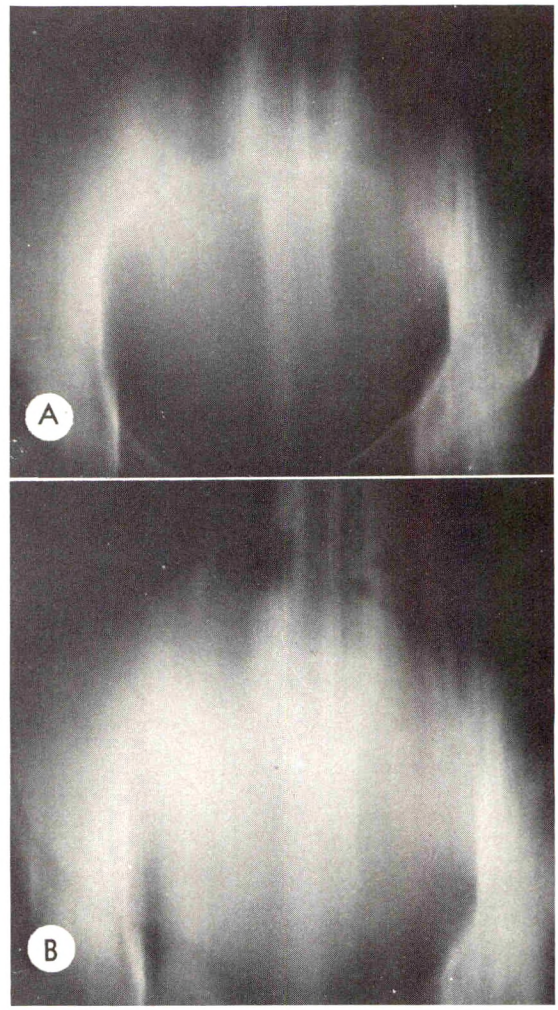

FIG. 4. (M.T., Case 19.) (*A*) Large, nondescript pelvic mass on preliminary tomogram. (*B*) Well-defined diffusely opacifying *leiomyoma* of uterus at urographic tomography.

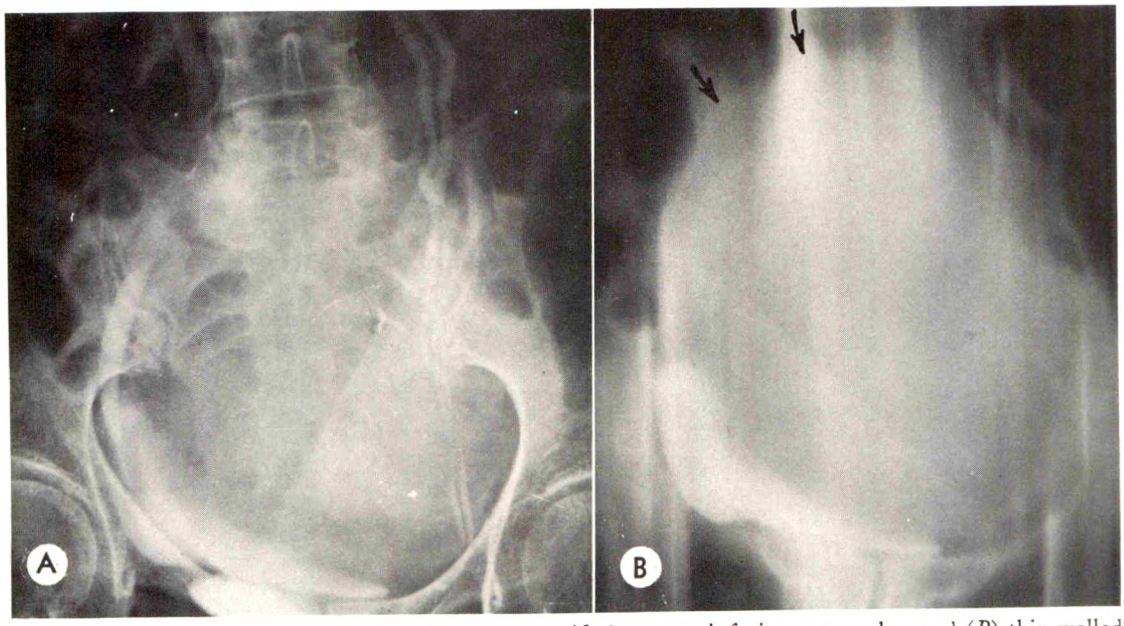

Fig. 5. (B.N., Case 8.) (*A*) Large, ill-defined, nonopacified mass at infusion urography, and (*B*) thin-walled cystic mass at urographic tomography (arrows) indicate probable ovarian etiology (*cystadenocarcinoma of ovary*).

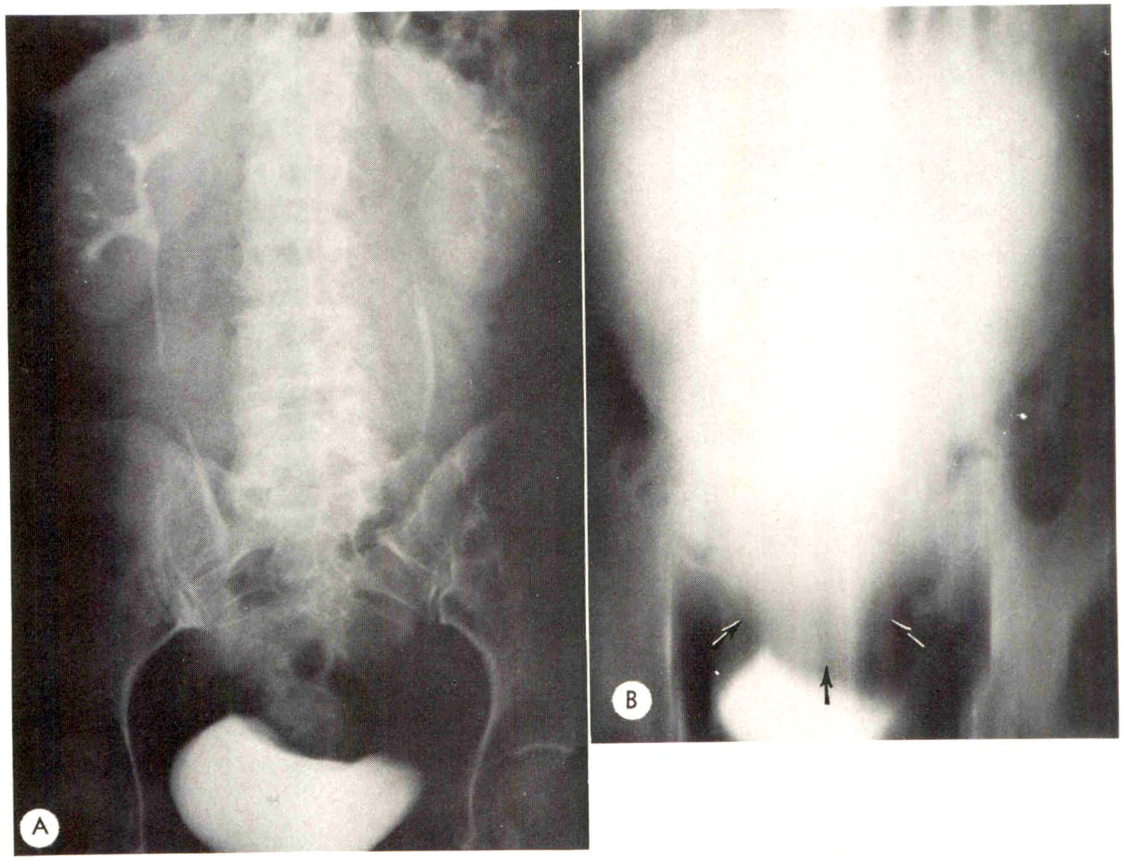

Fig. 6. (A.M., Case 21.) (*A*) Opacified mass on infusion urogram, and (*B*) opacified mass without wall on urographic tomogram arising from uterus (arrows) (*leiomyoma of uterus*).

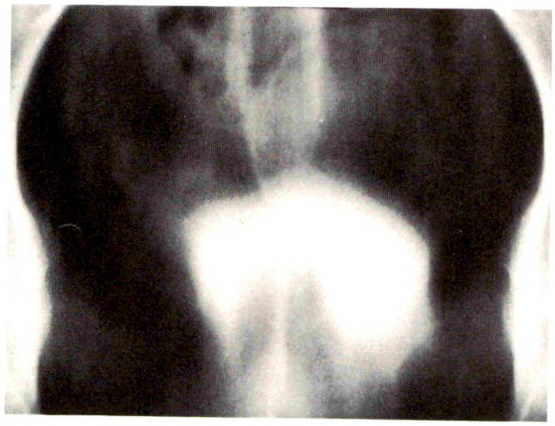

FIG. 7. (J.R., Case 4.) Small, thin-walled, cystic pelvic mass clearly demonstrated by urographic tomography (*corpus luteum ovarian cyst*) above the bladder.

cystic masses." Phillips *et al.*[6] have presented excellent results in demonstrating the "rim sign" in 80 per cent of cystic and mixed (solid and cystic) ovarian neoplasms, and homogeneous opacification in 80 per cent of uterine leiomyomas, but were unable to distinguish uterine from ovarian masses in the remaining 20 per cent. Additionally, none of their 50 tubo-ovarian abscesses was identified by non-tomographic urography.

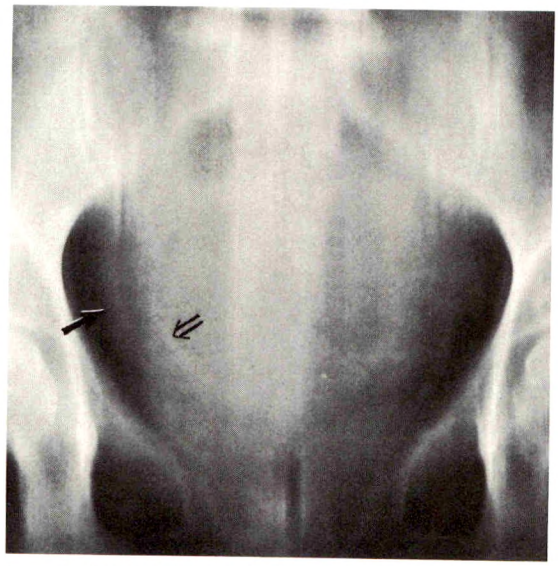

FIG. 8. (N.H., Case 14.) A 12 cm. thick-walled mass (solid arrow) with second thin opacified inner margin (double tailed arrow) at urographic tomography (*subacute abscess*).

Imray,[2] however, demonstrated only 43 per cent of cystic ovarian tumor walls, 85 per cent of uterine fibroids, and 33 per cent of pelvic abscesses. These figures are closer to our own non-tomographic experience. Recently, Love *et al.*[3] presented their initial small series with infusion tomography of the female pelvis in 9 patients.

Our concurrent experience in 25 pelvic-abdominal masses indicates that the use of tomography during high dose urography permits one to differentiate the cystic lesions (ovarian, uterine, abscess, etc.) from the solid, non- or poorly opacifying tumors

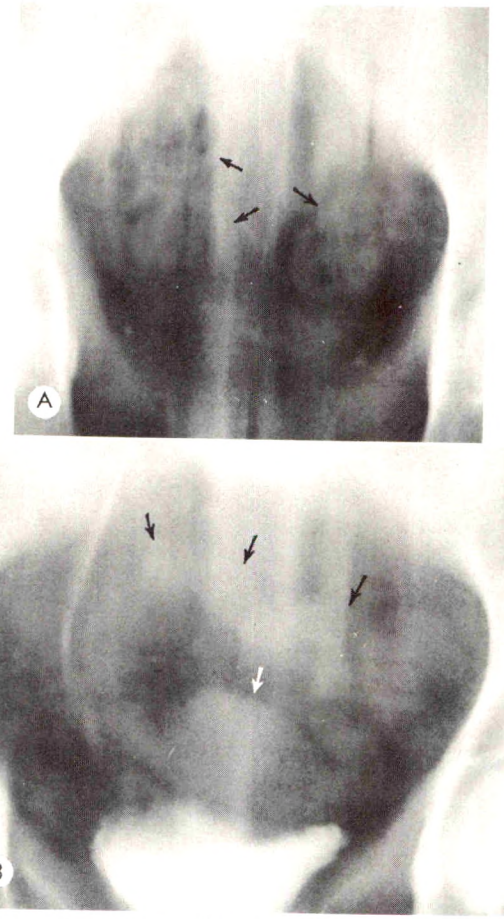

FIG. 9. (*A*) Urographic tomogram of deep pelvis showing *bowel* end-on (arrows) and *bladder* but absent uterus (post total hysterectomy and bilateral salpingo-oophorectomy). (*B*) Different patient at urographic tomography illustrating *normal uterus* (white arrow) and *sigmoid colon* (black arrows).

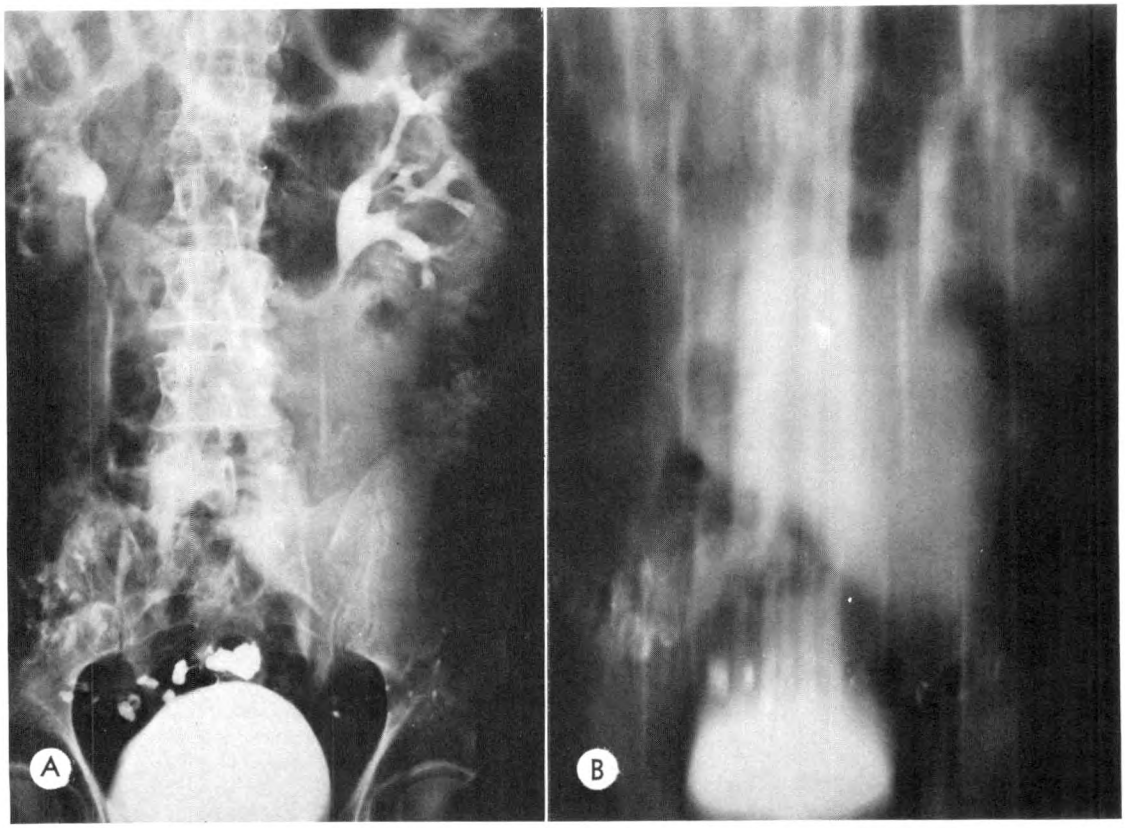

Fig. 10. (S.H., Case 22.) (*A*) Infusion urogram (30 min.) showing left midabdominal mass and residual colonic barium and (*B*) urographic tomogram (17 cm.) demonstrating sharply marginated opacified solid tumor (*mesenteric fibrosarcoma*).

(uterine, ovarian, etc.). Good quality tomography will clearly demonstrate a wall of variable thickness in cystic masses in many cases not otherise delineated by standard high dose urography (Fig. 3; and 5). The non- or poorly opacified solid uterine fibroid will fail to show a wall, has a lobular surface (Fig. 1*A*; and 4), and may be seen to arise from the uterus with tomographic sections at appropriate levels (Fig. 6). Thus, with tomography, that group of nonopacifying pelvic-abdominal masses can usually be differentiated. Further, the addition of tomography in the evaluation of the opacifying mass provides greater definition and increased diagnostic accuracy (Fig. 2).

The differentiation of the cystic pelvic-abdominal mass from nearby adjacent bowel is not difficult (Fig. 9) since wall thickness, change in shape, and tubular character of the bowel become apparent on serial tomograms. The opacified adjacent uterus is easily identifiable when appropriate tomographic levels are obtained. The bladder can be segregated because of its location and should be sought for on later

Table III

PELVIC-ABDOMINAL TOMOGRAPHY UROGRAPHY

Indications
Post menopausal female
Known or suspected mass
Premenopausal female
Septicemia, suspected abscess
Malignancy known or highly suspect
Male
Any age; gonadal shielding
Contraindications
Possible or known pregnancy
History of serious reaction to contrast medium

TABLE IV

RADIATION EXPOSURE DURING PELVIC-ABDOMINAL TOMOGRAPHY AND STANDARD ROENTGENOGRAPHY*

	kVp	mA	Time (sec.)	Absorbed dose (mrads)†	Ovarian Level (cm.)†
Standard Roentgenography	70	300	0.1	80	10
	80	300	0.1	102	12.5
	90	300	0.1	143	12.5
Tomography	68	200	2.0	200	10
	68	200	2.0	235	12.5

* Courtesy of Edward W. Webster, Ph.D., Section of Radiologic Physics.
† 20 cm. phantom (10 cm. ovary).
25 cm. phantom (12.5 cm. ovary).

urographic films to exclude a dilated bladder or a large bladder diverticulum which may simulate a pelvic mass. It should be noted that the case of fibrosarcoma of the mesentery in a male (Fig. 10) demonstrates that this technique need not be limited to the evaluation of female pelvic-abdominal masses.

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Superior technical assistance of Misses Brown, Rivers, Turner, and Circhy is acknowledged.

REFERENCES

1. Birnholz, J. C. Uterine opacification during excretory urography: definition of previously unreported sign. *Radiology*, 1972, *105*, 305–307.
2. Imray, T. J. Evaluation of pelvic masses during infusion excretory urography. Presented at the 75th Annual meeting of the American Roentgen Ray Society, San Francisco, California, 1974, Sept. 24–27.
3. Love, L., Melamed, M., Cooper, R. A., Moncada, R., and Schwartz, H. Infusion tomography of female pelvis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *122*, 299–307.
4. Martin, D. J., Griscom, N. T., and Neuhauser, E. B. D. Further look at total body opacification effect. *Brit. J. Radiol.*, 1972, *45*, 185–192.
5. O'Connor, J. F., and Neuhauser, E. B. D. Total body opacification in conventional and high dose intravenous urography in infancy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 63–71.
6. Phillips, J. C., Easterly, J. F., and Langston, J. W. Contrast enhancement of pelvo-abdominal masses: rim sign. *Radiology*, 1974, *112*, *112*, 17–21.

Vol. 125, No. 2

TOMOGRAPHY OF THE KIDNEY BED AS AN AID IN DIFFERENTIATING RENAL PELVIC TUMOR AND STONE*

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ABSTRACT:

Plain film tomography is of value in detecting small amounts of calcium in "non-opaque" stones. The use of this technique to help differentiate renal pelvic tumor from stone in patients presenting with a filling defect in the renal pelvis is suggested. Four cases in which plain film tomography detected calcium in a renal pelvic stone that could not be seen on routine roentgenograms are presented.

TOMOGRAPHY of the kidney bed has been of great help in differentiating renal pelvic stone from tumor. We have seen 4 cases recently in which a filling defect in the renal pelvis was demonstrated by pyelography and a renal pelvic tumor was suspected. The plain roentgenogram of the abdomen failed to reveal calcification indicative of a calcified renal pelvic stone. However, tomography of the kidney bed (without contrast medium) revealed faint calcification corresponding to the defect and indicated that the pelvic filling defect was a calculus. The presentation of the roentgenographic findings in these cases is the purpose of this report.

DISCUSSION

The radiolucent filling defect of the renal pelvis has often posed a problem in differential diagnosis, frequently necessitating surgery in order to determine its etiology. Urography, retrograde pyelography, and clinical and laboratory data may not be diagnostic in many cases. The major considerations in the differentiation of a radiolucent filling defect of the renal pelvis are calculus, tumor, and blood clot. Less often the defect is caused by inflammatory conditions such as pyelitis cystica or fungus ball, or by a variety of pseudotumors including vascular impressions.[2,5] These latter conditions can usually be readily differentiated by clinical history, laboratory findings, or renal angiography.

However, often neither the urogram, pyelogram, angiogram, clinical presentations nor laboratory findings are conclusive in deciding whether clot, tumor, or calculus is present. Any of the 3 may present with hematuria, and all of them may obstruct the ureteropelvic junction causing hydronephrosis and colicky pain. In contradistinction to calculi or tumors, blood clots usually change in size and shape over several days, so that a diagnosis of this condition can be established by serial studies which obviate the need for surgery. Pelvic tumors are the most difficult to differentiate from radiolucent calculi. Urine cytology positive for malignant cells, when present, establishes the diagnosis of tumor. However, if urine cytology is negative the dilemma regarding the etiology of the pyelographic filling defect remains. "Brushing" of pelvic lesions with pelvic catheters to obtain cytology has been tried with limited success and potential hazard[4] while endoscopy to visualize these lesions has yet to be perfected.

The ability to demonstrate calcification in the filling defect enables a definite diagnosis of renal pelvic stone to be made in these cases. Renal pelvic tumors rarely, if ever, calcify, and if they do the calcification is scanty and stippled as compared to the

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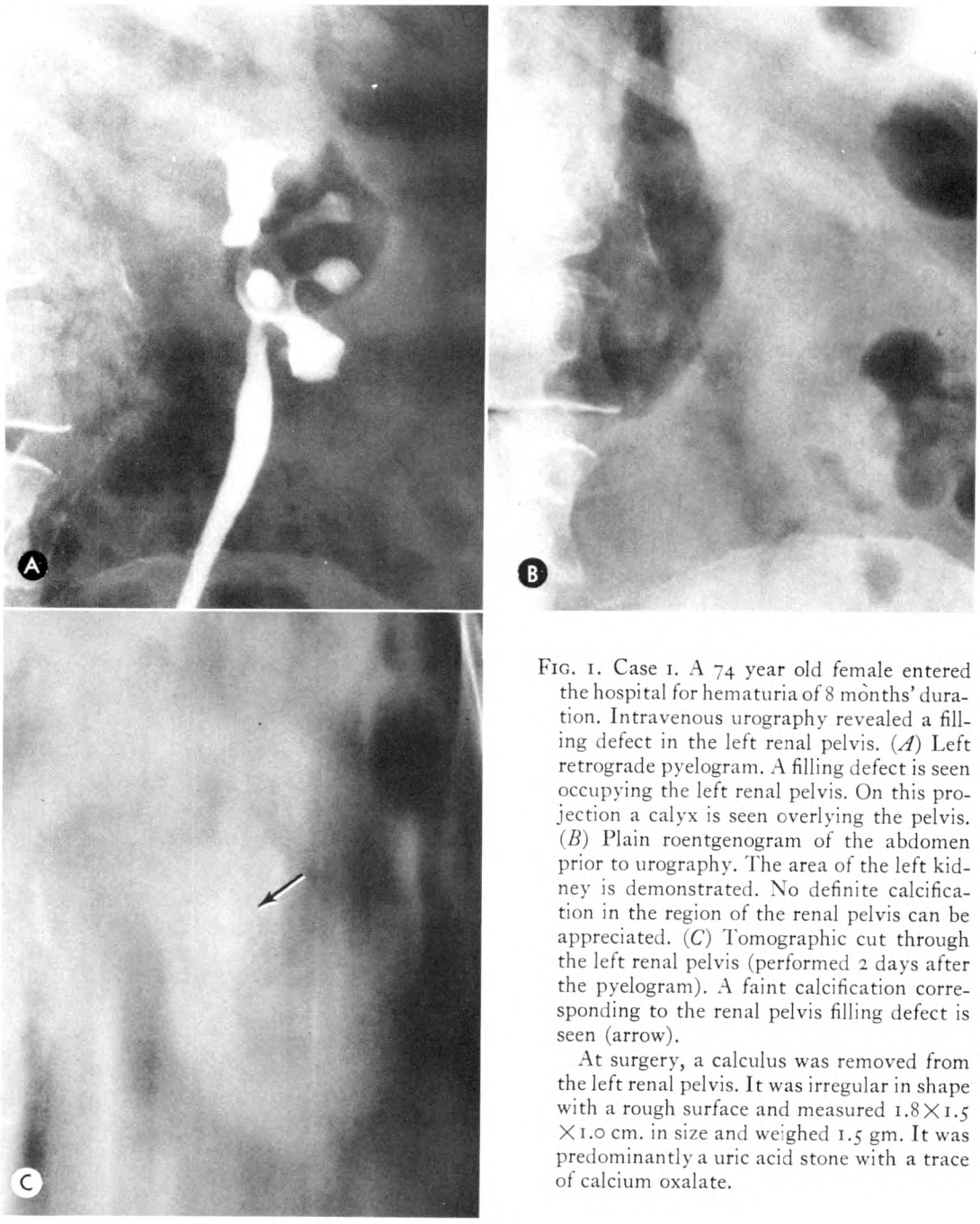

Fig. 1. Case 1. A 74 year old female entered the hospital for hematuria of 8 months' duration. Intravenous urography revealed a filling defect in the left renal pelvis. (*A*) Left retrograde pyelogram. A filling defect is seen occupying the left renal pelvis. On this projection a calyx is seen overlying the pelvis. (*B*) Plain roentgenogram of the abdomen prior to urography. The area of the left kidney is demonstrated. No definite calcification in the region of the renal pelvis can be appreciated. (*C*) Tomographic cut through the left renal pelvis (performed 2 days after the pyelogram). A faint calcification corresponding to the renal pelvis filling defect is seen (arrow).

At surgery, a calculus was removed from the left renal pelvis. It was irregular in shape with a rough surface and measured 1.8×1.5×1.0 cm. in size and weighed 1.5 gm. It was predominantly a uric acid stone with a trace of calcium oxalate.

homogeneous and ring-like calcification of stones. Radiolucent calculi are generally composed of uric acid or less often cystine; however, a "pure" stone is extremely rare (except in patients with gout, leukemia, or lymphoma). Most calculi are a mixture of substances, and uric acid calculi frequently contain small amounts of calcium oxalate or calcium phosphate.[3] The amount of calcium may not be sufficient to be detected on plain roentgenograms of the abdomen but may be seen when tomography is utilized as shown in these cases. In 2 of the cases (Case 1 and IV; Fig. 1; and 4)

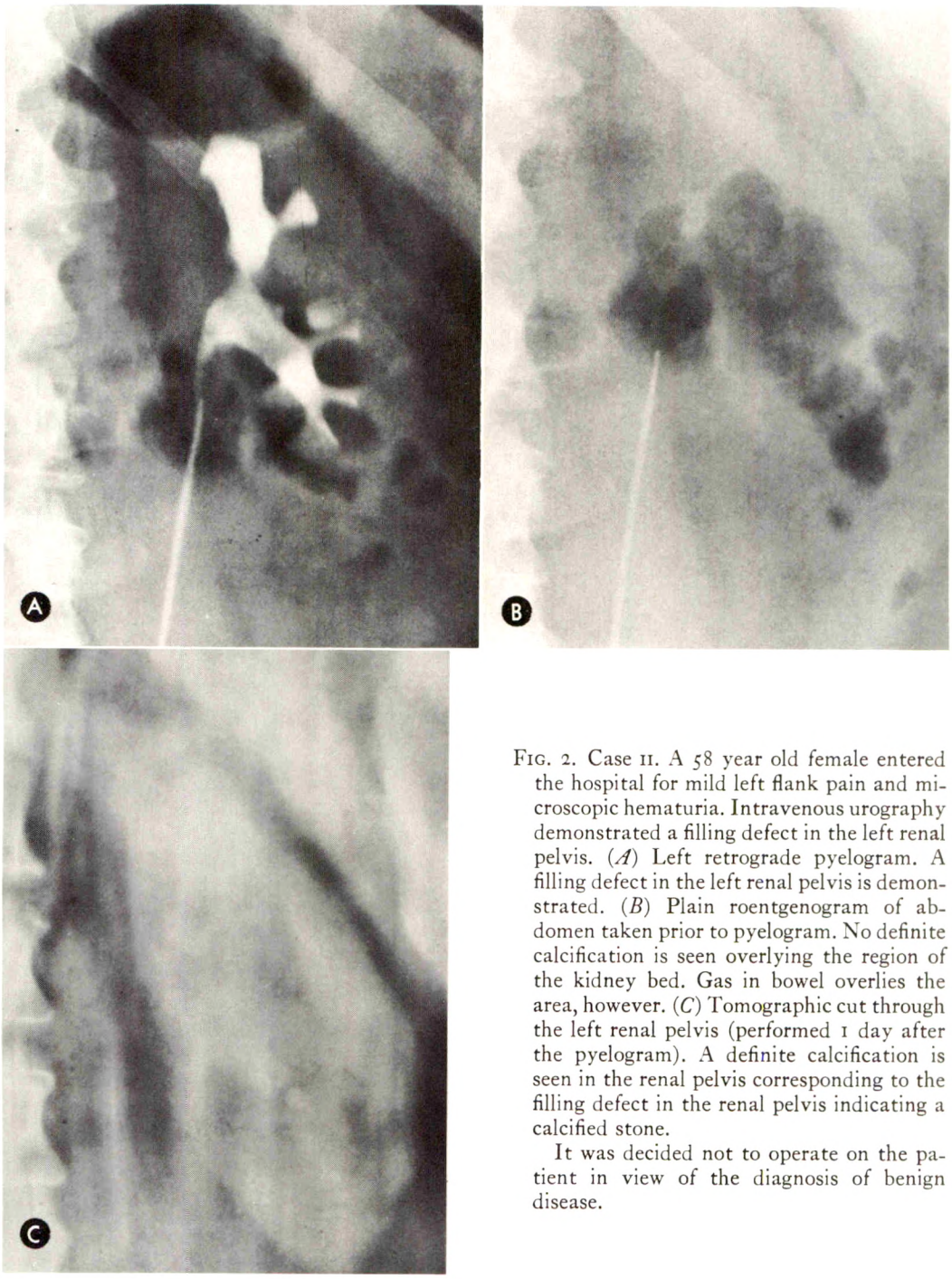

Fig. 2. Case 11. A 58 year old female entered the hospital for mild left flank pain and microscopic hematuria. Intravenous urography demonstrated a filling defect in the left renal pelvis. (*A*) Left retrograde pyelogram. A filling defect in the left renal pelvis is demonstrated. (*B*) Plain roentgenogram of abdomen taken prior to pyelogram. No definite calcification is seen overlying the region of the kidney bed. Gas in bowel overlies the area, however. (*C*) Tomographic cut through the left renal pelvis (performed 1 day after the pyelogram). A definite calcification is seen in the renal pelvis corresponding to the filling defect in the renal pelvis indicating a calcified stone.

It was decided not to operate on the patient in view of the diagnosis of benign disease.

surgery was performed and uric acid calculi mixed with calcium oxalate were removed. The other 2 cases (Fig. 2; and 3) were poor operative risks and were not operated on because of the knowledge that a stone and not a tumor was present. Accurate diagnosis of stone disease, besides preventing unnecessary surgery (when surgery for calculous disease is not indicated), enables the surgeon to make a suitable

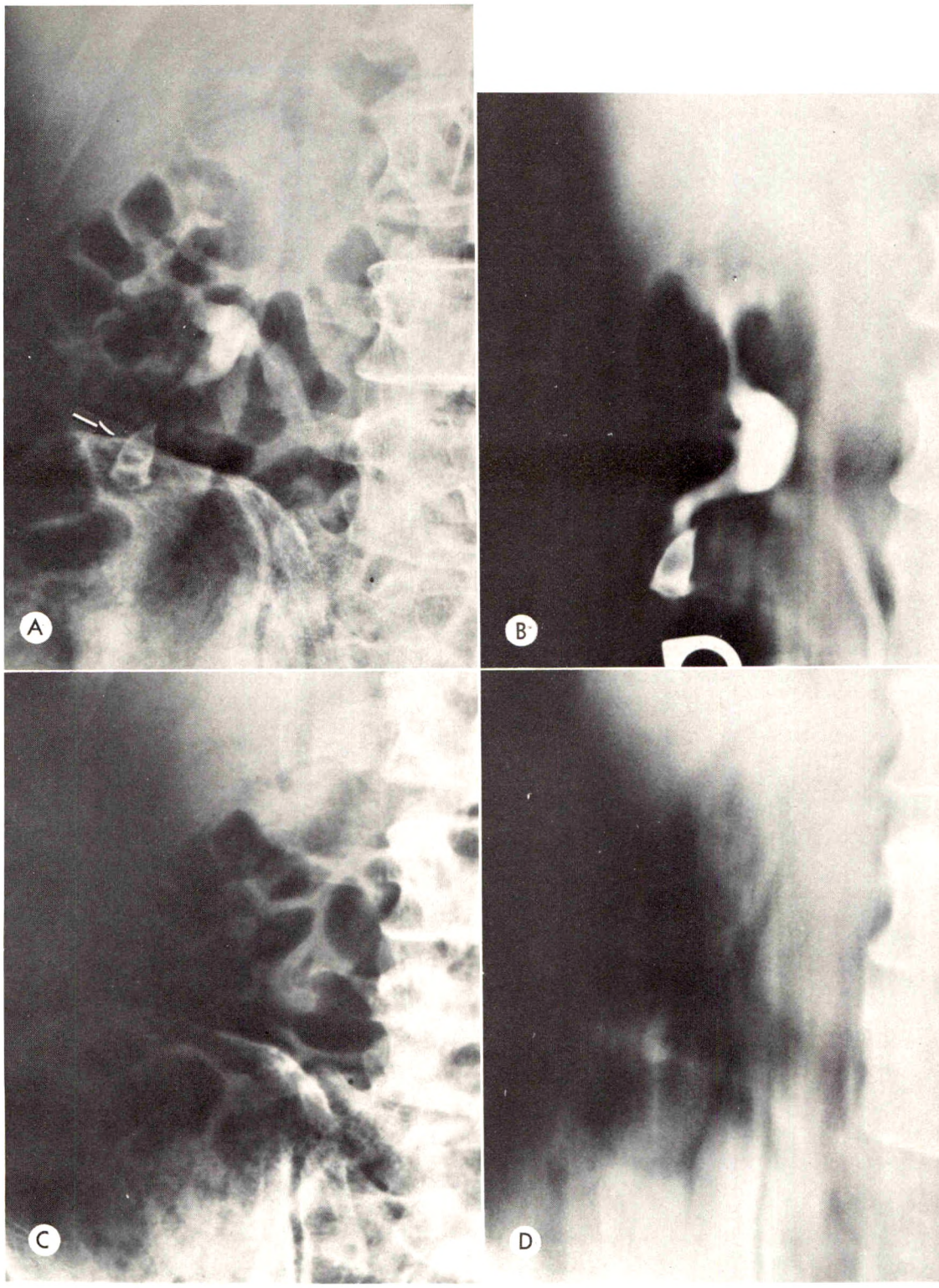

FIG. 3. Case III. A 71 year old male entered the hospital because of symptoms of prostatism. (*A*) Intravenous urogram. The right kidney is somewhat inferiorly placed with the lower pole overlying the iliac crest. A filling defect is seen in the lower pole calyx (arrow). (*B*) Tomographic cut of right kidney (taken during urogram). The irregular filling defect in the lower pole calyx is seen to better advantage. (*C*) Plain roentgenogram of abdomen (taken prior to urogram). Large amount of gas overlies the right kidney. A calcification in this area is not seen. (*D*) Tomographic cut of right kidney (taken 1 day after urogram). A calcification at the lower pole of the right kidney is clearly seen which corresponds to the filling defect on the urogram and represents a calcified stone.

The patient underwent prostatectomy. No surgery on the right kidney was performed.

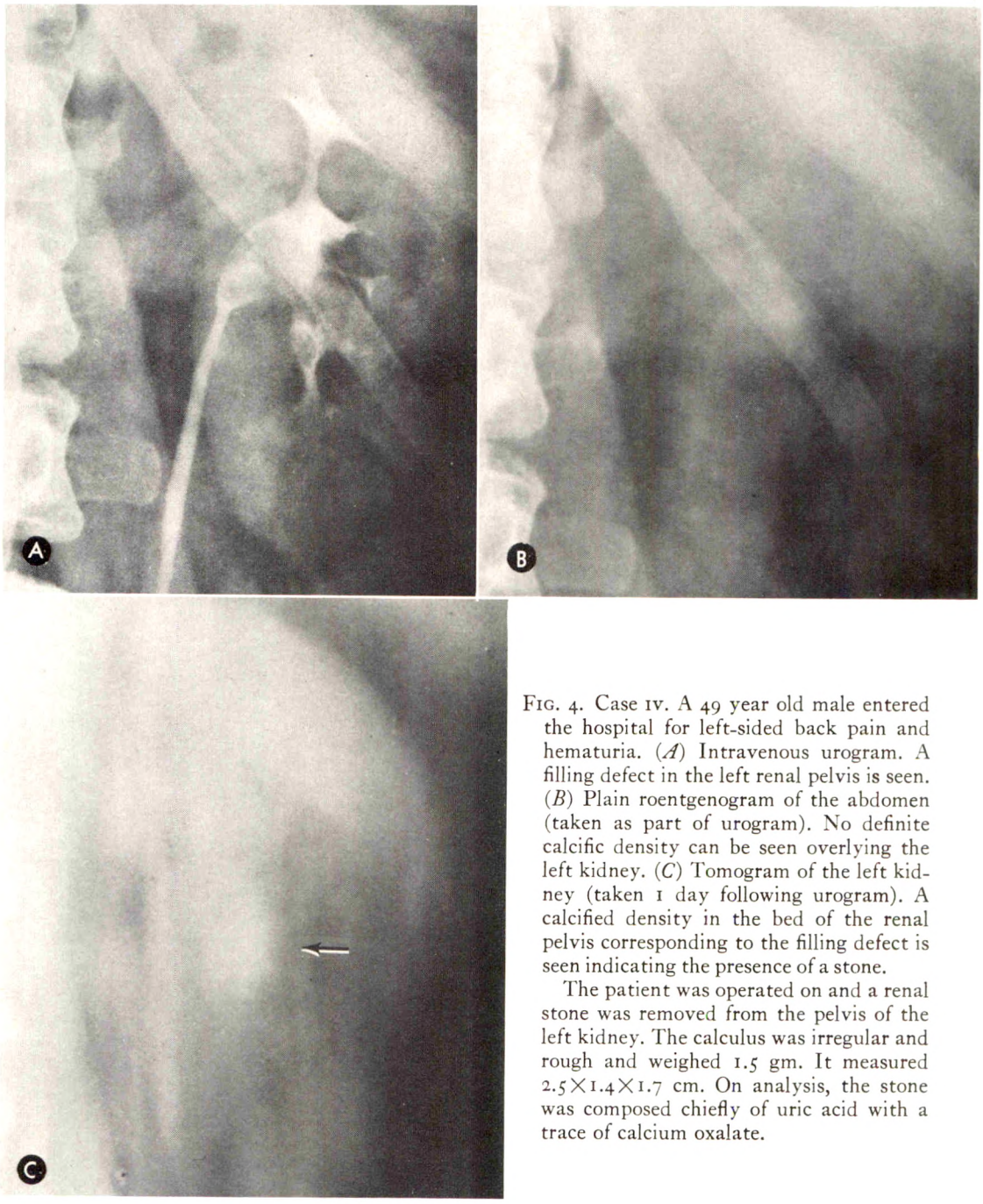

Fig. 4. Case IV. A 49 year old male entered the hospital for left-sided back pain and hematuria. (*A*) Intravenous urogram. A filling defect in the left renal pelvis is seen. (*B*) Plain roentgenogram of the abdomen (taken as part of urogram). No definite calcific density can be seen overlying the left kidney. (*C*) Tomogram of the left kidney (taken 1 day following urogram). A calcified density in the bed of the renal pelvis corresponding to the filling defect is seen indicating the presence of a stone.

The patient was operated on and a renal stone was removed from the pelvis of the left kidney. The calculus was irregular and rough and weighed 1.5 gm. It measured 2.5×1.4×1.7 cm. On analysis, the stone was composed chiefly of uric acid with a trace of calcium oxalate.

flank approach to the lesion for pyelolithotomy knowing that he will not have to remove the kidney for tumor.

We have used tomography with an arc of 25°–30° in these cases (as in nephrotomography) which gives a 0.3–0.5 cm. thick cut.[1] Most of the filling defects are larger than this size and therefore should be well demonstrated with cuts of this width if multiple cuts every 0.5 or 1 cm. are obtained. A greater arc such as 40° would give a thinner tissue cut (0.1–0.2) and therefore

might detect small amounts of calcium but the chance of missing the lesion could occur and many more tomographic cuts to cover the pelvic area would be necessary. Arcs of less than 20° would give tissue cuts which were too thick and might miss the small amount of calcium in these stones. An ideal method might be to localize the defect with a tomographic cut with the 25°–30° arc and then obtain thinner tissue cuts with a 40° arc for maximum detail.

The use of plain film tomography of the kidney bed has been described previously to detect and better evaluate the number of renal calculi present in patients suspected of this condition.[6] The author studied by plain film tomography 97 patients with clinical suspicion of intrarenal calcifications. It was possible to demonstrate calcifications not seen on plain roentgenograms in 7 patients. In 37 of the patients calcifications were noted on plain roentgenograms, but tomography showed them to be more extensive than had been appreciated.

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The authors would like to thank Mr. Scott Warren and Ms. Ruth Kramer for technical assistance and Mr. George Ozaki and Mr. Brian Zietlow for photographic work.

REFERENCES

1. Bosniak, M. A. Nephrotomography: relatively unappreciated but extremely valuable diagnostic tool. *Radiology*, 1974, *113*, 313–321.
2. Brown, R. C., Jones, M. C., Jr., Boldus, R., and Flocks, R. H. Lesions causing radiolucent defects in renal pelvis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *119*, 770–778.
3. Emmett, J. L., and Witten, D. M. Clinical Urography. Third edition. W. B. Saunders Company, Philadelphia, 1971, p. 608.
4. Gill, W. B., Lu, C. T., and Thomsen, S. Retrograde brushing: new technique for obtaining histologic and cytologic material from ureteral, renal pelvic and renal caliceal lesions. *J. Urol.*, 1973, *109*, 573–578.
5. Grace, D. A., Taylor, W. N., Taylor, J. N., and Winter, C. C. Carcinoma of renal pelvis: 15 year review. *J. Urol.*, 1967, *98*, 566–569.
6. Madsen, E. H. Value of tomography for demonstration of small intrarenal calcifications. *Brit. J. Radiol.*, 1973, *45*, 203–206.

VOL. 125, NO. 2

LATERAL DISPLACEMENT OF THE BLADDER*

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ABSTRACT:

Displacement of the bladder from its usual midline position is almost always significant. Most of the common and uncommon causes of such displacement are illustrated, described, and discussed.

LATERAL displacement of the bladder, as with other midline structures (for example, the pineal body or mediastinum), should always be considered abnormal and requires explanation. A list of causes of lateral displacement of the bladder that we have seen roentgenographically upon review of our departmental files is presented in Table 1. Even if the cause is not readily apparent from the roentgenogram, lateral displacement of the bladder implies the presence of a mass or mass effect.

Analysis of the tissues and organs surrounding the bladder reveals some of the potential causes of displacement from the midline. Immediately lateral to the bladder lie the blood vessels, lymph nodes, and areolar connective tissue, with the hips and innominate bones situated more laterally (Fig. 1). The remnants of the müllerian and wolffian ducts (the uterus and the vagina in females and the seminal vesicles and vasa deferentia in males) are posterior to the bladder, with the rectum still more posterior. The so-called "true ligaments" of the bladder extend from the lateral pelvic wall to the lateral wall of the bladder. They are formed by a thickening of endopelvic fascia and extraperitoneal tissue surrounding the neurovascular bundle that supplies the bladder. These "ligaments" do not effectively prevent bladder displacement.[5]

PELVIC TISSUES LATERAL TO THE BLADDER

Blood Vessels. The external iliac artery, internal iliac artery and its branches, and pelvic veins are lateral to the bladder. Pelvic hematomas frequently result from penetrating or nonpenetrating injuries to the bony pelvis. Although such hematomas usually surround the bladder and produce a characteristic elevation and symmetrical compression from both sides ("tear-drop bladder"),[13] occasionally they are unilateral and displace the bladder from the midline (Fig. 2*A*). Visualization of a pelvic fracture or bullet fragments usually suggests a hematoma as the cause of the bladder displacement. A hematoma may occur following cystoscopy, presumably as a result of a quick-sealing perforation or injury to perivesical veins about the bladder base (Fig. 2, *B* and *C*). In addition, leakage of contrast material outside the bladder may occur during cystography following such an injury.

Massive extraperitoneal hemorrhage is a serious and life-threatening complication

TABLE I

CAUSES OF LATERAL DISPLACEMENT OF THE BLADDER

Aneurysm	Soft tissue tumor
Hematoma	Fecal impaction
Lymphadenopathy	Inguinal hernia
Lymphocele	Prostate tumor
Bladder diverticulum	Adnexal cyst or tumor
Innominate bone tumor	Urachal remnant
Rheumatoid cyst	Extension of thigh or retroperitoneal abscess or tumor
Hip arthroplasty	
Abscess	Sacral meningomyelocele

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† James Picker Foundation Scholar in Radiological Research.

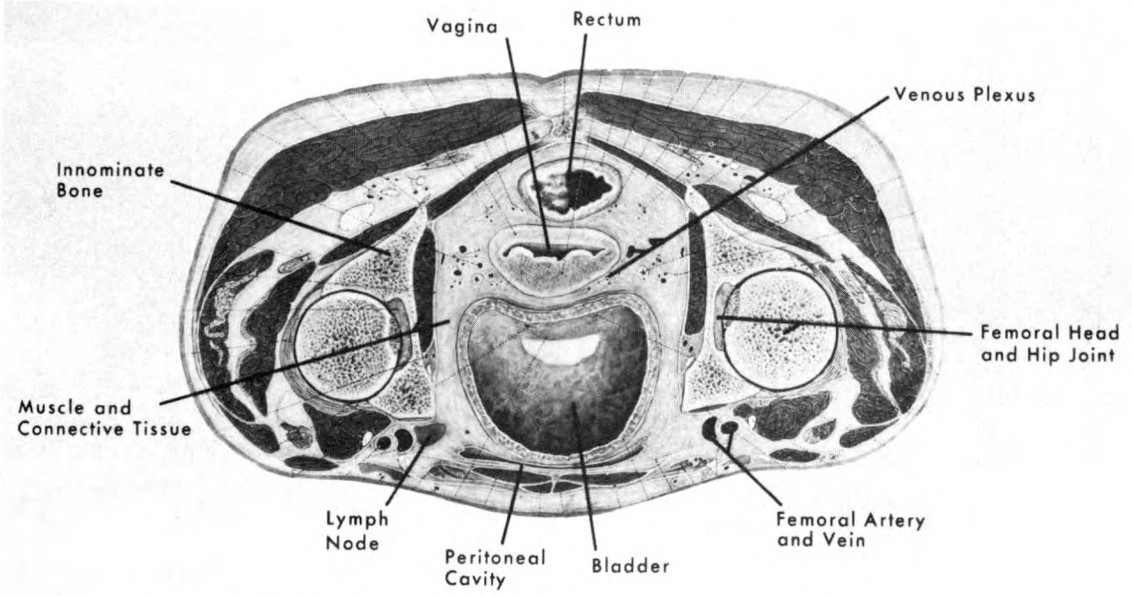

FIG. 1. Cross-section of female pelvis through the level of the bladder. (Modified, with permission from Eycleshymer and Schoemaker and Appleton-Century-Crofts.[3]

of pelvic fractures;[8] a pelvic hematoma seen on urography may be the first indication of a severe hemorrhage. Although bleeding caused by pelvic fractures was thought to originate in the myriad of pelvic veins near the fracture site, recent studies have indicated that certain pelvic fractures may be associated with injuries of specific arteries or large veins.[9,15] In such cases angiography may be required to define the bleeding site for pharmacoangiographic or surgical intervention. Because the extent of such hemorrhage may be difficult to assess clinically, the presence of stability of lateral displacement of the bladder may be of great clinical significance.

Lymphatics. Involvement of retroperitoneal lymph nodes by lymphoma can affect the course of the ureters, producing focal or extensive lateral displacement of 1 or both ureters. Unilateral iliac lymphadenopathy may displace the bladder from the midline (Fig. 3, *A* and *B*). The presence of palpable lymphadenopathy elsewhere or the previous diagnosis of lymphoma should suggest this cause of bladder displacement. Lymphangiography is necessary for definitive diagnosis.

Pelvic lymphoceles or lymphocysts arise in over 20 per cent of patients undergoing radical pelvic lymph node dissection[2] and are another cause of bladder displacement. Less commonly, lymphoceles arise as a complication of a renal transplant procedure.[15] To prepare the recipient vascular pedicle, lymphatic tissue surrounding the external iliac and hypogastric vessels must be excised.[6] If the disrupted lymphatic channels are not ligated, postoperative leakage of lymph may accumulate in the retroperitoneal space. Bladder displacement and obstructive uropathy may then be demonstrated by urography, and be associated with gradual reduction of renal function. In the absence of significant obstruction or diminished renal function, surgical management may be deferred to allow the lymph accumulation to resorb spontaneously. Sometimes surgical drainage is necessary (Fig. 3, *C* and *D*).

Bladder. A large, single diverticulum of the bladder commonly occurs posterolaterally and can produce lateral deviation of the remainder of the bladder. Such bladder diverticula usually opacify during intravenous urography and, therefore, allow ready

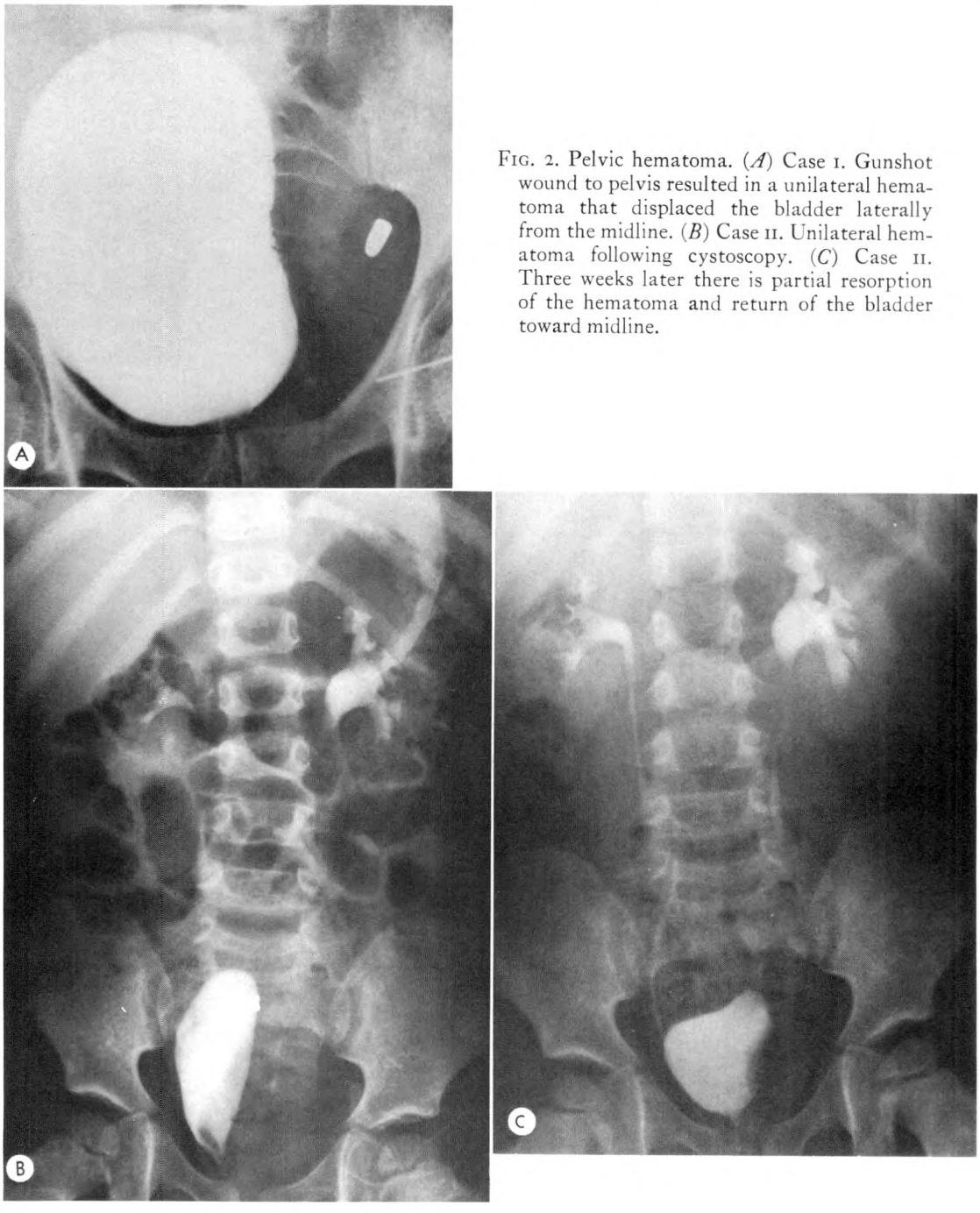

Fig. 2. Pelvic hematoma. (*A*) Case 1. Gunshot wound to pelvis resulted in a unilateral hematoma that displaced the bladder laterally from the midline. (*B*) Case 11. Unilateral hematoma following cystoscopy. (*C*) Case 11. Three weeks later there is partial resorption of the hematoma and return of the bladder toward midline.

explanation of the bladder deviation.[17] Occasionally, however, those diverticula having narrowed or obstructed necks fail to opacify during urography (Fig. 4*A*). In such instances, retrograde cystography will usually demonstrate diverticular filling (Fig. 4*B*). In men, the bladder is usually markedly trabeculated because of outlet obstruction caused by prostatic enlargement. Occasionally a single, large, congenital diverticulum is demonstrated in a child.[4] Although medial deviation of the

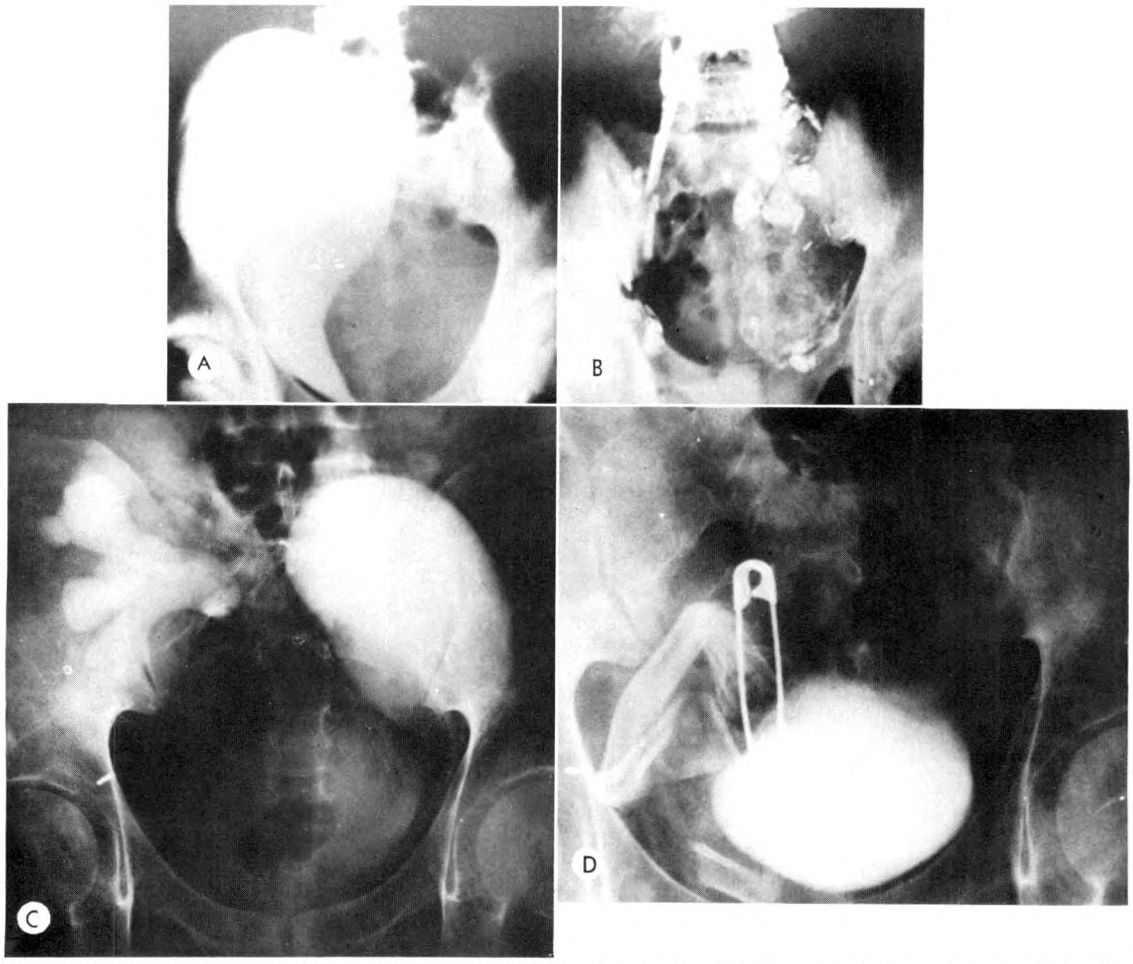

FIG. 3. Lymphatic abnormalities. Case III. Lymphomatous nodes displace the bladder from midline. (*A*) Cystogram and (*B*) lymphangiogram. (*A* and *B* reprinted with permission, from Korobkin *et al.* and *Radiology*.[7]) Case IV. (*C*) A huge lymphocele developed after renal transplant and caused marked hydronephrosis and bladder displacement. (*D*) Five days after drainage of the lymphocele, the bladder resumed its midline position.

distal ureter is reportedly characteristic[17] and helpful in the diagnosis of single, large, bladder diverticulum, this finding is nonspecific because it occurs with other lesions that produce lateral deviation of the bladder.

Hip and Innominate Bone. Medial expansion of any bone tumor originating in the lateral pelvic wall may eventually displace the bladder from the midline. Tumors most frequently arising from the innominate bone include aneurysmal bone cyst, osteogenic sarcoma, Ewing's sarcoma, and chondrosarcoma.[10] Although the specific cause of bladder displacement is usually obvious in these instances (Fig. 5*A*), the diagnosis of aneurysmal bone cyst may be difficult if the medial margin is not apparent roentgenographically (Fig. 5*B*).

Rheumatoid arthritis of the hip can result in inflammatory cysts of the adjacent bursae or adjacent bone.[18] Although it is rare, these cysts can project into the pelvic soft tissues, producing either unilateral or bilateral compression of the bladder. If the compression is unilateral, the bladder is displaced lateral to the midline. Bilateral compression roentgenographically simulates pelvic lipomatosis or diffuse perivesical hematoma.

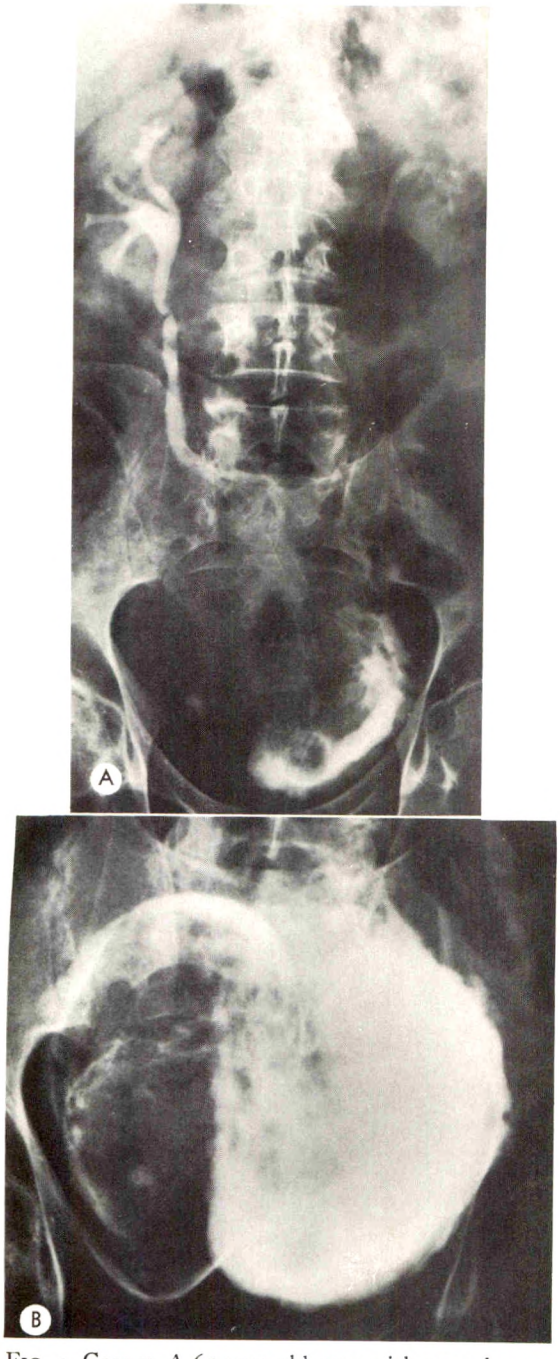

Fig. 4. Case v. A 65 year old man with gross hematuria. (*A*) Excretory urogram shows lateral displacement of the bladder, which contains a large blood clot. (*B*) Cystogram shows hugh diverticulum filled with clot and outlined by contrast material.

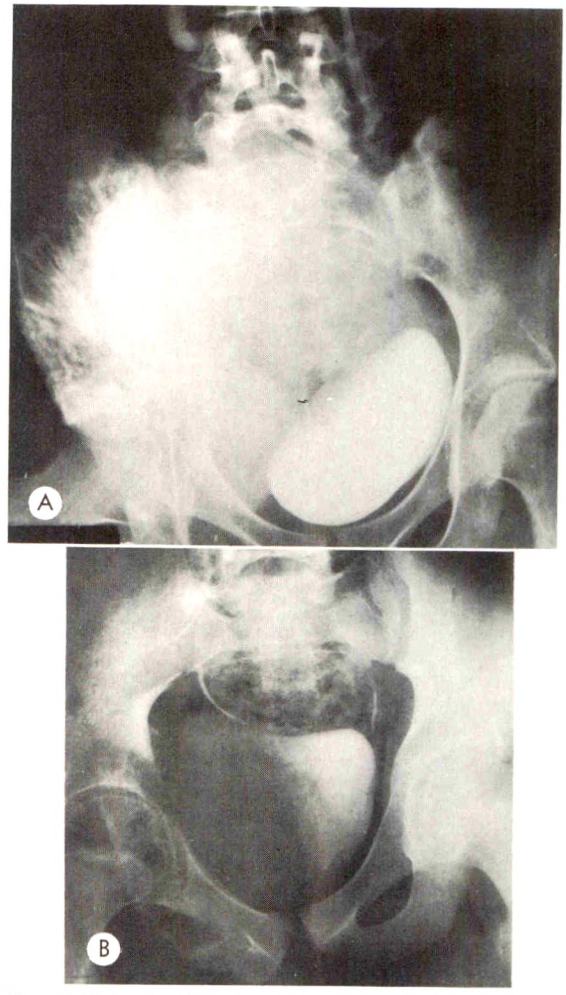

Fig. 5. Tumors of the innominate bone. (*A*) Case vi. Osteogenic sarcoma of the ilium displaces the ureter and bladder. (*B*) Case vii. Aneurysmal bone cyst arising in the ischium. The osseous nature of the pelvic mass is not apparent on this examination.

Total hip arthroplasty has come into widespread use in the therapy of severe osteoarthritis and rheumatoid arthritis.[14] The Charnley-Müller and McKee-Farrar prostheses, commonly used at our hospital, consist of a metallic femoral component and an acetabular cup. The acetabular cup is anchored in place by an acrylic resin deposited in the surgically enlarged acetabulum and in holes drilled in the innominate bone. Resin may occasionally extrude into the pelvis because of excessive enlargement of the acetabulum or infection that results in an abscess extending into the pelvis. The soft tissue mass that results may displace the bladder from the midline.

Areolar Connective Tissue. Pelvic ab-

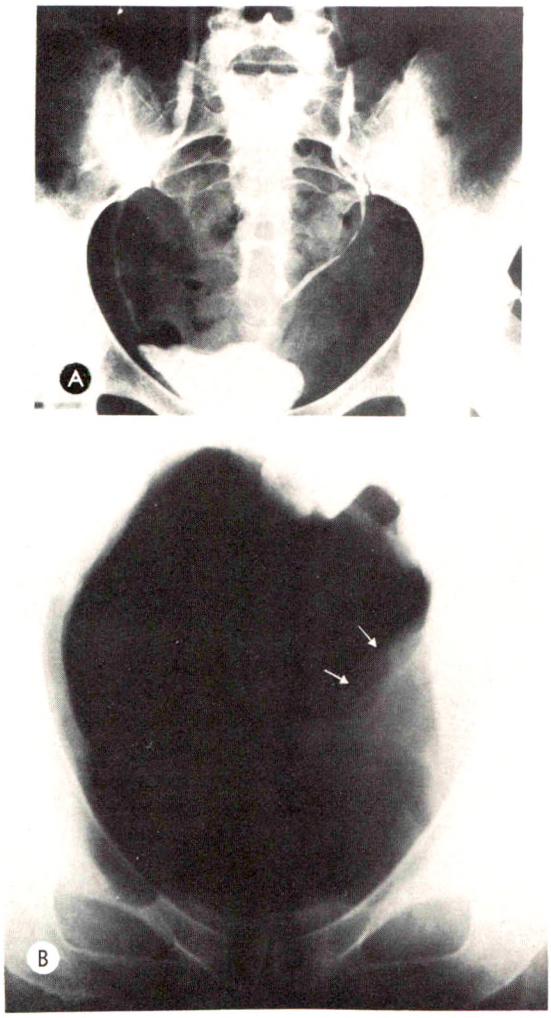

FIG. 6. Tumors of pelvic connective tissue. Case VIII. (*A*) Benign myxoma producing slight medial displacement of the ureter and lateral displacement of the bladder. (*B*) Pelvic pneumogram shows the soft tissue tumor arising from the lateral pelvic wall (arrows).

scesses can complicate inflammatory disease of the fallopian tubes or the appendix. History and physical findings often indicate the source of any abnormalities that may be seen roentgenographically. A variety of soft tissue tumors can arise from the connective tissue lateral to the bladder. We encountered 2 benign myxomas that displaced the bladder laterally: 1 arose near the bladder floor, elevating the bladder and displacing it to the iliac bone; the other occurred higher in the pelvis and was clearly

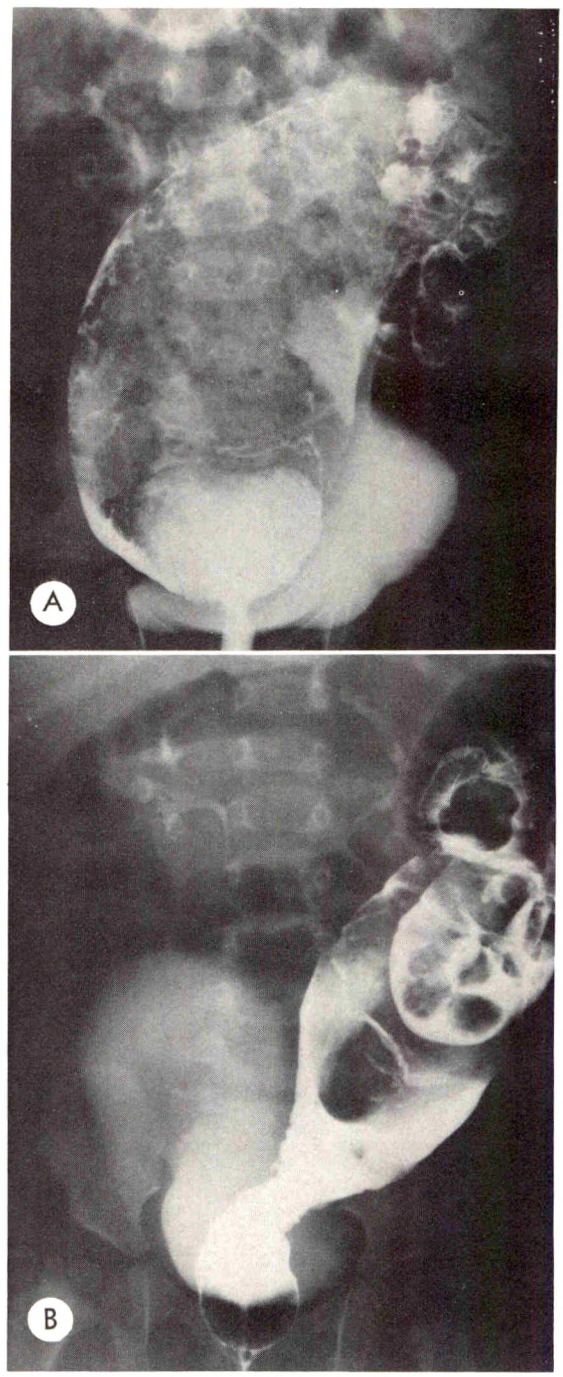

FIG. 7. Megacolon. (*A*) Case IX. Barium enema examination demonstrates the massive distention of the rectosigmoid colon down to the level of the anus. Biopsy specimen showed normal ganglion cells in the distal rectum. (*B*) Case X. Because the aganglionic segment of the colon lies well proximal to the anus in this patient with Hirschsprung's disease, the lower portion of the bladder is not laterally displaced.

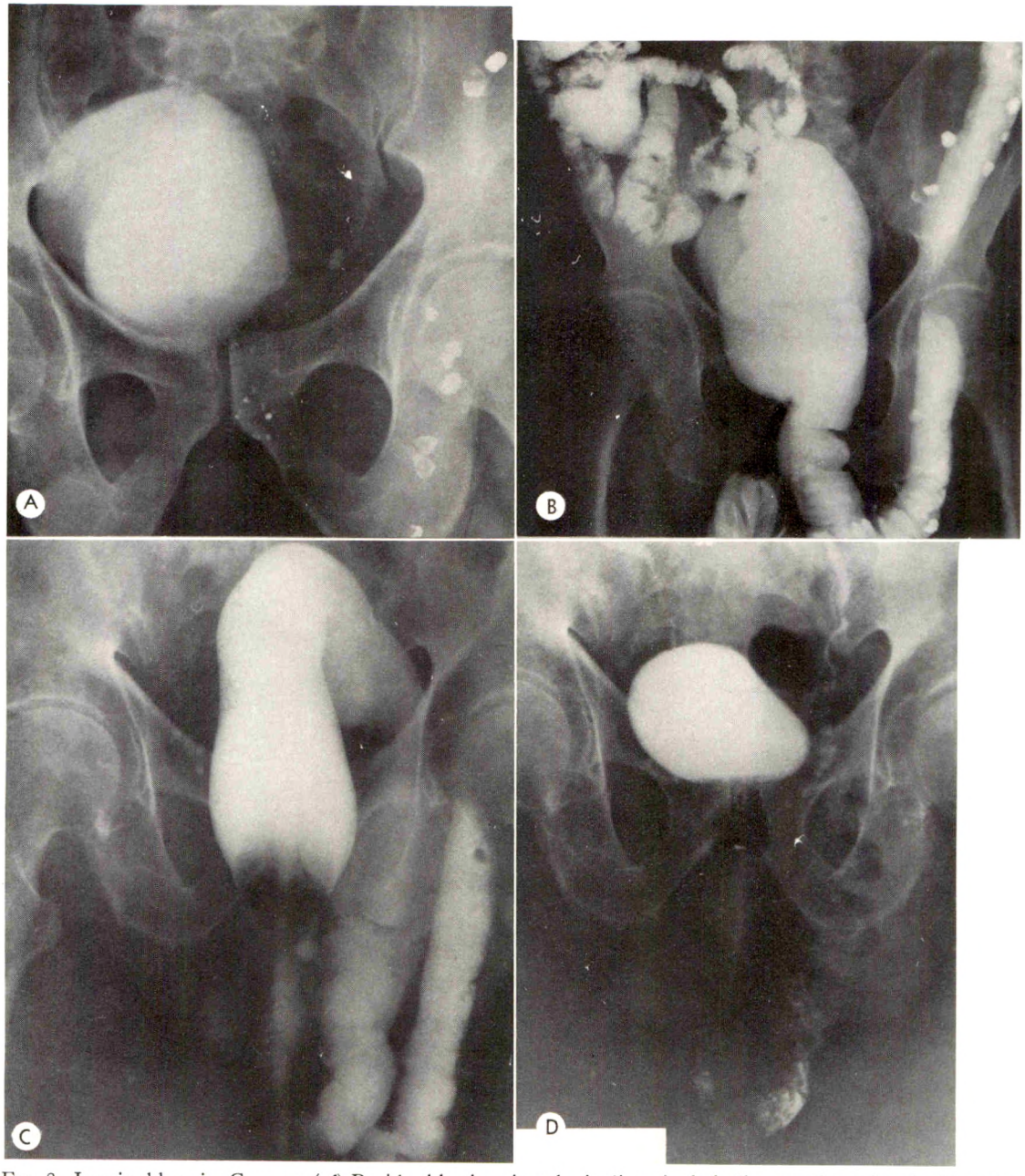

Fig. 8. Inguinal hernia. Case xi. (*A*) Residual barium in colonic diverticula indicates a large inguinal hernia. (*B*) Note the wide space between the afferent and efferent loops of the hernia at the dilated inguinal ring. Case xii. (*C*) A large scrotal hernia has a much smaller inguinal ring than in Case xi, through which the afferent and efferent loops pass. (*D*) There is only a slight impression on the superolateral aspect of the bladder.

demonstrated by pelvic pneumography (Fig. 6, *A* and *B*). Although these examples are of benign lesions, over 80 per cent of retroperitoneal masses are malignant.[1]

PELVIC TISSUES ADJACENT TO BLADDER

Colon. Although the rectum lies posterior to the bladder, it can distend asymmetrically in the pelvis and displace the bladder

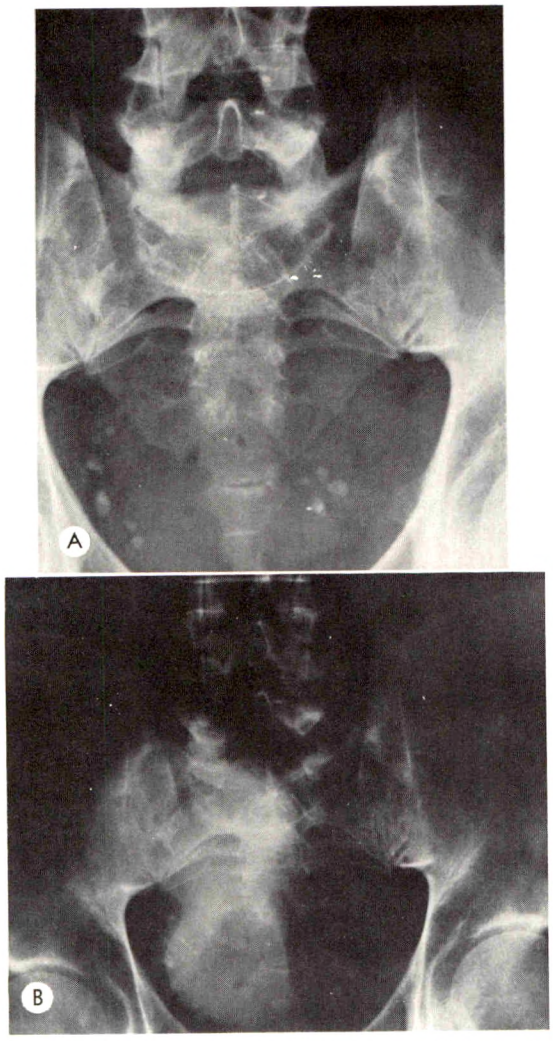

FIG. 9. Case XIII. Prostatic carcinoma. (*A*) Asymmetry of the pelvic phleboliths suggests a possible pelvic mass on the left. (*B*) Lateral displacement of the bladder is produced by the eccentric spread of prostatic carcinoma.

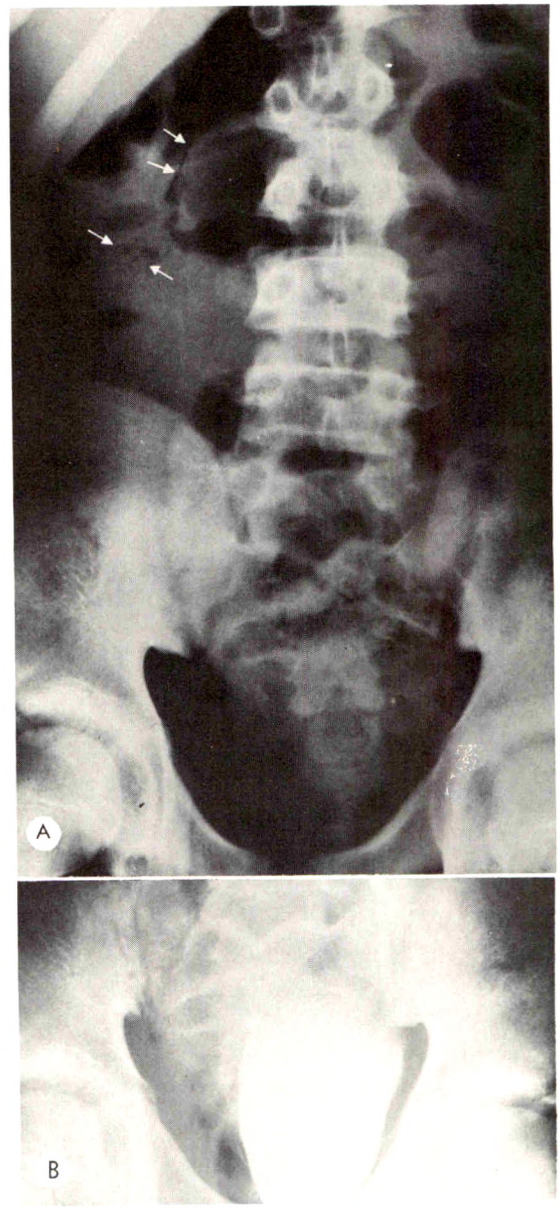

FIG. 10. Case XIV. Perinephric abscess extending into the pelvis. (*A*) Lucent linear streaks and bubbles (arrows) can be seen along the right psoas margin following trauma to the flank. (*B*) Several days later the bladder was laterally displaced by a pelvic abscess.

from the midline. If the level of obstruction is at the anus, as in "psychogenic" megacolon, the entire bladder may be laterally displaced (Fig. 7*A*); if the site of obstruction is more proximal, only the superior portion of the distended bladder may be displaced (Fig. 7*B*).

The frequent association of constipation with urinary tract infection in children is not well recognized. Neumann *et al.*[12] found that abnormal bowel habits suggesting constipation were present in 45 of 131 children with urinary tract infections. In those patients undergoing urography, the bladder wall appeared irregular or the bladder was displaced. When both the constipation and urinary tract infection were treated, only 9 of the 45 patients had recurrence of the

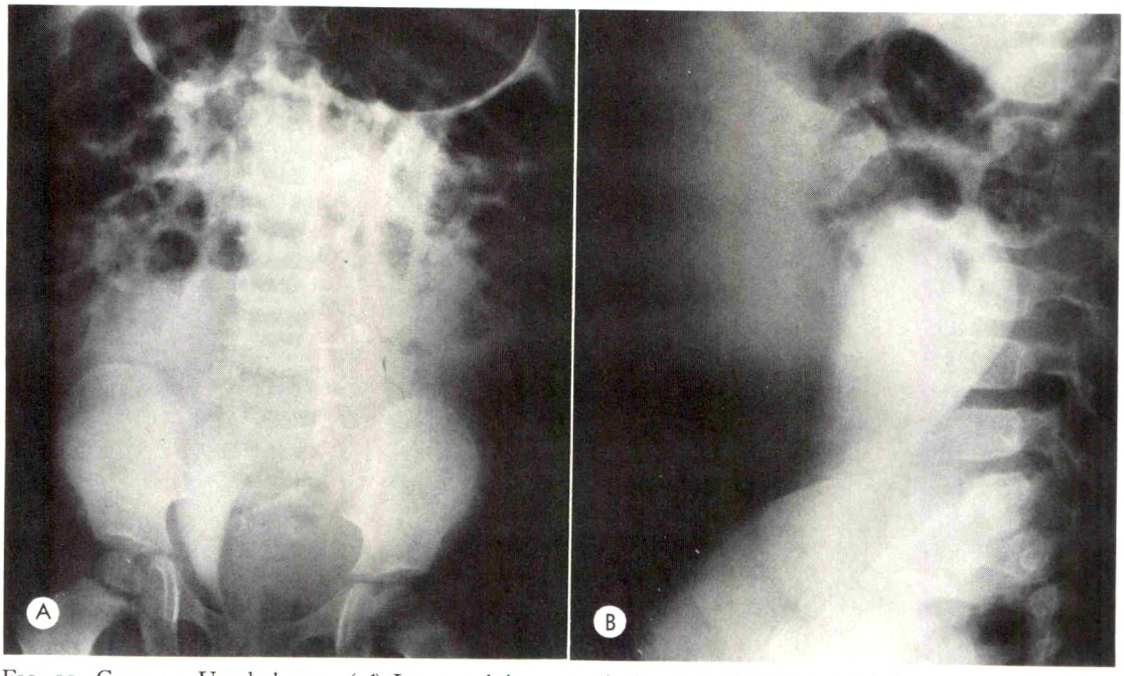

Fig. 11. Case xv. Urachal cyst. (*A*) Large pelvic mass displaces the bladder and left ureter to the right. (*B*) Lateral roentgenogram shows the mass anterior and superior to the bladder, extending to the region of the umbilicus.

infection within 1 year. In a similar group of 18 patients treated for only their urinary tract infection, 16 had a recurrence within 1 year. This suggests that constipation plays a role in the production of urinary tract infections in children. The authors postulated that displacement of the bladder and posterior urethra by the full rectum in the fixed and limited space of the bony pelvis produces interference with micturition and, in turn, urinary stasis.

The inguinal canal is anterolateral to the bladder. Most small inguinal hernias produce no impression on the bladder, but massive colonic hernias into the scrotum may displace the bladder to the side opposite the hernia (Fig. 8, *A* and *B*). The presence and degree of such bladder displacement probably are related to the degree of dilatation of the inguinal ring: the wider the ring, the more widely separated the afferent and efferent colon loops and the greater the unilateral mass effect produced by the bowel loops (Fig. 8, *C* and *D*).

Prostate Gland and Adnexa. Most adnexal tumors ascend into the lower abdomen and produce only a superior impression on the bladder. In our experience, they only rarely displace the bladder from the midline. In a similar fashion, prostate tumors usually impress the bladder from below but only occasionally does the lesion grow eccentrically enough to produce lateral displacement of the bladder (Fig. 9, *A* and *B*).

Spread from Extraperitoneal Tissues

Retroperitoneal Abscess and Neoplasm. The pelvic extraperitoneal space is an extension of the retroperitoneal space. Consequently, it is not surprising that retroperitoneal abscesses and tumors arising in the abdomen can spread directly to the pelvis and displace the bladder. Figure 10, *A* and *B*, illustrates displacement of the bladder by a perinephric abscess that had extended down the psoas sheath into the pelvic soft tissues about the bladder and rectum, displacing the bladder from the midline.

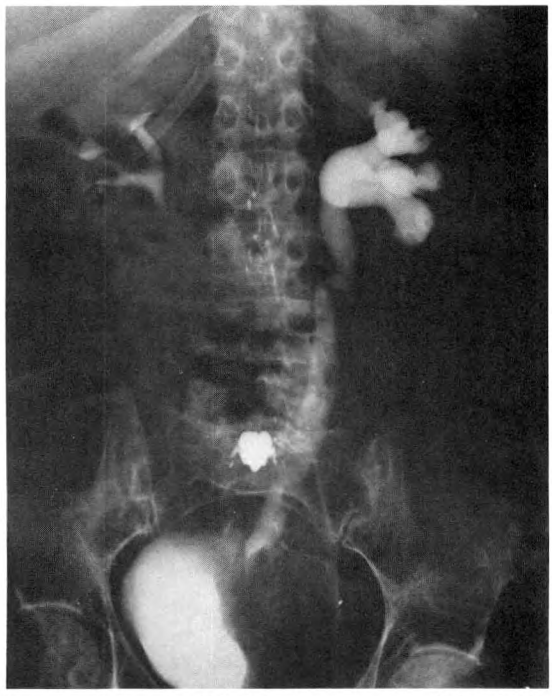

FIG. 12. Case XVI. Rhabdomyosarcoma of the thigh. The tumor has spread directly into the left side of the pelvis, causing ureteral obstruction as well as bladder displacement. Metastasis to L-4 and L-5 is also present.

Urachal Remnant. The urachus is a normal, elongated embryologic remnant of the early communication between the bladder vertex and the allantoic duct.[11] In the normal adult, the urachus is a fibromuscular appendage, with a minute potential lumen. Patency of the urachus anywhere along its embryologic course can result in stasis and a cystic mass. Cystography usually demonstrates those structures that retain bladder communication; injection of contrast material into an umbilical sinus is necessary for diagnosis if the urachal remnant does not communicate with the bladder. Although urachal cysts are usually midline in position, massive distention of these remnants can assume an eccentric pelvic position and displace the bladder to one side (Fig. 11, *A* and *B*). The extraperitoneal origin of this remnant allows easy access into the extraperitoneal pelvic space.

Thigh. Soft tissue tumors of the thigh can spread directly into the pelvis, displacing the bladder from the midline (Fig. 12). This displacement may represent either direct physical extension or lymphatic dissemination. Again, history and physical findings will usually indicate the cause of the bladder displacement.

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We are grateful to the following roentgenologists for the use of their cases in this report: Cases II, IX, and XV, Dr. Hooshang Taybi; Case IV, Dr. John Carolan; Case VIII, Drs. Fred Margolin and James Moore; and Case X, Dr. Walter A. Griesbach.

REFERENCES

1. ARMSTRONG, J. R., and COHN, I., JR. Primary malignant retroperitoneal tumors. *Am. J. Surg.*, 1965, *110*, 937–943.
2. DODD, G. D., RUTLEDGE, F., and WALLACE, S. Postoperative pelvic lymphocysts. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *108*, 312–323.
3. EYCLESHYMER, A. C., and SCHOEMAKER, D. M. A Cross-Section Anatomy. Second edition. Appleton-Century-Crofts, New York, 1970, p. 109.
4. FORSYTHE, I. W., and SMYTH, B. T. Diverticulum of bladder in children: study of 13 cases. *Pediatrics*, 1959, *24*, 322–329.
5. HEALEY, J. E., JR., and SEYBOLD, W. D. A Synopsis of Clinical Anatomy. W. B. Saunders, Philadelphia, 1969, p. 204.
6. KOEHLER, P. R. Injuries and complications of lymphatic system following renal transplantation. *Lymphology*, 1972, *5*, 61–67.
7. KOROBKIN, M., PICK, R. A., MERTEN, D. F., PERLOFF, D. L., and PALUBINSKAS, A. J. Etiologic radiographic findings in children and adolescents with nonuremic hypertension. *Radiology*, 1974, *110*, 615–625.
8. MARGOLIES, M. N., RING, E. J., WALTMAN, A. C., KERR, W. S., JR., and BAUM, S. Arteriography in management of hemorrhage from pelvic fractures. *New England J. Med.*, 1972, *287*, 317–321.
9. MOTSAY, G. J., MANLOVE, C., and PERRY, J. F. Major venous injury with pelvic fracture. *J. Trauma*, 1969, *9*, 343–346.
10. MURRAY, R. O., and JACOBSON, H. G. The Radiology of Skeletal Disorders: Exercises in Di-

agnosis. Williams & Wilkins Company, Baltimore, 1971, pp. 1251–1263.
11. Myers, G. H., Jr. Bladder and urethra. In: Clinical Urography: An Atlas and Textbook of Roentgenologic Diagnosis. Edited by J. L. Emmett and D. M. Witten. Third edition. Volume three. W. B. Saunders Company, Philadelphia, 1971, pp. 1531–1604.
12. Neumann, P. Z., deDomenico, I. J., and Nogrady, M. B. Constipation and urinary tract infection. *Pediatrics*, 1973, *52*, 241–245.
13. Ney, C., and Friedenberg, R. M. Radiographic Atlas of the Genitourinary System. J. B. Lippincott Company, Philadelphia, 1966, pp. 512–518.
14. Pepper, H. W., and Noonan, C. D. Radiographic evaluation of total hip arthroplasty. *Radiology*, 1973, *108*, 23–29.
15. Schweizer, R. T., Cho, S., Kountz, S. L., and Belzer, F. O. Lymphoceles following renal transplantation. *A.M.A. Arch. Surg.*, 1972, *104*, 42–45.
16. Stone, H. H., Rutledge, B. A., and Martin, J. D., Jr. Massive crushing pelvic injuries. *Am. Surgeon*, 1968, *34*, 869–878.
17. Theros, E. G. Editor. Genitourinary Tract Disease Syllabus. American College of Radiology, Chicago, 1973, p. 13.
18. Watson, J. D., and Ochsner, S. F. Compression of bladder due to "rheumatoid" cysts of hip joint. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 695–696.

COMPENSATORY RENAL HYPERTROPHY AFTER TREATMENT FOR WILMS' TUMOR*

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ABSTRACT:

In children who undergo nephrectomy for Wilms' tumor, compensatory renal hypertrophy occurs rapidly despite concomitant irradiation and chemotherapy. Review of preoperative and postoperative intravenous pyelograms of 21 children showed all to have significant enlargement of the remaining kidney. Sixty-five per cent renal hypertrophy occurred during the first 2 months after nephrectomy; by 11 months, renal hypertrophy is complete and renal growth is no longer accelerated.

LAUFER and Griscom[7] showed that compensatory renal hypertrophy in infants with unilateral multicystic kidney disease begins promptly at birth, with the kidney reaching 75 per cent of total normal renal mass at 4 months and approximately 100 per cent of total normal renal mass after 1 year. We evaluated the degree of compensatory renal hypertrophy which occurs following nephrectomy in older children. We believe this information is important in management decisions regarding patients with Wilms' tumor.

The consideration of further insult to the renal function remaining after unilateral nephrectomy for Wilms' tumor always presents a problem in irradiation and chemotherapeutic management. Knowledge of the potential compensatory renal hypertrophy during all phases of the anticipated treatment regimen is essential.

MATERIAL AND METHOD

Twenty-one children with unilateral Wilms' tumor seen from 1965 through 1973 were selected for retrospective evaluation to determine the degree of compensatory renal hypertrophy that occurred following nephrectomy. The youngest patient was 8 months at the time of diagnosis while the oldest was 6 years. The children were arbitrarily grouped by age as follows: 0 to 24 months, 24 to 48 months, and more than 48 months of age. Clinical records showed that all patients had unilateral nephrectomy followed by postoperative irradiation as well as treatment with dactinomycin at the time of operation or shortly thereafter. All children had a preoperative intravenous pyelogram and at least 2, usually 4 or more, postoperative intravenous pyelograms. Kidney size was assessed using measurements of renal length (L) and width (W) and the distance from the top of L_1 to the bottom of L_4 vertebral bodies as seen on the 5 minute postinjection pyelogram.[3] The film at 5 minutes after injection was chosen because it is a routine film time, when the outline of the renal parenchyma is well visualized and maximum postinjection renal swelling occurs.[1]

As a basis for assessing renal size on subsequent pyelograms, several established facts were considered. Kidney weight is best correlated with kidney area.[8] Multiplying kidney length by kidney width gives a linear relationship to postmortem kidney weight.[11] In normal children, a graph of the distance from the top of L_1 to the bottom of L_4 vertebral bodies, according to age, is identical to a graph of kidney length as a function of age.[3,5] Thus the kidney area

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(length times width) and the distance between L_1 and L_4 produce approximately a 1:1 ratio.

We have adopted the phrase relative renal hypertrophy (RRH) to indicate accelerated or augmented renal growth following contralateral nephrectomy. It represents renal growth beyond the expected normal renal growth. One hundred per cent RRH may be defined as the maximum eventual gain in renal size above the expected normal renal size. RRH is derived by dividing the renal area (L×W) by the height of lumbar vertebral bodies (top L_1 to bottom L_4) seen on the child's last pyelogram, then subtracting the similarly calculated measurement made from the child's initial preoperative pyelogram. Percentages less than 100 per cent RRH represent values at times between initial preoperative pyelograms and the time of the final examination. Theoretically, the normal kidney grows at a predictable rate; thus, exaggerated renal growth relative to the age of the child can be detected. Our method simplifies the identification of compensatory renal hypertrophy and also allows for easy follow-up evaluation of the hypertrophy as the child grows.

RESULTS

The age distribution of the 21 patients studied is given in Table I. The per cent RRH as a function of months after diagnosis is shown in Figure 1. The points plotted on the graph represent the average per cent RRH attained following nephrectomy for all patients examined at the time indicated on the abscissa. By 2 months, 65 per cent RRH has occurred, and by 4 months this has increased to 75 per cent RRH. The graph demonstrates a plateau at 90 per cent RRH by 11 months. By this time, renal hypertrophy is complete, *i.e.*, renal growth is no longer accelerated.

When our data are analyzed as a function of the 3 arbitrarily chosen age groups, it is seen that RRH occurred the least rapidly in the children under 2 years of age (Table II). In only 63 per cent of this group was RRH complete 12 months after nephrectomy. In children more than 2 years of age, the process was considerably faster, with at least 50 per cent of the children achieving total RRH by 6 months.

Table I

AGE DISTRIBUTION

Age at Diagnosis	Patients
0–24 months	8 (38 per cent)
24–48 months	7 (33 per cent)
>48 months	6 (29 per cent)

DISCUSSION

Nephrectomy, followed by irradiation and combined dactinomycin and vincristine chemotherapy, remains the treatment of choice for patients with unilateral Wilms' tumor.[9] During irradiation of the nephrectomized kidney bed, radiation is scattered to the remaining kidney. Vaeth[12] found that despite substantial doses of radiation to the remaining kidney it stayed as large or was larger than normal in all children studied, and none developed radiation nephritis up to 10 years following treatment. Mitus and associates[10] state that dactinomycin has not been found to impair renal function. Jereb *et al.*[6] reviewed 16 patients with Wilms' tumor and showed through measurements of glomerular filtration rate and para-aminohippuric acid (PAH) clearance that none had clinically manifest renal dysfunction following treatment with radiation and dactinomycin. However, because dactinomycin is an antimetabolite, renal hyper-

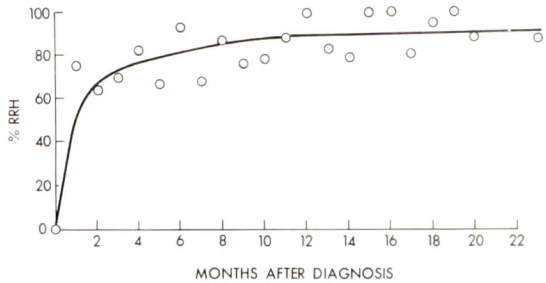

Fig. 1. Per cent of relative renal hypertrophy (RRH) as a function of months after diagnosis.

TABLE II

ATTAINMENT OF COMPLETE RELATIVE RENAL HYPERTROPHY BY AGE AT DIAGNOSIS

Months after Nephrectomy	Per Cent of Age Group Attaining Complete RRH		
	0–24 mo.	24–48 mo.	>48 mo.
3	0	29 per cent	17 per cent
6	13 per cent	57 per cent	50 per cent
9	13 per cent	57 per cent	67 per cent
12	63 per cent	57 per cent	83 per cent

RRH = relative renal hypertrophy.

trophy might well be delayed. Our study does not evaluate compensatory renal growth in the absence of these therapeutic methods—all of our patients had both forms of postoperative treatment. Rather, it reflects the changes in renal growth under the influence of this standardized regimen. Certainly at 1 year following treatment of a Wilms' tumor, about two-thirds of children will have experienced nearly complete compensatory renal growth.

We had anticipated that renal hypertrophy might occur faster in the younger age group. This does not appear to be the case, however; the most rapid changes were noted in the older children. We were not able to define the early postnephrectomy growth rate as most of the patients did not have an intravenous pyelogram until 2 months after surgery.

At diagnosis, no patient had a renal length on the normal side below the mean value for age as described by Hodson *et al.*[5] Fifty-three per cent (11 of 21) of the patients had renal lengths near the mean, while an additional 38 per cent (8 of 21) were within 1 standard deviation of the mean. Nine per cent (2 of 21) patients had a renal length within 2 standard deviations of the mean. On the final pyelogram, all but 1 of our patients had renal lengths greater than 1 standard deviation, and often greater than 2 standard deviations, beyond the normal values as defined by Hodson *et al.*[5] The 1 exception also had a renal length exceeding normal for his age.

Bilateral Wilms' tumor occurs in 4.1 per cent to 13 per cent of all Wilms' tumor cases.[2,14] Improved survival of this group is reflected in the more aggressive treatment of the primary as well as metastatic lesions. The number of survivors has been increased by the current therapeutic regimen and with aggressive treatment of sites of pulmonary and hepatic metastasis.[4,13]

Eight selected cases of bilateral Wilms' tumor patients having unilateral nephrectomy, followed by irradiation and chemotherapy, were reviewed and analyzed by our method. These cases were not included in the 21 cases described above. The hypertrophic response pattern of these 8 patients was similar to those with unilateral disease. Therefore, the RRH pattern alone did not enable us to identify the presence of a second Wilms' tumor in the remaining kidney.

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We appreciate Dr. Hooshang Taybi's cooperation in contributing several of the cases analyzed.

REFERENCES

1. Arkless, R. Normal kidney's reaction to intravenous pyelography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 746–749.
2. Cochran, W., and Froggatt, P. Bilateral nephroblastoma in two sisters. *J. Urol.*, 1967, *97*, 216–220.
3. Currarino, G. Roentgenographic estimation of

kidney size in normal individuals with emphasis on children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *93*, 464–466.

4. Delorimier, A. A., Belzer, F. O., Kountz, S. L., and Kushner, J. H. Treatment of bilateral Wilms' tumor. *Am. J. Surg.*, 1971, *122*, 275–281.
5. Hodson, C. J., Drewe, J. A., Karn, M. N., and King, A. Renal size in normal children: radiographic study during life. *A.M.A. Arch. Dis. Child.*, 1962, *37*, 616–622.
6. Jereb, B., Aperia, A., Berg, U., Broberger, O., and Båryd, I. Renal function in long-term survivors after treatment for nephroblastoma. *Acta paediat. scandinav.*, 1973, *62*, 577–584.
7. Laufer, I., and Griscom, N. T. Compensatory renal hypertrophy: absence *in utero* and development in early life. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 464–467.
8. Ludin, H. Radiologic estimation of kidney weight. *Acta radiol.* (Diag.), 1967, *6*, 561–574.
9. Margolis, L. W., Smith, W. B., Wara, W. M., Kushner, J. H., and De Lorimier, A. A. Wilms' tumor: interdisciplinary treatment program with and without dactinomycin. *Cancer*, 1973, *32*, 618–622.
10. Mitus, A., Tefft, M., and Fellers, F. X. Long-term follow-up of renal functions of 108 children who underwent nephrectomy for malignant disease. *Pediatrics*, 1969, *44*, 912–921.
11. Moëll, H. Kidney size and its deviation from normal in acute renal failure: roentgen-diagnostic study. *Acta radiol.*, 1961, Suppl. 206, 5–74.
12. Vaeth, J. M. Remaining kidney in irradiated survivors of Wilms' tumor. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 148–151.
13. Wedemeyer, P. P., White, J. G., Nesbit, M. E., Aust, J. B., Leonard, A. S., D'Angio, G. J., and Krivit, W. Resection of metastases in Wilm's tumor: report of 3 cases cured of pulmonary and hepatic metastases. *Pediatrics*, 1968, *41*, 446–451.
14. Young, D. G., and Williams, D. I. Malignant renal tumours in infancy and childhood. *Brit. J. Hosp. Med.*, 1969, *2*, 740–744; 749–752.

RENAL OSTEODYSTROPHY IN PATIENTS ON HEMODIALYSIS AS REFLECTED IN THE BONY PELVIS*

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ABSTRACT:

Roentgen studies in a group of 131 patients on maintenance hemodialysis reveal renal osteodystrophy in 25 patients (19 per cent). In 24 patients of the latter group (98 per cent), the various manifestations of renal osteodystrophy appear in the bony pelvis. They include: osteomalacia; osteitis fibrosa; osteosclerosis; dialysis osteopenia; and extraosseous calcifications. Some of the changes, *i.e.*, bending of bone, Looser's lines, metaphyseal fractures and dialysis osteopenia, are due, at least in part, to the weight bearing nature of the bony pelvis. These findings are rarely, if ever, seen in the digits or in other non-weight bearing bones.

It is therefore proposed to evaluate the bony pelvis, together with the magnification digital roentgenogram, when a less costly and less time consuming skeletal survey is needed in hemodialized patients.

THE development of renal osteodystrophy in patients on maintenance hemodialysis is not an unusual occurrence. A higher incidence has been noted in those patients compared to non-hemodialized patients with similar chronic renal disease.[4,9] The roentgen manifestations of renal osteodystrophy, namely, osteomalacia, osteitis fibrosa, osteosclerosis, dialysis osteopenia, and soft tissue calcifications, are well documented in the literature.[6,11–13,19] We have previously stressed the less costly and less time consuming value of direct magnification digital roentgenography[4] in evaluating renal osteodystrophy in hemodialyzed patients. The digit, easily accessible for roentgenography, is a non-weight bearing cortical bone which readily shows changes of bone resorption. Changes due to stress, however, are not seen in the digit,[7] and examination of a weight bearing bone is needed to complete an economical and rapid skeletal survey of patients suspected of having or being followed for renal osteodystrophy.

The bony pelvis was chosen as the weight bearing bone, because roentgenograms of the pelvis are readily available for every hemodialyzed patient from urograms, cystograms, or complete skeletal surveys.

The purpose of this communication is to describe the roentgen manifestations of renal osteodystrophy as they appear in the bony pelvis and to review briefly the current pathogenic concepts of skeletal changes in chronic renal insufficiency.

CLINICAL MATERIAL

During the period from January, 1963, through December, 1972, 131 patients with end-stage renal disease of varying etiology entered the program of hemodialysis at our institution.[18] As part of the initial evaluation, all patients had a complete skeletal survey, and most had intravenous or retrograde urography, or both, and cystography. Biopsy of the posterior iliac crest was performed in 45 patients. After the inception

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Partially supported by Otho S. A. Sprague Foundation, National Institutes of Health, Division of Research Resources, R. R. 48 and Training Grant AMO 05614, and the Kidney Research Fund of Passavant Pavilion, Northwestern Memorial Hospital.

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of maintenance hemodialysis, skeletal survey roentgenography was repeated at intervals of from 6 to 12 months, or more frequently if indicated. The level of serum parathyroid hormone was determined serially in 15 patients observed during the latter part of this period.

Renal osteodystrophy, verified by roentgen examination, bone biopsy, or both, developed during this period in 25 patients (19 per cent of the total). In 24 patients of the latter group (96 per cent), the manifestations of renal osteodystrophy were reflected in the bony pelvis. The specific findings are given in Table 1.

ROENTGEN MANIFESTATIONS

Because of the weight bearing function of the pelvis and surrounding structures, the roentgen examination of the pelvis may reveal: Looser's lines, coxa vara deformities, Otto pelvis deformities, metaphyseal fractures, widening of the growth plates, and frank fractures.

OSTEOMALACIA

Decreased mineralization of osteoid is the fundamental disorder underlying osteomalacia.[2,15] In chronic renal failure, intestinal absorption of calcium is impaired, and there are hypocalcemia, hyperphosphatemia, and secondary hyperparathyroidism.[15,21] "Resistance" to the biological action of vitamin D on the intestinal mucosa comes about, with further decline of calcium absorption. Consequently, the ionized plasma pool of calcium is even more decreased, causing additional stimulation of parathyroid hormone secretion. It has been stated[2] that the vitamin D resistance of uremia may also explain the depression of bone formation and apposition rate and the delay in the maturation of organic bone matrix.

Table I

RENAL OSTEODYSTROPHY MANIFESTATIONS IN PELVIC ROENTGENOGRAMS OF 24 PATIENTS

Roentgen Findings*	No. of Patients	Percentage
Advanced osteomalacia	9	37.5
Coxa vara	2	8.3
Otto pelvis	2	8.3
Wide growth plates	2	8.3
Osteitis fibrosa	11	45.8
Metaphyseal fracture	1	4.1
Osteolysis at insertion of muscles and tendons	6	25
Wide joints	4	16.6
Osteosclerosis	4	16.6
Extraosseous calcifications	9	37.5
Arterial calcifications	7	29.1
Periarticular calcifications	2	8.3
Dialysis osteopenia	1	4.1
Avascular necrosis†	4	16.6

* In most instances, there was more than 1 finding on each pelvic roentgenogram.
† See explanations in text.

The nature of vitamin D resistance in uremia is yet to be fully elucidated. Many studies have shown that the patient with uremia requires a greater intake of vitamin D than the normal individual, and that the metabolism of vitamin D is altered in uremia.[15,21] It has been established that the loss of kidney cells interferes with the conversion of vitamin D to its active metabolite, lack of which is ultimately responsible for the defect in calcium absorption.[5] The dietary intake of calcium may also be inadequate. Additionally, if the calcium content of the dialysate is below 3 mg. per cent, the plasma pool of ionized calcium can be further decreased.[9] Hyperphosphatemia, the result of inability of the diseased kidney to excrete dietary phosphorus adequately, may also contribute to hypocalcemia.

Osteomalacia in the adult will appear in the pelvis as decreased bone density, Looser's lines, and bending of bone. The decreased bone density can best be seen in the change in the clarity of the trabeculae in the pelvis and proximal femurs (Fig. 1). There is osteoclastic osteolysis of both the secondary trabeculae which run perpendicular or oblique to major trabeculae, and also the major trabeculae, which run parallel to the lines of stress.[7] The osteoblasts attempt to reform the trabeculae by producing osteoid. However, because of poor

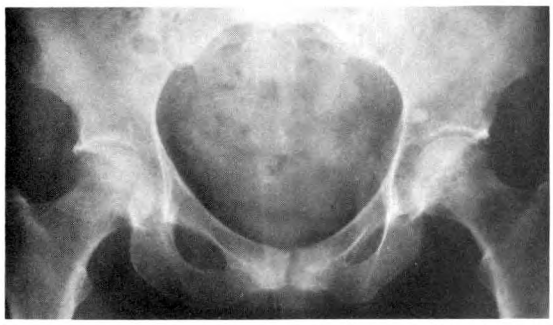

Fig. 1. Pelvis of a 31 year old woman with chronic glomerulonephritis and roentgen signs of osteomalacia. The trabeculae in the femoral head and in the area above the acetabulum are indistinct.

mineralization, excessive osteoid develops. This gives a hazy appearance to the remaining trabeculae.

Looser's lines are thin, radiolucent lines perpendicular to the cortex at sites of stress, and composed of woven bone (nonlamellar bone formed by the abnormal collagen).[19] They are seen in the ischial and pubic rami, the medial margins of the femoral neck and shaft, and the lesser trochanter.[11,16]

The poorly mineralized osteoid allows the bones surrounding the pelvis to bend, leading to development of coxa vara (Fig. 2) and Otto pelvis[6] (Fig. 3; and 4).

Fig. 2. Pelvis of a 25 year old man with familial vitamin D resistant rickets on hemodialysis after 2 unsuccessful renal transplants. Bilateral coxa vara has developed as a result of osteomalacia. Note the increase in thickness of the cortex on the medial aspect of the femoral neck, the result of realignment after bending had occurred. The bony trabeculae are covered by "ground glass" osteoid.

Decreased bone density, Looser's lines, and bending of bone also occur in rickets; however, in this condition there are changes about the unfused epiphysis and metaphysis. Widening of the zone of provisional ossification between epiphysis and metaphysis by more than 1 mm. is the earliest sign of rickets. Later, cupping and fraying of the metaphysis are seen.[19,20] The unfused femoral epiphysis allows one to observe these changes (Fig. 3).

OSTEITIS FIBROSA

Hypocalcemia, hyperphosphatemia, and vitamin D resistance associated with renal failure ultimately result in secondary hyperparathyroidism. The increase in serum parathyroid hormone level causes increased osteocytic and osteoclastic bone reabsorption, with resultant osteolysis. This occurs at the endosteal, Haversian, and subperiosteal surfaces.

Reabsorptive activity is seen roentgenographically when resorption cavities are filled with woven bone or fibrous tissue (*e.g.*, osteitis fibrosa).[11] Subperiosteal reabsorption is caused by osteocytes located in the lacunae and canaliculi of cortical

Fig. 3. Pelvis of a 16 year old boy with hereditary interstitial nephritis and unfused epiphyses. There is bilateral protrusion of the acetabulum (Otto pelvis). The zones of provisional ossification in the unfused femoral epiphyses, the cartilage of "Y," and the sacroiliac joints are wide.

bone.[15] Endosteal and Haversian osteolysis is also caused by osteoclasts. Subperiosteal reabsorption is more readily identified roentgenologically because of greater osteocytic *versus* osteoclastic activity.

Roentgenograms of the pelvis can permit identification of osteolysis in areas of increased remodelling. Metaphyseal fractures are caused by osteolysis in these areas.[10] This explains the increased incidence of metaphyseal hip fractures in patients with renal osteodystrophy (Fig. 4).

There also is increased osteolysis at sites of stress from tendon insertions and joints, such as the sacroiliac joint and the symphysis pubis (Fig. 5). Presumably, these sites of stress develop electrical potential differences at their surfaces, and osteolysis occurs at the surface with the positive charge.[8] The "widened" joint spaces have been shown to be a combination of poorly mineralized woven bone and fibrous tissue which has replaced the lamellar bone in the subchondral region.[11]

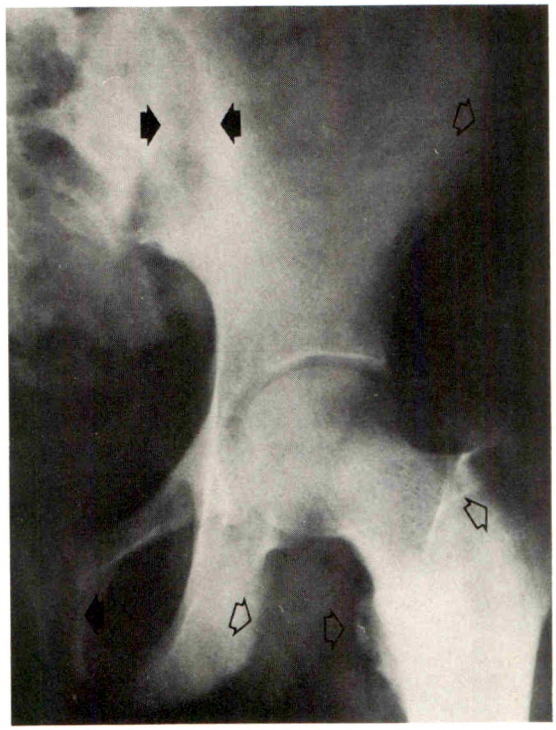

Fig. 5. Pelvis of a 50 year old woman with chronic pyelonephritis. There are signs of osteolysis in the anterior superior iliac spines, ischial tuberosities, and trochanters (open arrows), the sites of insertion of muscles. The osteolysis is identified as subperiosteal resorption. The wide symphysis pubis and sacroiliac joints (black arrows) are the result of osteolysis and replacement by woven bone and fibrous tissue (osteitis fibrosa).

OSTEOSCLEROSIS

Osteosclerosis will appear in the pelvis as an area of increase in bone density. The disorder is thought to develop because of

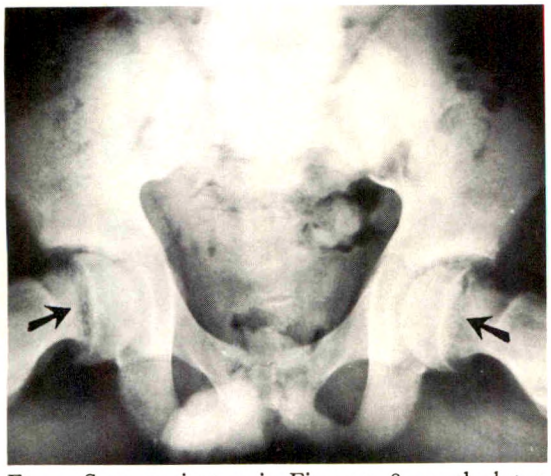

Fig. 4. Same patient as in Figure 3, 8 months later. There are fractures of both femoral metaphyses (arrows), resulting in slippage of the epiphyses. The fractures are a direct result of osteolysis occurring in areas of increased remodelling of bone.

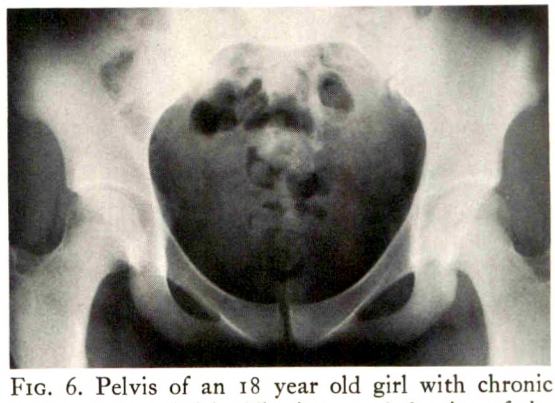

Fig. 6. Pelvis of an 18 year old girl with chronic glomerulonephritis. The increased density of the bony pelvis and femurs is characteristic of osteosclerosis, representing excessive but poorly mineralized osteoid.

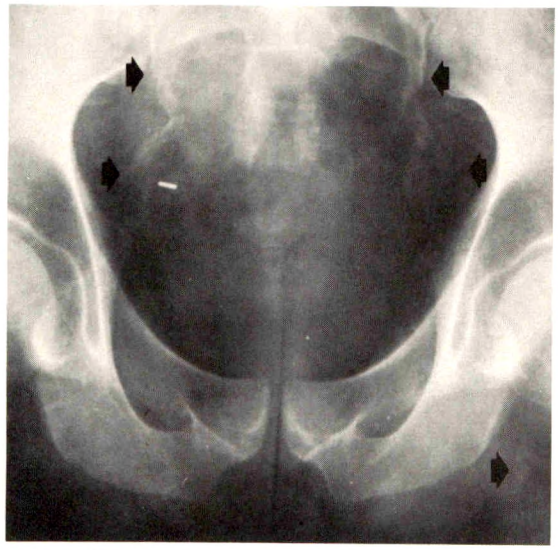

FIG. 7. Pelvis of a 33 year old man with medullary cystic renal disease, who developed, while on hemodialysis, profuse arterial calcifications (a sign of Mönckeberg's arteriosclerosis). The iliac and femoral arteries are calcified (arrows). Note clips from present working transplanted kidney. The transplant has not reversed the calcification.

the direct effect of increased parathyroid hormone secretion on osteoblasts to enhance osteoid production, while there is only partial mineralization of the osteoid due to osteomalacia.[17] Thus, even though there is only partial mineralization of osteoid, there is such an excessive amount of osteoid that the bone appears dense (Fig. 6).

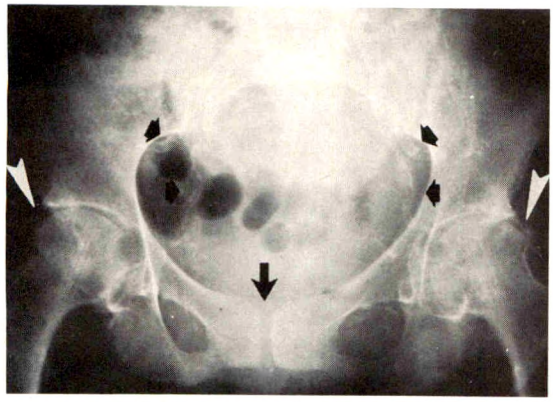

FIG. 8. Pelvis of a 56 year old woman with nephrosclerosis. In addition to calcified arteries (black arrows), there are calcifications in the symphysis pubis (vertical black arrow), indicating chondrocalcinosis. Note also periarticular calcifications of the hips (white arrows).

EXTRAOSSEOUS CALCIFICATIONS

Extraosseous calcifications are more likely to occur when the product of the plasma calcium (in mg./100 ml.) and plasma phosphorus (in mg./100 ml.) is greater than 75.[7,20] In renal osteodystrophy the product increases chiefly because the level of plasma phosphorus is elevated, due to inability of the diseased kidneys to excrete ingested dietary phosphorus. Hypercalcemia, secondary to "tertiary" ("autonomous") hyperparathyroidism or to excessive vitamin D administration, may also contribute to an elevated calcium-phosphorus product. Extraosseous calcifications in the bony pelvis are most commonly seen as calcifications of the internal and external iliac arteries and superficial femoral arteries (Fig. 7; and 8). The calcifications are located in the tunica media, rather than in the intima as in arteriosclerosis.[14]

Periarticular calcifications can also be

FIG. 9. Tomogram of the left hip of an 18 year old girl with medullary cystic renal disease, who while on maintenance hemodialysis developed an undisplaced fracture of the femoral neck characteristic of dialysis osteopenia.

observed around the hip joints and the symphysis pubis. They present as chrondrocalcinosis or tumoral calcinosis[14] (Fig. 8).

DIALYSIS OSTEOPENIA

Recently a new presentation of bone disease in renal osteodystrophy has been described—dialysis osteopenia.[13] It is characterized primarily by a progressive reduction in bone mass (*i.e.*, osteoporosis), but changes of osteomalacia and osteitis fibrosa are also present. Its roentgenographic manifestation is that of stress fractures which heal slowly, although with abundant callus (Fig. 9). In addition to the femoral neck, they can occur in the pubic and ischial rami.

AVASCULAR NECROSIS

Avascular necrosis is seen fairly frequently in renal transplant recipients, as well as in other patients receiving high doses of steroids. Avascular necrosis may occur in the course of maintenance hemodialysis without any demonstrable underlying etiological factor. Twenty-three patients with avascular necrosis were seen in a series of 350 patients on maintenance hemodialysis.[3] In our series of 131 patients on hemodialysis, we did not observe avascular necrosis occurring in the femoral head, except after renal transplantation and high doses of steroids (Fig. 10).

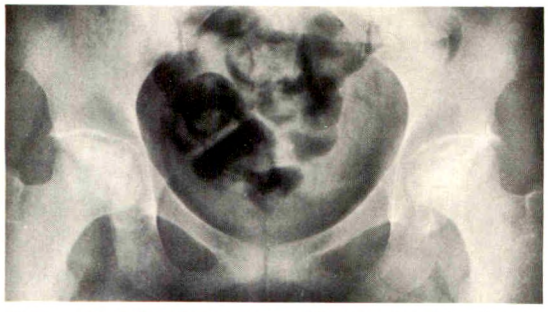

Fig. 10. Pelvis of a 22 year old woman with chronic glomerulonephritis who had a transplant 2 years earlier. The transplanted kidney was rejected 2 months later following heavy doses of corticosteroids. While on hemodialysis she developed bilateral aseptic necrosis of the hips. Note the fragmentation, flattening, and sclerotic changes of the femoral heads.

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REFERENCES

1. Agus, Z. S., and Goldberg, M. Pathogenesis of uremic osteodystrophy. *Radiol. Clin. North America*, 1972, *10*, 545–556.
2. Avioli, L. V. Vitamin D metabolism in uremia. *Kidney*, 1975, *8*, 1–6.
3. Bailey, G. L., Griffiths, H. J., Mocelin, A. J., Gundy, D. H., Hampers, C. L., and Merrill, J. P. Avascular necrosis of femoral head in patients of chronic dialysis. *Tr. Am. Soc. Artif. Int. Organs*, 1972, *18*, 401–404.
4. Calenoff, L., and Norfray, J. Magnification digital roentgenography: method for evaluating renal osteodystrophy in hemodialyzed patients. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *118*, 282–292.
5. DeLuca, H. F. Kidney as endocrine organ involved in calcium homeostasis. *Kidney Internat.*, 1973, *4*, 80–88.
6. Eastwood, J. B., Bordier, P. J., and De Wardener, H. E. Some biochemical, histological, radiological and clinical features of renal osteodystrophy. *Kidney Internat.*, 1973, *4*, 128–140.
7. Feist, J. H. Biological basis of radiologic findings in bone disease: recognition and interpretation of abnormal bone architecture. *Radiol. Clin. North America*, 1970, *8*, 183–214.
8. Frost, H. M. The Bone Dynamics in Osteoporosis and Osteomalacia. Charles C Thomas, Publisher, Springfield, Ill., 1966, p. 121.
9. Goldsmith, R. S., and Johnson, W. J. Role of phosphate depletion and high dialysate calcium in controlling dialytic renal osteodystrophy. *Kidney Internat.*, 1973, *4*, 154–160.
10. Kirkwood, J. R., Ozonoff, M. B., and Steinbach, H. L. Epiphyseal displacement after metaphyseal fracture in renal osteodystrophy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 547–554.
11. Krempien, E. R., Mehls, O., and Malluch, H. Skeletal abnormalities in chronic renal insufficiency before and during maintenance hemodialysis. *Kidney Internat.*, 1973, *4*, 116–127.
12. Meema, H. E., Rabinovich, S., Meema, S., Lloyd, G. J., and Oreopoulos, D. G. Improved radiological diagnosis of azotemic osteodystrophy. *Radiology*, 1972, *102*, 1–10.

13. Parfitt, A. M., Massry, S. G., and Winfield, A. C. Osteopenia and fractures occurring during maintenance hemodialysis. *Clin. Orthopedics*, 1972, *87*, 287–302.
14. Parfitt, A. M. Soft tissue calcifications in uremia. *A.M.A. Arch. Int. Med.*, 1969, *124*, 544–556.
15. Rasmussen, H., and Bordier, P. The Physiological and Cellular Basis of Metabolic Bone Disease. Williams & Wilkins Company, Baltimore, 1974, pp. 19–51; 144–155.
16. Reynolds, W. A., and Karo, J. J. Radiologic diagnosis of metabolic bone disease. *Orthop. Clin. North America*, 1972, *3*, 521–543.
17. Robinson, R. A. Ultrastructural appearance of bone cells and bone matrix in renal osteodystrophy. *A.M.A. Arch. Int. Med.*, 1969, *124*, 519–529.
18. Roguska, J., Simon, N. M., del Greco, F., and Krumlovsky, F. A. Ten year experience with maintenance hemodialysis for chronic uremia. *Tr. Am. Soc. Artif. Int. Organs*, 1974, *20*, 597–582.
19. Shapiro, R. Radiologic aspects of renal osteodystrophy. *Radiol. Clin. North America*, 1972, *10*, 557–568.
20. Smith, R. Pathophysiology and management of rickets. *Orthop. Clin. North America*, 1972, *3*, 601–621.
21. Stanbury, S. W. Bone disease in uremia. *Am. J. Med.*, 1968, *44*, 714–724.

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CROHN'S DISEASE OF THE ESOPHAGUS*

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ABSTRACT:

Two cases of Crohn's disease involving the esophagus are described. Both patients had Crohn's disease elsewhere. Multiple intramural fistulous tracts are seen in both patients, and this is a characteristic feature of Crohn's disease. One patient developed a spontaneous esophago-bronchial fistula. Even though the esophageal involvement of Crohn's disease is rare, it should be suspected when a chronic esophageal inflammatory lesion develops in a patient who has Crohn's disease elsewhere, especially without hiatus hernia or other chronic disease.

CROHN'S disease is a chronic inflammatory disorder of unknown etiology and may involve any part of the intestinal tract but typically involves the terminal ileum.[11] Farther away from the terminal ileum, the intestinal tract becomes less frequently involved by Crohn's disease. Recently, an increasing number of cases of Crohn's disease involving the gastroduodenal area has been reported suggesting that involvement of the upper gastrointestinal tract may be more common than generally believed.[2,13] However, there have been only a few reports of Crohn's disease involving the esophagus. The purpose of this communication is to report 2 cases of Crohn's disease of the esophagus which demonstrate its characteristic roentgenographic features.

REPORT OF CASES

CASE 1. (E.B.) This 34 year old white male was admitted to the Graduate Hospital of the University of Pennsylvania on February 2, 1971, because of cough, fever and chest pain of about 2 weeks' duration. The patient had been suffering from ileocolitis including anal and perianal complications for 17 years and had undergone 4 resections of involved small and large bowel (Fig. 1). He had been on steroids for a long period. In June, 1970, about 7 months prior to the current admission, he developed mild dysphagia, and an esophagogram demonstrated irregular, somewhat nodular thick mucosal folds mainly in the mid-esophagus (Fig. 2).

On admission a chest roentgenogram and an esophagogram showed an esophago-bronchial

FIG. 1. Case 1. A small bowel study demonstrates marked shortening of the small bowel and anastomosis of the small bowel to the ascending colon from multiple previous operations. Recurrent Crohn's disease is obvious in the distal small bowel and proximal ascending colon.

* From the Graduate Hospital and the Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

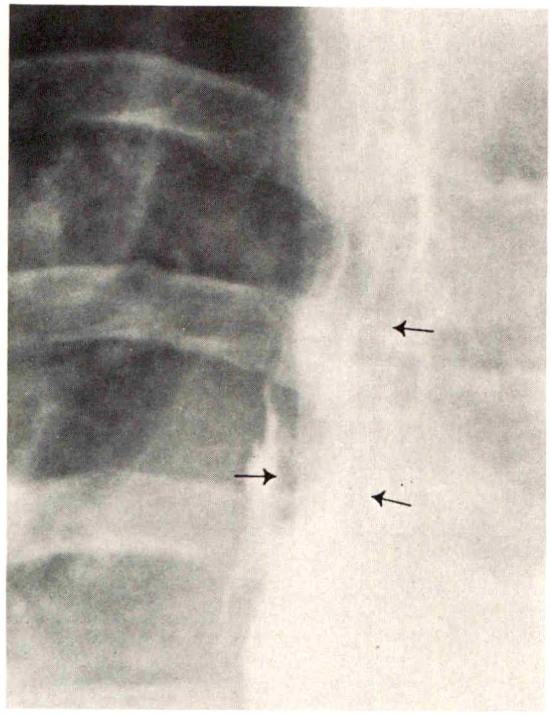

FIG. 2. Case 1. An esophagogram in June, 1970, demonstrates irregular, thick mucosal folds (arrows) in the mid-esophagus with a suggestion of nodularity.

fistula and a lung abscess. An esophagoscopic examination confirmed esophagitis and the fistula. One throat culture revealed monilia. The patient was placed on penicillin, intravenous hydrocortisone and mycostatin oral suspension. The patient gradually improved and was discharged from the hospital on March 5, 1971.

One month later, the patient was re-admitted for coughing spells and fever. An esophagogram demonstrated multiple transverse fistulous tracts at the right lateral aspect of the mid-esophagus as well as a longitudinal fistula parallel to the esophageal wall (Fig. 3, *A* and *B*). He was discharged after about 5 weeks medical treatment consisting of hyperalimentation, steroids, penicillin, and mycostatin. However, the patient was re-admitted in July, 1972, with the complaints of choking sensation and cough upon ingestion of liquid. A chest roentgenogram and an esophagogram demonstrated a recurrent esophago-bronchial fistula (Fig. 4). The patient expired 6 weeks after admission.

At autopsy, there were multiple fistulous tracts in the markedly thickened wall of the mid-esophagus, some of them penetrating into the mediastinum and lung. The microscopic examination of the esophagus, as well as the small and large bowel, showed transmural chronic inflammatory changes representing Crohn's disease without granuloma.

CASE II. (A.A.) This 56 year old Black female was seen at the Hospital of the University of Pennsylvania complaining of dysphagia of 6 months' duration. During a recent admission to another hospital, a barium swallow examination demonstrated the suggestion of tumor in the distal esophagus but biopsy revealed no tumor. Resection of the rectum and part of the colon had been carried out in the past because of multiple fistulae including a recto-vaginal fistula due to Crohn's disease.

Physical examination on admission was unremarkable except for the presence of a permanent colostomy, evidence of prior abdominoperineal surgery, and remnants of perianal inflammation and fistulous tract.

A barium swallow examination (Fig. 5) and cine esophagogram (Fig. 6) showed irregular narrowing of the distal esophagus and multiple transverse and longitudinal intramural fistulous tracts parallel to the esophageal wall. An esophagoscopy showed mucosal irregularity and a benign appearing stricture in the distal esophagus. There was no hiatus hernia. The remainder of the upper gastrointestinal tract and small bowel was unremarkable. The barium enema examination through the colostomy was also within normal limits.

The patient underwent resection of the stenotic distal esophagus. The surgical specimen showed small mucosal ulcerations and an inflammatory mass centered around intramural fistulae. Other sinus tracts were also found which had dissected into and through the thickened muscle layer. The histological examination showed transmural chronic inflammation characterized by aggregates of lymphocytes, plasma cells and also non-caseating granulomas. There were tremendous hypertrophy of the tunica muscularis, fibrosis in the layers of the esophageal wall, and multiple sinus tracts. The histological appearance of the esophagus was similar to that of the previously resected colon and rectum which were typical for Crohn's disease.

DISCUSSION

The involvement of the esophagus by

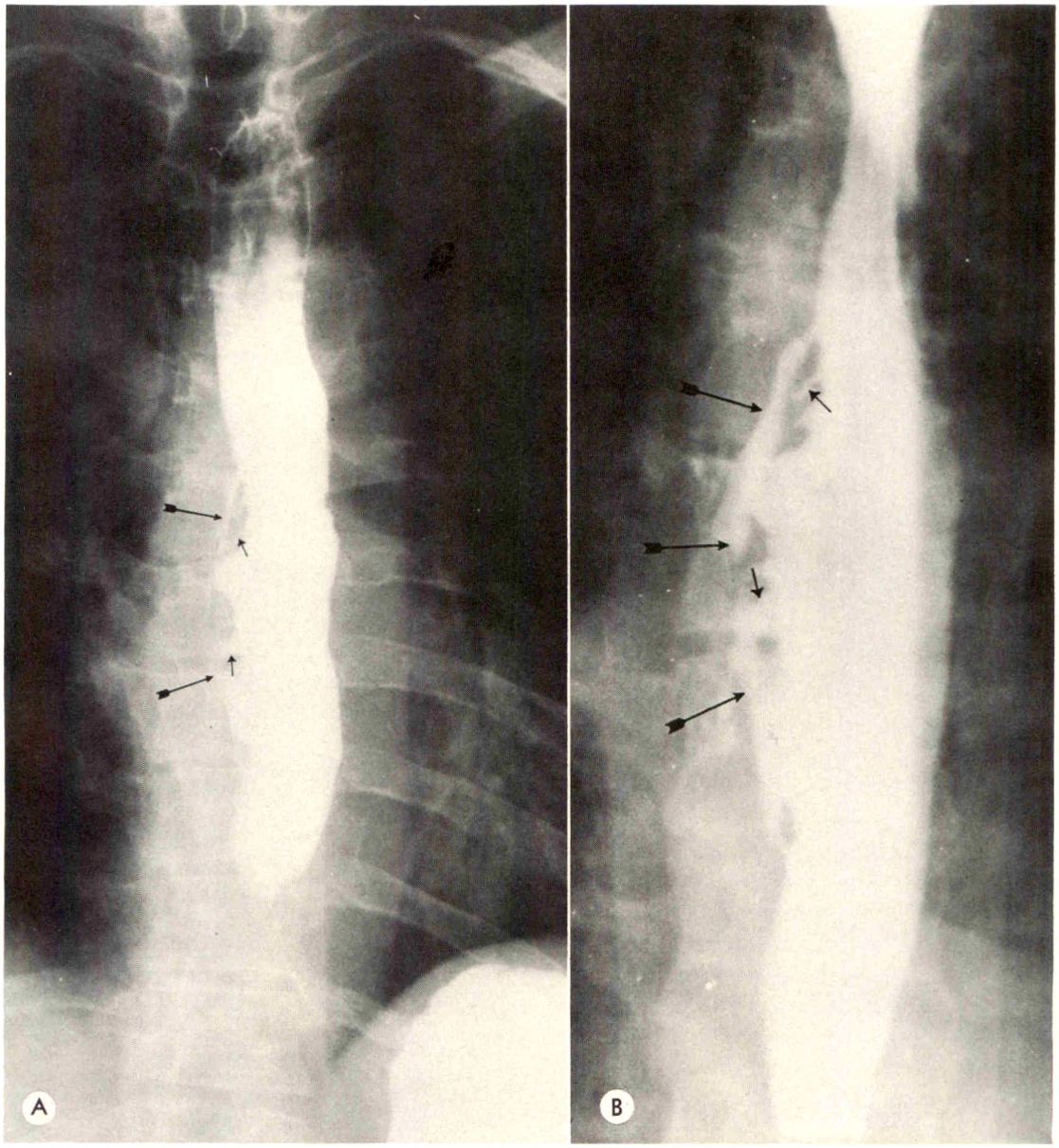

Fig. 3. Case 1. (*A*) An esophagogram in May, 1971, shows a longitudinal fistulous tract along the wall of the mid-esophagus (long arrows) and multiple transverse fistulous tracts (short arrows). No hiatus hernia is present. (*B*) A close-up of a spot film of the mid-esophagus demonstrates the longitudinal (long arrows) and transverse (short arrows) intramural fistulous tracts.

Crohn's disease is rare. Marshak[11] stated that he had not identified Crohn's disease of the esophagus with his personal experience of 8,000 cases of regional enteritis and 4,000 of granulomatous colitis. However, there have been a few cases of esophageal involvement of Crohn's disease in the literature recently.[1,2,4–6,8]

A thickened mucosal fold in the distal esophagus,[4] irregular stricture of the lower esophagus,[7] ulcer in the mid and lower esophagus,[3,8] rigid deformity of the distal esophagus,[4] and a polypoid mass in the lower esophagus[5] have been reported in patients who had evidence of Crohn's disease elsewhere. A narrowing of the distal esoph-

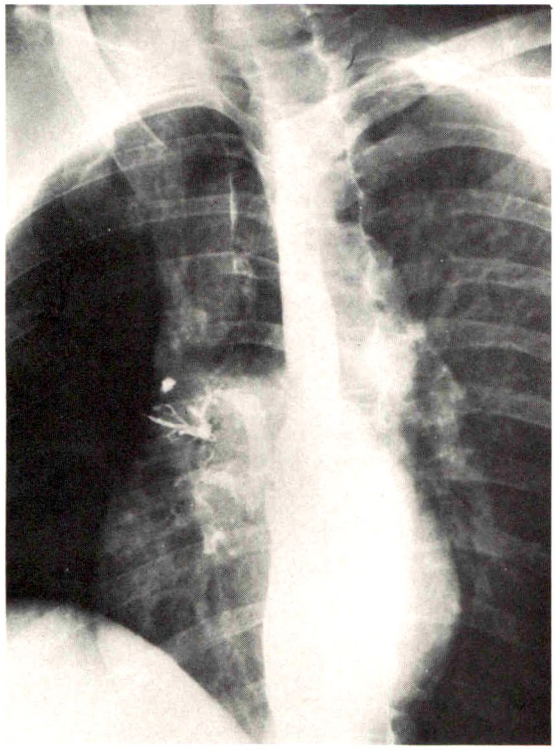

FIG. 4. Case I. There is a recurrent spontaneous esophago-bronchial fistula seen on an esophagogram in July, 1972.

agus with hiatus hernia in a patient who had ileitis is also reported.[1] LiVolsi and Jaretzki[9] and Madden *et al.*[10] reported cases of isolated esophagitis of the distal esophagus with hiatus hernia without Crohn's disease elsewhere, but with pathological findings suggesting Crohn's disease. Also ulcerative esophagitis is reported in patients with chronic ulcerative colitis.[6,12]

The characteristic histological feature of Crohn's disease is transmural chronic inflammatory change with granulomas. Granulomas are found in approximately 90 per cent of cases, but in less than 50 per cent of those patients who are treated with steroids.[11] Granulomas are not essential for pathological diagnosis of Crohn's disease.

One of our cases (Case I) did not have granulomas in the autopsy specimen. The patient had a long history of documented Crohn's disease involving the small and large bowel for which he had been treated with steroids, and he had had multiple operations for various complications. The esophageal involvement in this patient occurred after a long-standing ileocolitis. The clinical and roentgenographic course of the esophageal involvement consisted of remissions and exacerbations of about $2\frac{1}{2}$ years' duration until the patient expired. Initially, the esophagus showed irregular thick mucosal folds; subsequently a spontaneous esophago-bronchial fistula developed about

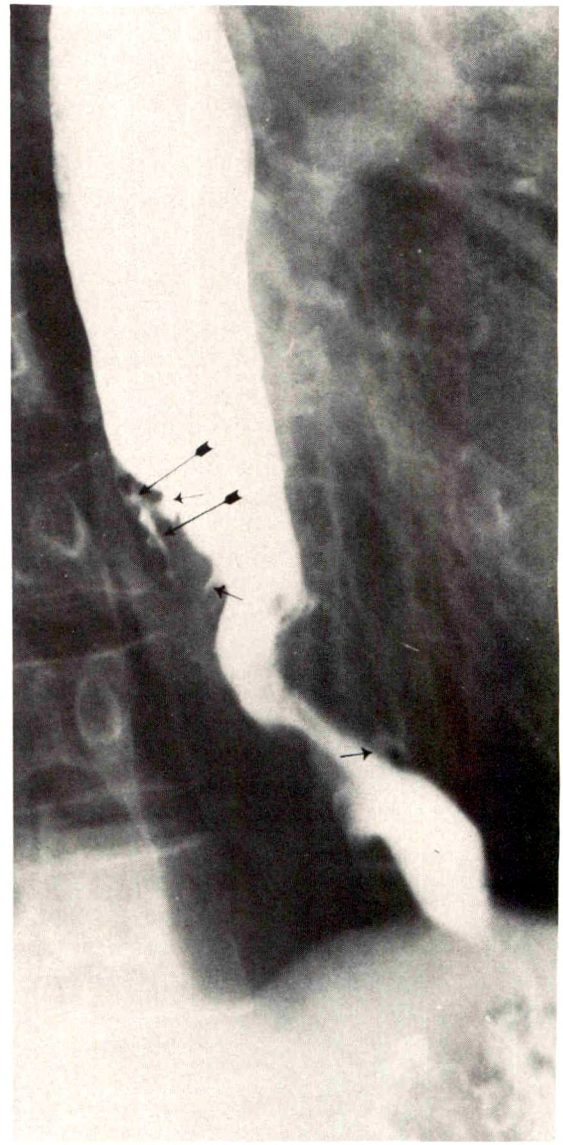

FIG. 5. Case II. There are multiple transverse (short arrows) and longitudinal (long arrows) intramural fistulous tracts in the irregular and narrowed distal esophagus. No hiatus hernia is present.

7 months later. Also, he developed multiple intramural fistulous tracts including a longitudinal fistulous tract along the esophageal wall. The presence of monilia in one throat culture is somewhat confusing, but monilia is a frequent superinfection, and, furthermore, spontaneous esophago-bronchial fistula and multiple fistulous tracts are not a feature of esophageal moniliasis. Also there was no significant response of the esophageal lesion to the vigorous treatment with an oral antifungal agent, and this is not the usual response of esophageal moniliasis.

Case II did not have a hiatus hernia and showed typical histological features of Crohn's disease in the surgical specimen of the esophagus and the rectosigmoid.

One of the characteristic features of Crohn's disease is a spontaneous fistula to an adjacent organ, and Case I had a spontaneous esophago-bronchial fistula. The intramural fistulous tract, especially a longitudinal tract parallel to the wall, is described in both Crohn's disease and diverticulitis of the colon, but is much more common in Crohn's disease.[11] Both of our cases showed rather typical intramural transverse and longitudinal tracts. No esophageal diverticula are seen in the esophagogram or in the pathological specimen. It is interesting to note that the stomach and the duodenum were spared by Crohn's disease in both cases.

Chronic esophagitis of the distal end of the esophagus would be difficult to differentiate from reflux esophagitis in the presence of a hiatus hernia. Tuberculosis and chronic fungus infection may produce a broncho-esophageal fistula but there would be other evidence of tuberculosis or fungus infection roentgenographically, clinically and microscopically. Esophageal moniliasis may simulate an early stage of Crohn's disease of the esophagus, but moniliasis is a diffuse esophagitis rather than a localized or segmental process as is Crohn's disease. Furthermore, the roentgenological and clinical follow-up examinations will establish the diagnosis. Intramural diverticulosis of the esophagus may resemble multiple transverse fistulous tracts, but the longitudinal fistula and the spontaneous esophago-bronchial fistula are not seen with intramural diverticulosis of the esophagus.

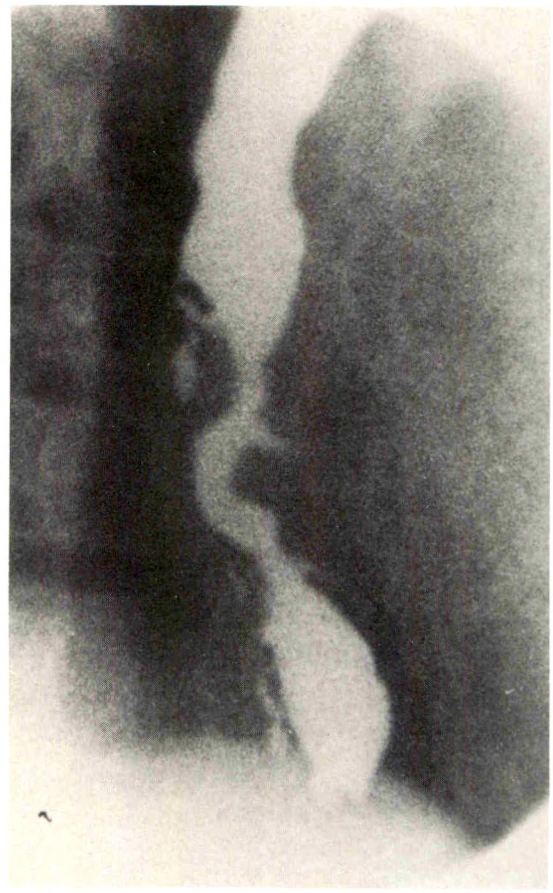

Fig. 6. Case II. A cine spot film demonstrates the multiple fistulous tracts.

The possibility of Crohn's disease of the esophagus should be suspected when chronic esophagitis develops in a patient who has Crohn's disease elsewhere. The diagnosis should be based on a combination of clinical, roentgenographic and pathological evidence.

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REFERENCES

1. Bagby, R. C., Rogers, J. V., and Hobbs, C. Crohn's disease of esophagus, stomach and duodenum: review with emphasis on radiographic findings. *South. M. J.*, 1972, *65*, 515–523.
2. Farman, J., Faegenburg, D., Dallemand, S., and Chen, C. K. Crohn's disease of stomach: "Ram's Horn" sign. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1975, *123*, 242–251.
3. Gelfand, M. D., and Krone, C. L. Dysphagia and esophageal ulceration in Crohn's disease. *Gastroenterology*, 1968, *55*, 510–514.
4. Haggitt, R. C., and Meissner, W. A. Crohn's disease of upper gastrointestinal tract. *Am. J. Clin. Path.*, 1973, *59*, 613–622.
5. Heffernon, E. W., and Kepkay, P. H. Segmental esophagitis, gastritis and enteritis. *Gastroenterology*, 1954, *26*, 83–88.
6. Knudsen, K. B., and Sparberg, M. Ulcerative esophagitis and ulcerative colitis. *J.A.M.A.*, 1967, *201*, 140.
7. Kyer, N. H., Cook, P. L., and Harper, R. A. K. Esophageal stricture associated with Crohn's disease. *Gut*, 1969, *10*, 549–554.
8. Legge, D. A., Carlson, H. C., and Judd, E. S. Roentgenologic features of regional enteritis of upper gastrointestinal tract. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 355–360.
9. LiVolsi, V. A., and Jaretzki, A. Case of Crohn's disease limited to esophagus. *Gastroenterology*, 1973, *64*, 313–319.
10. Madden, J. L., Ravid, J. M., and Haddad, J. R. Regional esophagitis: specific entity simulating Crohn's disease. *Ann. Surg.*, 1969, *170*, 351–367.
11. Marshak, R. H. Granulomatous disease of intestinal tract (Crohn's disease). *Radiology*, 1975, *114*, 3–22.
12. Rosendroff, C. and Grieve, N. W. T. Ulcerative esophagitis in association with ulcerative colitis. *Gut*, 1967, *8*, 344–347.
13. Thompson, W. M., Cockrill, H., Jr., and Rice, R. P. Regional enteritis of duodenum. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1975, *123*, 252–261.

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YEAST BEZOAR FORMATION FOLLOWING GASTRIC SURGERY*

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ABSTRACT:

Yeast bezoar seems to be a relatively common late complication after various gastric operations. The incidence of bezoar depends decisively on the nature of the operation. Vagotomy in particular is conducive to the formation of a bezoar. Vagotomy+Billroth I resection provided the most propitious conditions for the growth of yeast, for every one-half of the patients in this group developed a bezoar.

Yeast bezoars usually appear within a year of the operation. The majority disappear during the first follow-up year, many without any therapy. However, in some cases the bezoar was a rather inconvenient late complication of gastric surgery and one that gave symptoms. It is difficult to draw any definite conclusions concerning the effect of therapy on the disappearance of the bezoar. We used gastric lavage and antimycotics as well as substances that increase gastric acidity. There is still no known method of preventing the formation of yeast bezoars. In the present consensus, a change in the acid conditions and disturbed gastric motility postoperatively are conducive to the formation of a bezoar.

BEZOAR formation in the gastric remnant as a complication of surgical management of gastric disease has been reported in recent years. The most commonly encountered concretions have been phytobezoars composed of the fibers of fruit and vegetables.[4,5,7,9–11] Yeast organisms have also been found to form a bezoar-like mass in the resected stomach. The first yeast bezoars were reported by Borg *et al.* in 1966.[3] Some mention of them has appeared subsequently in the literature,[1,12,13] and the most comprehensive report was published by Jepsen in 1968.[8] The fungus occurs in man generally as a saprophyte. Fungus infections in the mouth, esophagus, lungs, and even in other organs are known to occur in patients in poor condition or suffering from a serious disease.[6,8,13] Reduction in gastric motility and acid secretion after vagotomy and gastric resection probably predispose to the formation of phytobezoars and yeast bezoars.

MATERIAL

The cases were collected from patients seen from 1968–1974 at the Second Surgical Clinic of Meilahti Hospital, University of Helsinki. A yeast bezoar was found in 43 patients. The mean age of the patients was 51; the youngest was 28 and the oldest 78 years old. Fifteen of the patients were women and 28 men.

Forty patients had undergone gastric surgery and the bezoar first developed after the operation. Most operations had been done for either gastric or duodenal ulcer (Table I). The operative procedures are listed in Table II. Vagotomy and a gastric operation had been performed on 30 patients, and resection without vagotomy on 10 patients. Three patients were not operated; their primary diseases were duodenal ulcer, carcinoma of the lung, and diabetes.

The primary diagnosis was based on roentgenologic examinations of the stomach. In 28 (65 per cent) gastroscopy was also performed and was consistent with the roentgenologic finding. The gastric aspirate of 35 patients (82 per cent) was studied by plain microscopy. Massive yeast colonies

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Table I

OPERATIVE INDICATIONS FOR PATIENTS WHO LATER DEVELOPED A YEAST BEZOAR

Operative Indication	No. of Patients
Duodenal ulcer	28
Gastric ulcer	9
Hemorrhagic gastritis	1
Gastric carcinoma	2
No operation	3
Total	43

were detected in all the cases.

INCIDENCE

To elicit the incidence of yeast bezoars after operations of various types the authors made a prospective study of patients operated on during a period in 1970–1971 for the occurrence of bezoars. A total of 21 (11 per cent) yeast bezoars was detected in the composite of 189 patients undergoing gastric surgery. The group that consisted of miscellaneous gastric operations (gastrotomy, fundoplication, etc.) revealed no (0/9) yeast bezoars and the gastrography-duodenography group only 1 (1/35).

Yeast bezoar developed most commonly in the cases that had undergone vagotomy (Table III). Fifty per cent (10/20) of the patients undergoing vagotomy and Billroth I gastric resection had yeast bezoar, whereas none was observed in the vagotomy and pyloroplasty group (0/30). Of the patients subjected to gastric resection without vagotomy, 10 per cent had yeast bezoars.

Table II

NATURE OF THE OPERATION ON PATIENTS WHO LATER DEVELOPED A YEAST BEZOAR

Nature of Operation	No. of Patients
Vagotomy+Billroth I	21
Vagotomy+Billroth II	6
Vagotomy+pyloroplasty	2
Vagotomy+gastroenterostomy	1
Billroth I resection	4
Billroth II resection	6
No operation	3
Total	43

CLINICAL PICTURE

Twenty-four of the patients with a yeast bezoar displayed significant symptoms and 11, mild symptoms. Six patients were completely asymptomatic. The most common complaints were abdominal pain, vomiting, and a feeling of fullness (Table IV). It is of course important in considering the symptoms to remember that the patients were operated cases, which makes it impossible to decide with certainty which of the complaints were postoperative and which caused by the yeast bezoars.

The disease picture was characterized in some cases by periodicity, probably attributable to the variation in the size of the bezoar. The patients reported epigastric fullness especially when eating a meal. The large bezoar mass may break up in the process of vomiting and the patients may then be able to eject all or a part of the mass. A gastric lavage tube also easily breaks up the relatively fragile phytobezoar mass which may then pass into the intestine. Reduction in the size of the bezoar is often followed by an asymptomatic period until the colonies of fungi again grow to a disturbing size.

In addition to direct microscopy, each gastric aspirate was also cultivated for fungi. The most common species of fungi in the material were *Candida albicans* and *Torylopsis glabrata*. Beside these, 8 species of fungi were discovered: *Candida tropicalis*, *Candida krusei*, *Geotrichum candidum*, *Saccharomyces cerevisiae*, *Saccharomyces telluris*, *Kluyveromyces fragilla*, *Aspergillus fumigatus*, and the *Cephalosporium* species. Four different fungi were encountered in the course of 4 years in the stomach of 1 patient.

ROENTGENOLOGIC STUDIES

With a few exceptions, the presence of a yeast bezoar was established at the first postoperative roentgenologic examination

Table III

INCIDENCE OF YEAST BEZOARS IN CONNECTION WITH OPERATIONS OF DIFFERENT TYPES

Nature of Operation	No. of Operations	Incidence of Bezoars
Operations combined with Vagotomy		
Vagotomy+pyloroplasty	30	—
Vagotomy+Billroth I resection	20	10
Vagotomy+Billroth II resection	9	3
Vagotomy+gastroenterostomy	2	—
Total	61	13–21 per cent
Resections without Vagotomy		
Billroth I resection	30	4
Billroth II resection	39	3
Resection+Roux-Y	1	—
Total	70	7–10 per cent

which was made an average of 6 months after the operation.

Twenty-five (63 per cent) of the bezoars were detected within 1 year of the operation, 9 between 1 and 3 years, and 6 more than 3 years postoperatively. The latest bezoar was found 27 years after a Billroth II resection.

The bezoar was diagnosed in 17 cases after a single roentgenologic examination; the number of positive examinations ranged from 2 to 9 in the other cases. Roentgenologic examinations positive for the presence of a bezoar averaged 2.7 per patient. It was possible to evaluate bezoar-induced roentgenologic changes in 107 gastric examinations.

Thirty-seven patients were followed roentgenologically for an average of 22 months (range 6 to 48 months). Six patients were not followed at all. The bezoar disappeared in 28 cases during the follow-up and was still visible at its end in 9 cases. Eighteen bezoars disappeared during the first follow-up year and there were only 3 cases in which recovery occurred after 2 years (Table v). The patients whose bezoar did not disappear were followed-up for 22 months on average (range 6 to 48 months).

The material included 5 recurring yeast bezoars; 1 of the cases had several recidivations despite treatment for 4 years. The average duration of the recurrences was 2 years.

A typical roentgenologic appearance was a large, mottled filling defect which moved with a change in the patient's position (Fig. 5). In the erect roentgenogram the bezoar floated in the barium fluid, and the upper part of the bezoar projected from the contrast medium into the air bubble of the fundus (Fig. 1). With the patient supine the bezoar moved into the fundus and the roentgenogram showed that its upper surface adjoined the contour of the fundus (Fig. 2). The upper surface of the bezoar was distinctly curved in 90 per cent of the cases. There was not a single case of bezoar adherent to the wall, for contrast medium surrounded the bezoar completely. The yeast bezoars were generally large, an average of 7 cm. in diameter. Several large bezoars enlarged the gastric remnant giving it a balloon-like appearance (Fig. 3*A*).

Table IV

SUBJECTIVE COMPLAINTS OF PATIENTS WITH A BEZOAR

Symptoms	No. of Patients
Abdominal pain	12
Vomiting	10
Diarrhea	4
Feeling of fullness, eructations or flatulence	11
No symptoms	6
Total	43

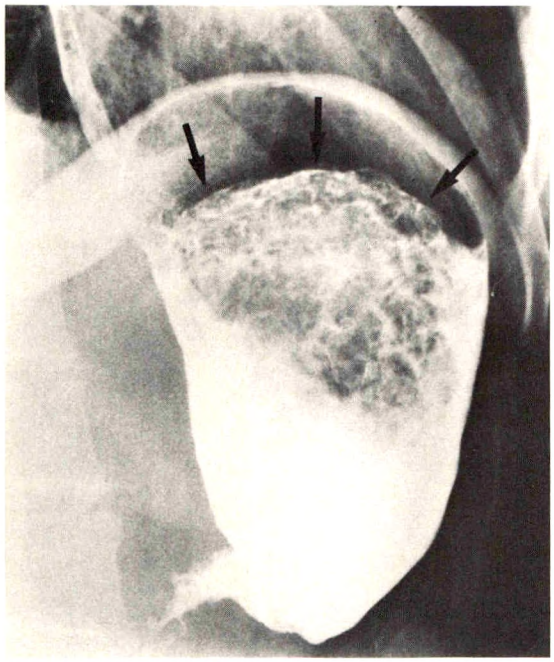

Fig. 1. A large yeast bezoar floating in contrast medium in the gastric remnant after a Billroth I resection. The upper curved surface of the bezoar is seen in the fundal air bubble (arrows). Emptying of the stomach is unobstructed.

There were some cases in which the bezoar filled the gastric remnant almost in its entirety and contrast medium was able to pass like a covering between the bezoar and the wall of the gastric remnant.

Numerous separate filling defects were seen in all the non-resected cases (3 unoperated patients, 1 patient who had pyloroplasty and 1 subjected to gastroenterostomy) and the roentgenologic appearance was consistent with food recently ingested (Fig. 4*A*). Gastroscopy and direct microscopy confirmed the presence of gastric bezoars (Fig. 4*B*). About a third of the patients with gastric resection had smaller separate fungus colonies around a solid bezoar in addition to a separate large bezoar. The contrast medium adhered readily to the mucus covering the uneven surface of the bezoar and the roentgenogram showed a fungoid formation (Fig. 3*B*). The gastric remnant generally emptied well and no real retention was seen. However, emptying of the stomach was somewhat delayed in some of the recently operated cases.

The lavage tube or gastroscope broke up the bezoar easily, resulting in a rapid change in the roentgenologic appearance. Vomiting of the fungus mass sometimes made the roentgenologic finding quickly negative.

In 1 case the bezoar was seen on the plain roentgenogram as a mass with translucent areas caused by air bubbles (Fig. 6).

Table V

DISAPPEARANCE OF YEAST BEZOARS

No. of Cases	Follow-up in Months
10	Under 6 months
8	6–12 months
7	12–24 months
3	Over 24 months
28	

TREATMENT

In the early years, when the authors were not familiar with the nature of yeast bezoar, Billroth II reconstruction was performed because of the steady growth of the bezoar, and operative evacuation of the

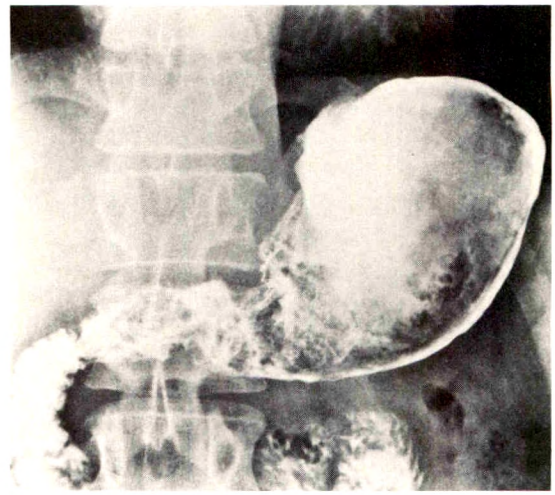

Fig. 2. The supine roentgenogram shows the bezoar adjoining the gastric fundus. Contrast medium envelops the entire bezoar showing that it is not fixed to the stomach wall.

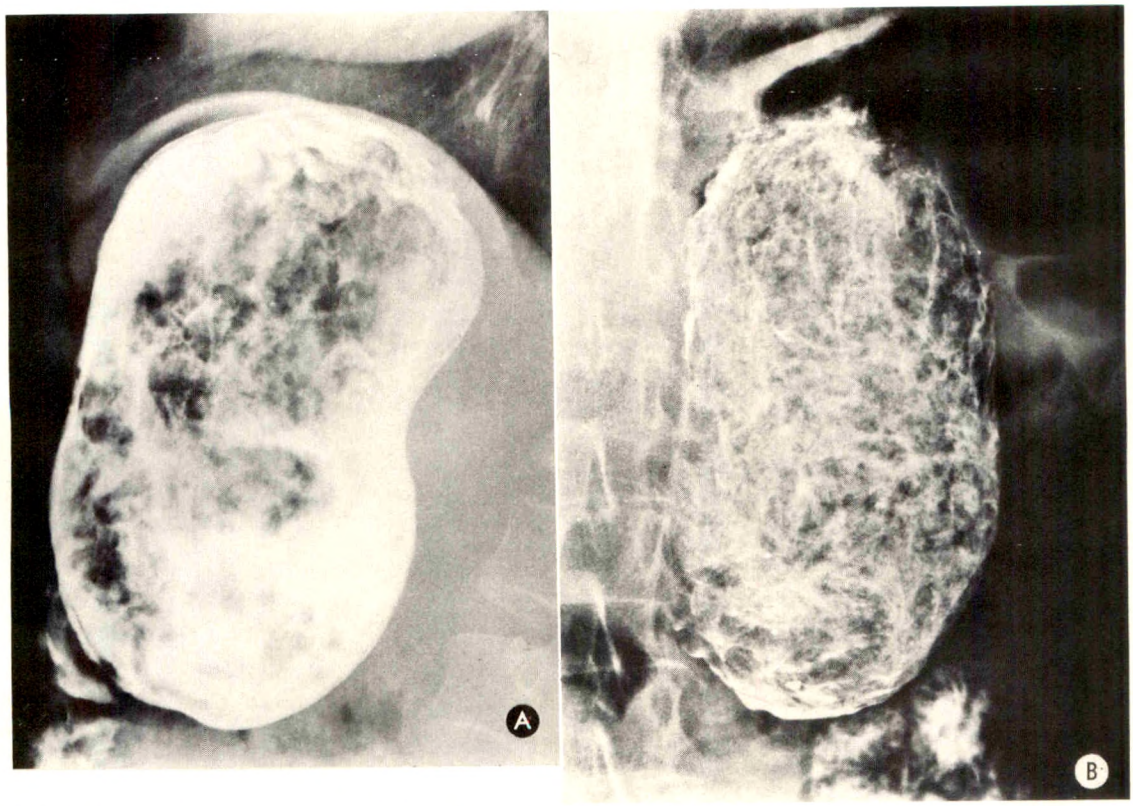

FIG. 3. (*A*) Supine roentgenogram. The solid bezoar which fills the entire gastric remnant causes an irregular, mottled filling defect. (*B*) Same patient an hour later. The bezoar now resembles a sponge with some contrast medium sticking to the surface.

bezoar was undertaken in 1 case.

Conservative therapy usually consisted of gastric lavage and antimycotics (Nystacid[R], Stereomycin[R]). Agents increasing gastric acidity, such as Pepsacid[R], lemon, and red wine, were given as nonspecific therapy. Some of the patients received no treatment at all. Disappearance of the bezoar in the different operative groups is noted in Table VI.

DISCUSSION

Seventy-five per cent of the 40 postoperative yeast bezoars in this series originated after an operation combined with vagotomy, and only 25 per cent formed after gastric resection alone.

The predisposing conditions for the formation of fungus masses appear to be created by vagotomy combined with a Billroth I procedure. The incidence of yeast bezoars in our series after this type of surgery was 50 per cent.

The change in conditions caused by gastric resection and especially by vagotomy is evidently the reason for bezoar formation. The most important factors mentioned in the literature are the change in both gastric motility and acid secretion and the relative stenosis caused by the operation. The growth of yeast organisms has been found to correlate with resection-induced achylia.[8]

Other factors that favor the growth of fungi are prolonged antibiotic therapy, infection, diabetes, and chronic renal and hepatic disease.[13] Our series included 3 primary bezoars, that is, unoperated patients, 1 of whom had diabetes and the other carcinoma of the lung. Eighteen (45 per cent) of the patients with bezoars were given a short course of tetracycline postoperatively.

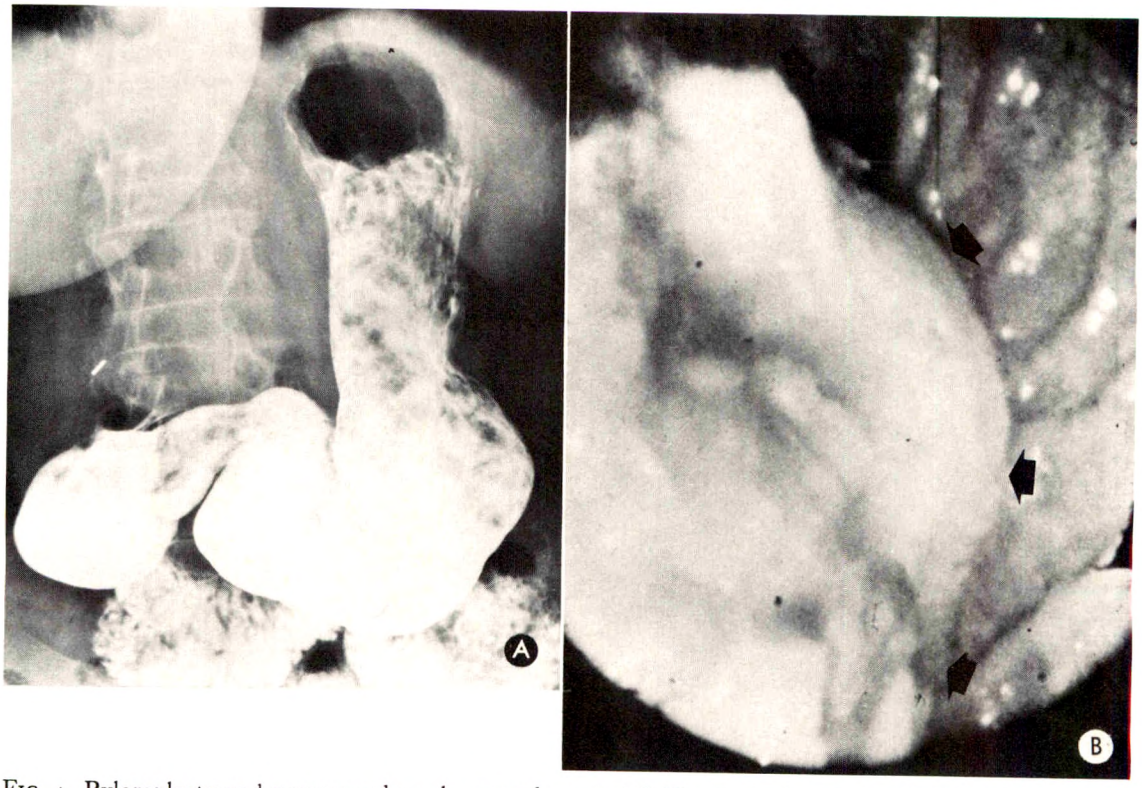

Fig. 4. Pyloroplasty and vagotomy have been performed. (*A*) The stomach contains several separate yeast bezoars. The roentgen appearance is consistent with that of recently ingested food. The stomach emptied freely, however. Gastroscopy and direct microscopy confirmed the diagnosis of yeast bezoar. (*B*) Gastrocamera picture of the same case shows a nodular bezoar mass of gray color (arrows).

However, this was evidently not of significance for the formation of the yeast bezoars, as the quantities of antibiotics received by operated patients with and without bezoar did not differ essentially.

Bezoar was very often an incidental finding at routine postoperative roentgenography which was usually made relatively soon after the operation. Sixty-two and one-half per cent of the bezoars in our series were detected within a year of surgery, but the latest was not seen until 27 years after resection. Yeast bezoars tend to recur despite therapy. It is not possible to be certain on the basis of the study how soon after the surgical procedure the bezoar forms in the resected stomach, for the postoperative roentgenologic control was scheduled in advance, usually for 3–6 months after surgery.

Roentgenologic examination proved to be a reliable method for diagnosing bezoars since the finding was without exception consistent with gastroscopy and microscopic studies.

In the differential diagnosis of yeast bezoars, conditions to be considered in addition to a tumor are retention of food, recently ingested food, jejunogastric intussusception, defects caused by sutures[14] and, of course, trichobezoars and phytobezoars. Differential diagnosis of a tumor is easy as the bezoar is generally not adherent to the stomach wall. Multiple bezoars can be readily interpreted as food in the stomach if the possibility of yeast bezoar is not borne in mind. A delayed roentgenogram which shows that the stomach empties freely but the bezoar masses remain, often enveloped in the contrast medium, is important for roentgenologic diagnosis.[9]

A repeat swallow of barium will show the

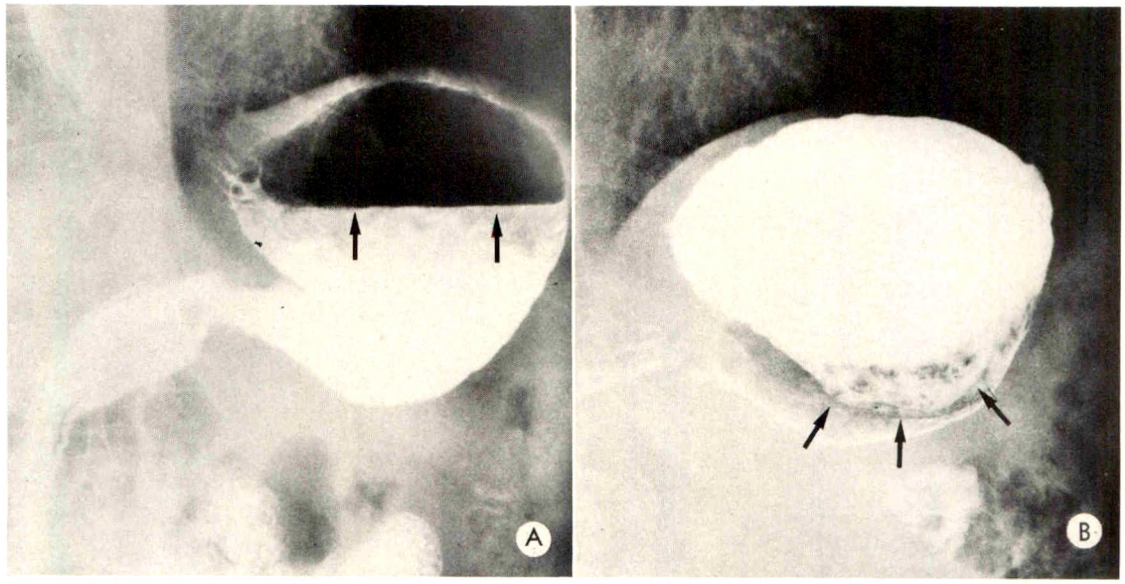

Fig. 5. (*A*) The horizontal fluid surface in the gastric remnant covers the curved upper surface of the bezoar. (*B*) Supine roentgenogram of the same patient shows the curved lower surface of the bezoar (arrows).

filling defects previously visualized if there are bezoars. If the diagnosis of retention is uncertain, gastric lavage is indicated. The filling defects seen at the subsequent re-examination will confirm the diagnosis of bezoar. In an erect roentgenogram simple retention shows a horizontal fluid level, while the upper surface of a solid bezoar is curved. A large bezoar may occasionally obstruct emptying of the stomach with a ball valve mechanism.[14]

Definite symptoms were seen in 24 patients (57 per cent). Every fourth patient had occasional vomiting. It was impossible to be certain which of the complaints were postoperative and which were caused by the bezoar. It is worthy of note that neither ulcer nor hemorrhage occurred in connection with postoperative yeast bezoars, as they often do in association with trichobezoars and phytobezoars in a non-operated stomach. The most common fungus species

Table VI

OUTCOME OF TREATMENT OF THE YEAST BEZOAR AFTER DIFFERENT GASTRIC OPERATIONS

Nature of Operation	No. of Bezoars	Therapeutic Result		
		Disappeared	Did not Disappear	Was not Followed
Vagotomy+Billroth I	21	12	6	3
Vagotomy+Billroth II	6	3	2	1
Vagotomy+pyloroplasty	2	2	—	—
Vagotomy+gastroenterostomy	1	1	—	—
Billroth I resection	4	3	—	—
Billroth II resection	6	4	1	1
Total	40	25	9	6
Not operated	3	3	—	—
Total	43	28	9	6

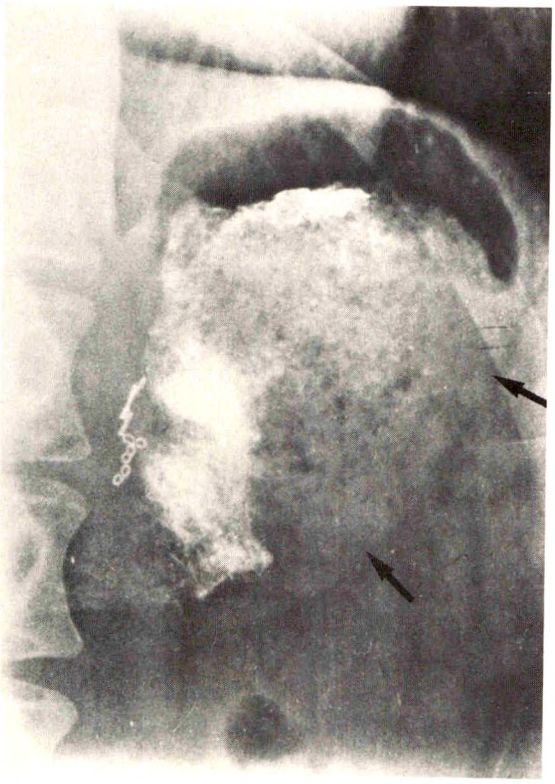

Fig. 6. Bezoar in which there are translucencies caused by air bubbles. Some biliary contrast medium swallowed the day before is on the bezoar surface.

were *Candida albicans* and *Torylopsis glabrata*, as they were in the bezoar material reported by Borg *et al.*[3] and Jepsen.[8]

The patients were given antimycotics perorally as well as substances that increased gastric acidity. Gastric lavage was also used as a therapeutic procedure. The importance of these methods is difficult to evaluate as some of the bezoars disappear without any treatment. According to Ahnlund and his co-authors,[1] gastric lavage removes the fungus for some time, but there is a recurrence after a few months.

In the early years when we were unfamiliar with the character of yeast bezoars, we changed the Billroth I resection to Billroth II as the therapeutic measure in 2 cases; the fungus mass was removed operatively in 1 case. The prognosis for yeast bezoar is generally good; 65 per cent of them disappear during the first follow-up year. On the other hand, the bezoar in 9 patients failed to disappear during observation for more than 2 years. Furthermore, some patients, in spite of therapy for several years, had a recurrent bezoar which gave manifest symptoms.

In several studies only phytobezoars deriving from food with a fibrous content have been reported as complications following gastric resection, while in others only yeast bezoars have been detected. There is a considerable discrepancy between the results, as neither diet nor antibiotic therapy accounts for the differences. Nine per cent of the ulcer patients operated on in the material of Goldstein *et al.*[7] had a postoperative phytobezoar, and the incidence rose to 14 per cent in the vagotomy+resection group. In our series a yeast bezoar was encountered in 11 per cent of the operated cases and in 50 per cent of the vagotomy +resection group. It is impossible to distinguish between phytobezoars and yeast bezoars roentgenologically. In our experience phytobezoars that form in an unoperated stomach are compact and difficult to break up, whereas yeast bezoars disintegrate easily. In contrast, in the opinion of Goldstein *et al.*,[7] a phytobezoar may occur either in friable or compact form, depending on its age.

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REFERENCES

1. Ahnlund, H. O., Pallin, R., Peterhoff, R., and Schönebeck, J. Mycosis of stomach. *Acta chir. scandinav.*, 1967, *133*, 555–562.
2. Asp, K., Peltokallio, P., and Perttala, Y. Schwamminfektion im Magen nach Resektion. *Langenbecks Arch. klin. Chir.*, 1970, *327*, 455–459.
3. Borg, I., Heijkenskjöld, F., Nilehn, B., and Wehlin, L. Massive growth of yeasts in resected stomach. *Gut*, 1966, *1*, 244–249.
4. Buchholz, R. R., and Haisten, A. S. Phytobezoars following gastric surgery for duodenal ulcer. *Surg. Clin. North America*, 1972, *52*, 341–352.

5. Cain, G. D., Moore, P., Jr., and Patterson, M. Bezoars-complication of postgastrectomy state. *Am. J. Digest. Dis.*, 1968, *13*, 801–809.
6. Drake, T. E., and Maíbach, H. I. Candida and candidiasis. 2. Clinical manifestations and therapy of candidal disease. *Postgrad. Med.*, 1973, *53*, 120–125.
7. Goldstein, H. M., Cohen, L. E., Hagen, R. O., and Wells, R. F. Gastric bezoars: frequent complication in postoperative ulcer patient. *Radiology*, 1973, *107*, 341–344.
8. Jepsen, O. L. Über Hefepilze im Mageninhalt nach Gastrektomie und Vagotomie. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1968, *109*, 269–279.
9. Mir, A. M., and Mir, M. A. Phytobezoar after vagotomy with drainage or resection. *Brit. J. Surg.*, 1973, *60*, 846–849.
10. Rigler, R. G., and Grininger, D. R. Phytobezoars following partial gastrectomy. *Surg. Clin. North America*, 1970, *50*, 381–386.
11. Rogers, L. F., Davis, E. K., and Harle, T. S. Phytobezoar formation and food boli following gastric surgery. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *119*, 280–290.
12. Schönebeck, J., Ahnlund, H. O., Pallin, B., and Peterhoff, R. Mycosis ventriculi. *Nord. med.*, 1967, *78*, 1134.
13. Smith, J. M. B. Mycoses of alimentary tract. *Gut*, 1969, *10*, 1035–1040.
14. Szemes, G. C., and Amberg, J. R. Gastric bezoars after partial gastrectomy: report of 5 cases. *Radiology*, 1968, *90*, 765–768.

GALLSTONE ILEUS*

THE IMPORTANCE OF CONTRAST EXAMINATIONS IN THE ROENTGENOGRAPHIC DIAGNOSIS

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ABSTRACT:

Our experience based on the analysis of 12 proved cases of gallstone ileus is presented emphasizing the fact that the plain abdominal roentgenogram may be inconclusive or deceiving. It allowed us to suggest the correct diagnosis in only 4 cases. A high index of suspicion, particularly in elderly female patients, followed by barium examination of the upper gastrointestinal tract leads to a correct and rapid diagnosis. The demonstration of a diverticulum-like structure or a fistulous tract adjacent to the first portion of the duodenum associated with jejunal dilatation and barium dilution is highly reliable.

THE diagnosis of gallstone ileus can be made preoperatively, with a high degree of accuracy, only by roentgenographic examination. For over 30 years[14] it has been well accepted that the presence of a triad of roentgenographic findings on the scout abdominal film is virtually pathognomonic.[5,7] This triad consists of air in the biliary tree, ectopically located gallstones, and a small bowel obstruction pattern. These observations, when present, allow for a rapid and reliable diagnosis leading to adequate treatment and an improved prognosis.

In the last few years, however, we have been impressed by a number of cases presenting with nonspecific or negative plain film abdominal examinations. In many of these cases the preoperative diagnosis was missed or made only subsequent to a contrast medium examination.

This report describes our personal experience on this subject, underlines the pitfalls in the roentgenological diagnosis, and stresses the need for contrast medium examinations in establishing a correct diagnosis.

MATERIAL

Our evaluation is based upon an analysis of 12 patients with proved gallstone obstruction.

Abdominal distention and diffuse abdominal pain of recent onset (2–3 days) accompanied by nausea and vomiting were the presenting symptoms in our group of patients. In 1 case the dominant symptom was hematemesis. The white blood cell count values varied between normal and as high as 30,000/mm.^{3}. The serum amylase level was occasionally elevated and reached 2,000 Somogyi units in 1 case. There were 8 females and 4 males in our series. The average age was 74 years, all patients being over 60.

RESULTS

PLAIN ABDOMINAL FILM

Supine and upright abdominal films were obtained in all cases, many times with repeat examinations in the days following the admission to the hospital.

Air in the biliary tree was visualized in 5 patients (Fig. 1). In 1 case this finding was initially missed, being recognized only in retrospect.

A faintly calcified and ectopically located gallstone was visualized in only 3 cases. These were located in the pelvis or mid-abdomen and were partially obscured by

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bone structures (Fig. 2*A*).

The small bowel pattern was considered compatible with intestinal obstruction in 6 cases. It consisted of dilated small bowel loops, air fluid levels on the upright film, and variable amounts of air within the colon. It was difficult at times to differentiate this pattern from that of a paralytic ileus; follow-up examinations and barium studies were requested for this reason. The remaining 6 cases were unremarkable, demonstrating only mild degrees of nonspecific ileus. Retrospectively, we questioned the presence of ill-defined areas of haziness over the midabdomen suggestive of fluid filled small bowel loops in 3 of these cases (Fig. 3*A*). This finding was debatable, however, and was not appreciated at the time of the examination.

In 4 cases combinations of specific roentgen-ray findings were demonstrated on the plain abdominal film. In only 1 patient, however, was the roentgenologic triad previously described present.

On the basis of the plain abdominal examination, the preoperative diagnosis of gallstone ileus was suggested in 4 cases and was missed in the remaining 8 patients.

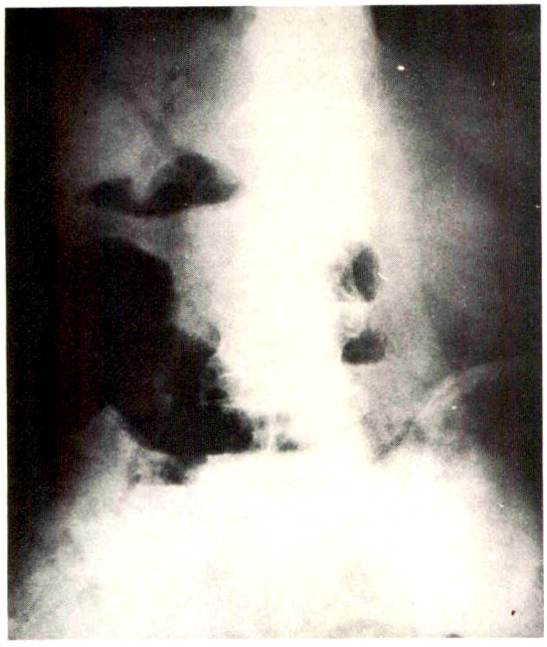

Fig. 1. Case 1. An 83 year old male. Upright abdominal film shows air within the biliary tree, distended air filled small bowel loops, and air fluid levels. At operation a large non-calcified calculus was found obstructing a jejunal loop.

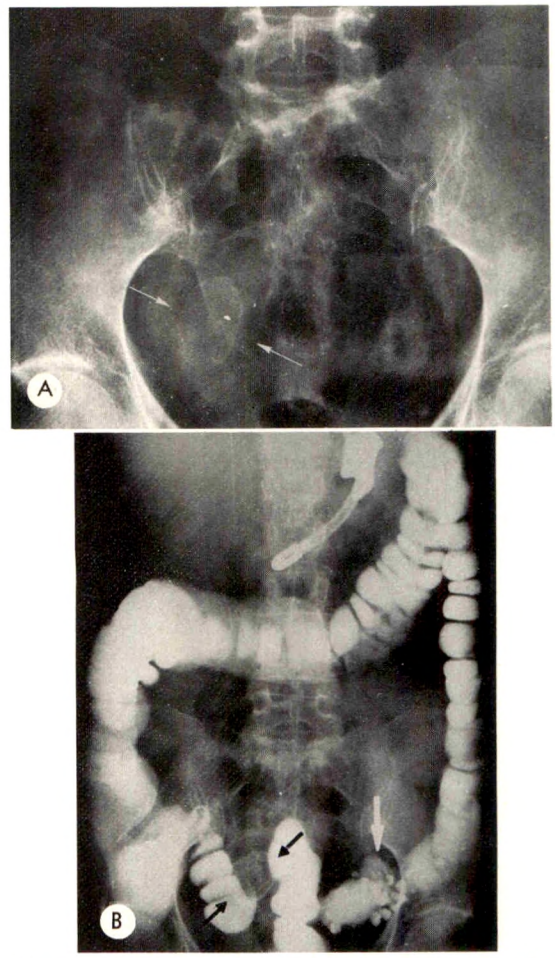

Fig. 2. Case 11. A 74 year old female. (*A*) Supine scout film. Oval 4 cm. long calcified gallstone is visualized in the right lower quadrant (arrows). (*B*) Barium enema examination with reflux into terminal ileum. The obstructing calculus is present in the terminal ileum (arrows). Diverticulosis of the sigmoid colon is demonstrated (single arrow). At operation the gallstone was found obstructing the sigmoid colon. Note, in addition, absence of air in the biliary system.

CONTRAST MEDIUM EXAMINATIONS

These were, in our series, the most rewarding investigations performed. They were obtained to reinforce a suggested diagnosis of gallstone ileus or to evaluate the nature and location of clinically or roentgenologically suspected small bowel ob-

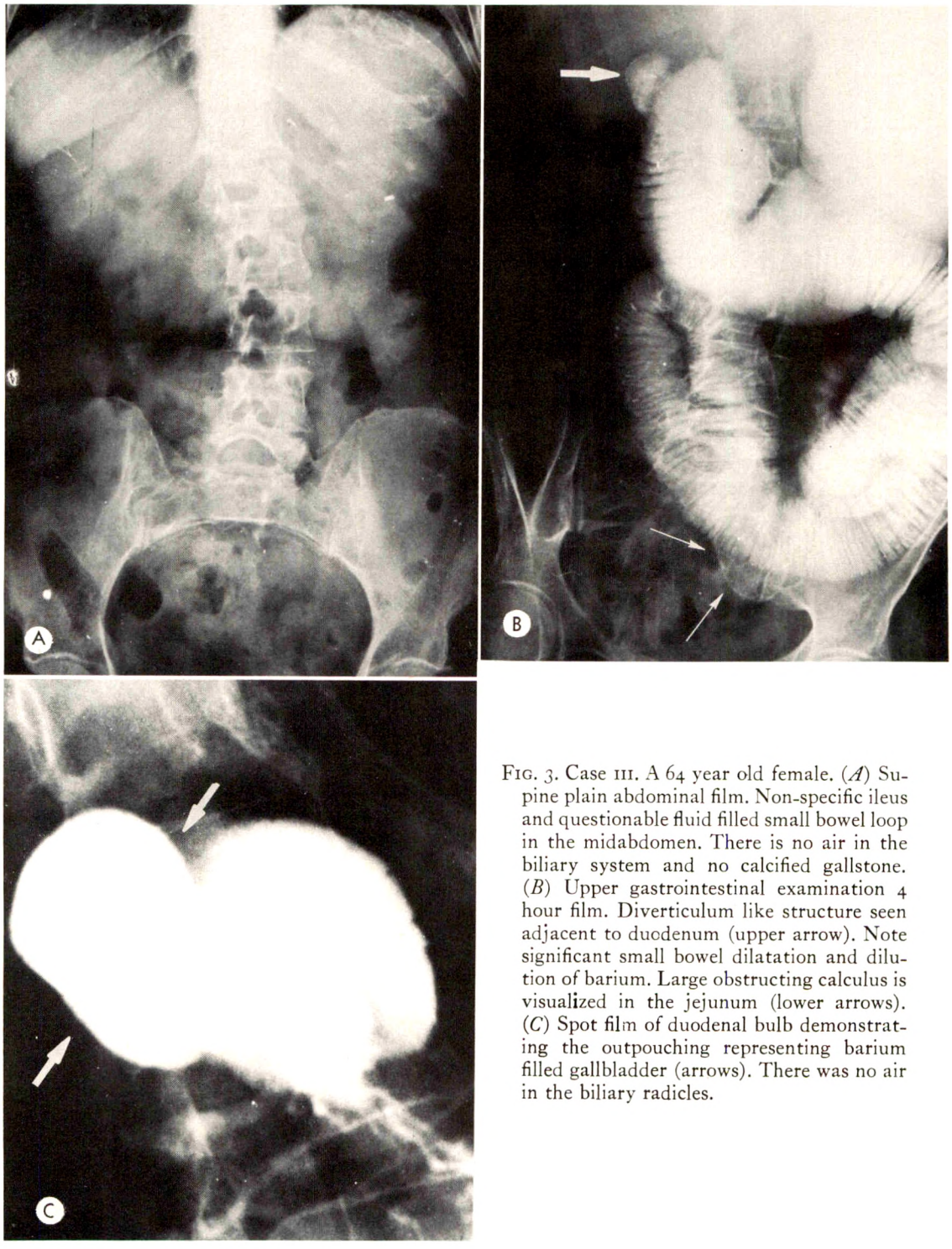

Fig. 3. Case III. A 64 year old female. (*A*) Supine plain abdominal film. Non-specific ileus and questionable fluid filled small bowel loop in the midabdomen. There is no air in the biliary system and no calcified gallstone. (*B*) Upper gastrointestinal examination 4 hour film. Diverticulum like structure seen adjacent to duodenum (upper arrow). Note significant small bowel dilatation and dilution of barium. Large obstructing calculus is visualized in the jejunum (lower arrows). (*C*) Spot film of duodenal bulb demonstrating the outpouching representing barium filled gallbladder (arrows). There was no air in the biliary radicles.

struction. When examined retrospectively, all 7 upper gastrointestinal examinations performed had definite findings permitting a positive diagnosis. Four cases demonstrated the barium filled gallbladder as a diverticulum-like structure adjacent to and filling from the first duodenal segment (Fig. 3, *B* and *C*; and 4). In 1 case a tubular structure representing a cholecystoduodenal fistulous tract was visualized (Fig. 5) while in the remaining 2 cases the entire extrahepatic biliary tree filled with barium. These findings were, unfortunately, twice misinterpreted as giant duodenal ulcers or localized duodenal perforations. In addition, small bowel obstruction manifested by significant dilatation and dilution of barium in the intestine was present in all the cases examined. In 2 cases delayed films were obtained and the obstructing calculus was visualized (Fig. 3*B*). Reflux into the terminal ileum during barium enema examination demonstrated the obstructing gallstone in 1 case (Fig. 2*B*). Oral and intravenous cholangiographic examinations of the biliary tract were performed in several of our cases and consistently showed non-opacification of the gallbladder. Visualization of the fistulous tract was never accomplished.

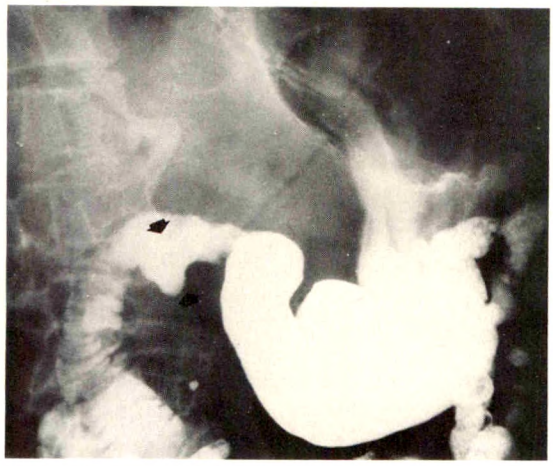

Fig. 4. Case IV. A 77 year old male. Upper gastrointestinal examination, right anterior oblique film. Note the diverticulum like structure filling from the first duodenal segment (arrows). There is no barium nor air within the biliary system.

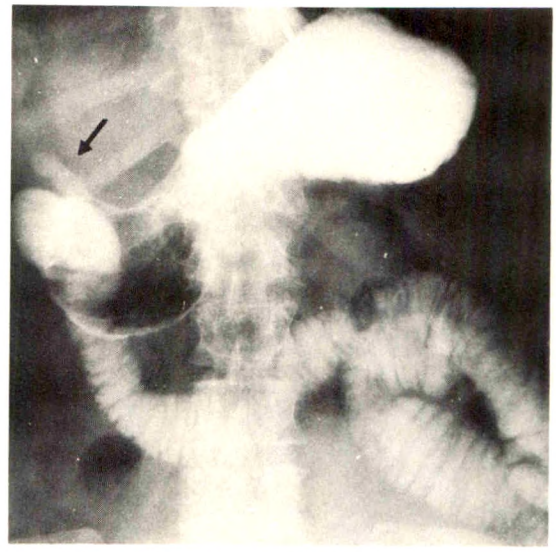

Fig. 5. Case V. An 84 year old female. Upper gastrointestinal examination, supine film. There is a fistulous tract in the right upper quadrant adjacent to the first portion of duodenum (arrow). Note dilatation of the proximal jejunal loops.

All cases were proved at surgery. In 4 cases the diagnosis was not suspected and was made only in the operating room. Six patients had the obstructing calculus located in the proximal jejunal loops, while in 5 it was found in the terminal ileum. In 1 patient (Fig. 2*B*) a large gallstone was visualized initially in the terminal ileum; however, at surgery it was found in the sigmoid colon within an area of narrowing produced by diverticulosis.

DISCUSSION

Delay in the diagnosis and treatment of gallstone ileus may lead to serious complications, such as electrolyte imbalance, ischemic lesions, ulcerations of the small bowel, abscess formation, and occasionally free perforation and peritonitis. Mortality rates as high as 30–50 per cent have been reported[7,9,13] and have been directly correlated with significant delay in diagnosis.

The plain abdominal film can be very helpful whenever the specific roentgenographic findings previously described are present. In our experience, however, a significant number of patients present with

non-specific and non-diagnostic abdominal roentgenograms. These allowed us to suggest the correct diagnosis in only one-third of our patients.

While the plain abdominal film was, in our hands, unreliable or at best unpredictable, we found contrast medium examinations safe and highly reliable methods of investigation. In 1961, Kirkland and Croce,[10] reviewing the literature, reported a 35 per cent rate of success in the diagnosis of gallstone ileus using scout abdominal films; at the same time they predicted that an accuracy rate of 80–90 per cent would result by using contrast medium examinations. This fact has not been sufficiently stressed in the literature; contrast medium examinations are often not performed, and the roentgenographic findings associated with these examinations are frequently misinterpreted. Familiarization with the roentgenographic findings of contrast medium examinations will significantly improve the diagnostic accuracy in gallstone obstruction.

On the upper gastrointestinal examination a localized, well contained collection of barium visualized laterally, adjacent to the first duodenal segment (Fig. 3, *B* and *C*; and 4), should not be confused with a penetrating ulcer since there should be no deformity or spasticity of the duodenum. Furthermore, the examination of the small bowel consistently shows dilatation and significant dilution of the barium column known to occur with gallstone obstructions.[5] The demonstration of this association of roentgenographic findings is highly reliable and should lead to the correct diagnosis. When there is doubt, follow through small bowel examination will demonstrate the offending calculus as a large, smooth, intraluminal filling defect (Fig. 3*B*).

Contrast medium examinations should be performed without delay in cases clinically suspected of small bowel obstruction even when the plain abdominal film is considered unremarkable. This is particularly true in elderly females who are prone to develop gallstone obstructions more frequently. It has been previously shown that 80–90 per cent of the patients with gallstone ileus are women[5] and about 75 per cent are patients over 60 years old.[4] In a survey of 179 cases of small bowel obstruction in the elderly, Brockis and Gilbert[3] reported the incidence of gallstone obstruction as being 10 per cent at ages 50–60, 14.5 per cent at ages 60–70, and 24 per cent at ages over 70. The general incidence of intestinal obstruction due to gallstone varies in different series from 0.4–5 per cent.[2,5,13]

With a high index of suspicion and appropriate use of contrast medium examinations, particularly in elderly patients, the diagnosis of gallstone obstruction can be made without delay and serious complications can be avoided.

CONCLUSIONS

1. The plain abdominal film in cases of gallstone obstruction may be inconclusive and misleading.

2. Contrast examinations should be performed without delay in elderly patients, particularly females, suspected of small bowel obstruction even when the scout abdominal film is unremarkable.

3. The demonstration of a diverticulum-like structure or a fistulous tract adjacent to the first duodenal segment associated with intestinal obstruction on the delayed films leads to a rapid and correct diagnosis.

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The authors are grateful to Dr. Richard Claps, Department of Radiology, Greater Paterson General Hospital, Wayne, New Jersey, for permission to use Case 1 in this series.

REFERENCES

1. Bergner, H. L. Internal biliary fistulas. *Am. J. Gastroenterol.*, 1965, *43*, 11–22.
2. Borman, C. N., and Rigler, L. G. Spontaneous

internal biliary fistula and gallstone obstruction with particular reference to roentgenologic diagnosis. *Surgery*, 1937, *1*, 349–378.

3. Brockis, J. G., and Gilbert, M. C. Intestinal obstruction by gallstones: review of 179 cases. *Brit. J. Surg.*, 1956–1957, *44*, 461–466.
4. Dean, O. G. Internal biliary fistulas: discussion of internal biliary fistulas based on 29 cases. *Surgery*, 1939, *5*, 857–864.
5. Eisenman, I. J., Finck, E. J., and O'Loughlin, B. J. Gallstone ileus: review of roentgenographic findings and report of new roentgen sign. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 361–366.
6. Epperson, D. P., and Walters, W. Spontaneous internal biliary fistulas. *Mayo Clinic Proc.*, 1953, *28*, 353–360.
7. Figiel, S. L., Figiel, J. S., Wietersen, K. F., and Dranginis, J. E. Gallstone obturation: clinical and roentgenographic considerations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1955, *74*, 22–38.
8. Grossman, T. E. Cholecystocolic fistula: unusual cause of diarrhea. *Am. J. Gastroenterol.*, 1971, *55*, 277–283.
9. Hicken, M. F., and Coray, B. Q. Spontaneous gastrointestinal biliary fistulas. *Surg., Gynec. & Obst.*, 1946, *82*, 723–730.
10. Kirkland, K. C., and Croce, E. J. Gallstone intestinal obstruction: review of literature and presentation of 12 cases, including 3 recurrences. *J.A.M.A.*, 1961, *176*, 494–497.
11. Lally, E. T., and Jeffery, F. R. Acute rupture of gallbladder demonstrated by intravenous cholangiography. *Brit. J. Radiol.*, 1972, *45*, 466–467.
12. Mujahed, Z. Non-opacification of gallbladder and bile ducts: previously unreported cause. *Radiology*, 1974, *112*, 297–298.
13. Pitman, G. R., and Davies, A. Clinical and radiological features of spontaneous internal biliary fistulae. *Brit. J. Surg.*, 1963, *50*, 414–425.
14. Rigler, L. G., Borman, C. N., and Noble, J. F. Gallstone obstruction: pathogenesis and roentgen manifestations. *J.A.M.A.*, 1941, *117*, 21, 1753–1759.
15. Scott, H. I., and Bethea, R. Perforation of gallbladder. *Int. Surg.*, 1972, *57*, 339.
16. Shehadi, W. H. Roentgenologic observations in cases of fistulae of biliary tract. *J.A.M.A.*, 1960, *174*, 2204–2208.
17. Tanga, R. M., and Ewing, J. B. Primary malignant tumors of gallbladder: report of 43 cases. *Surgery*, 1970, *67*, 418–426.
18. Wagner, R. G., and Passaro, E. Choledochoduodenal fistula secondary to duodenal ulcer. *A.M.A. Arch. Surg.*, 1971, *103*, 21–24.

MEDIASTINAL NEUROBLASTOMA AND GANGLIONEUROMA*

THE DIFFERENTIATION BETWEEN PRIMARY AND SECONDARY INVOLVEMENT ON THE CHEST ROENTGENOGRAM

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ABSTRACT:

The roentgenologic diagnosis and differentiation of mediastinal neurogenic tumors are possible on the chest roentgenogram as a rule. The soft tissue mass may be ill-defined and the tumor "ghost-like" in the case of primary neuroblastoma, but it is usually obvious in ganglioneuroma and metastatic disease. The presence of calcifications differentiates neurogenic tumors from other posterior mediastinal tumors of childhood. They are common in primary and rare in secondary disease. Rib erosions and displacement are striking in neuroblastoma (after a few months of age), more subtle in ganglioneuroma, and absent with secondary involvement. In 3 out of 7 posterior mediastinal neuroblastomas the diagnosis and treatment were delayed, as the adjacent rib changes were not appreciated for some time. "Dumbbell" shaped tumors are usually associated with vertebral changes and myelography is indicated even in the absence of neurologic deficit. Thoracic deformity and disability subsequent to laminectomy, radiation therapy, or both, are present in all survivors.

THE roentgenologic recognition of the tumorous nature of a primary posterior mediastinal neuroblastoma may be difficult since the neoplasm may present as an ill-defined "ghost" mass simulating the roentgen appearance of a pleural pulmonary process.[1,13] Institution of adequate treatment is therefore occasionally delayed.

Intrathoracic neurogenic tumors are known to be common in the young.[6,8,10,12] The importance of early diagnosis and the benign nature of the mediastinal neuroblastomas *versus* the primary abdominal ones have also been stressed.[2,10,14,15]

We reviewed the clinical and roentgenologic documentation of patients diagnosed with neuroblastoma, ganglioneuroblastoma and ganglioneuroma seen at our institution in the last 10 years. Our aim was to detect the salient roentgenologic features which permit the improvement of the diagnostic accuracy on the chest roentgenogram. The study was prompted by a recently encountered case of a posterior mediastinal mass in a child with left lower lobe pneumonia in whom the tumor simulated right upper lobe collapse. In 3 out of 7 posterior mediastinal neuroblastomas the diagnosis and treatment were delayed, since the adjacent rib changes were not appreciated for some time (Cases II, III, IV).

MATERIAL AND METHOD

The available roentgenologic and clinical documentations in patients with the diagnoses of neuroblastoma, ganglioneuroblastoma, and ganglioneuroma were reviewed. The study comprises patient material seen from 1963 to 1973 included in the hospital tumor registry and our departmental coding.

A total of 44 patients had acceptable documentation in the last 10 years. Table 1 indicates the distribution of tumors by histologic diagnosis, age of the patient, and the number of deceased children, disregarding the location of the neoplasm.

* Presented at the 17th Annual Meeting of the Society for Pediatric Radiology, San Francisco, California, September 23, 1974.
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Table I

DISTRIBUTION OF NEUROBLASTOMAS, GANGLIONEUROBLASTOMAS AND GANGLIONEUROMAS FROM 1963 TO 1973

Age (yr.)	Total	Neuroblastoma	Ganglioneuroblastoma	Ganglioneuroma
0–1	13	13 (4)		
1–2	11	10 (6)		1
2–3	4	3 (3)		1
3–5	10	8 (8)	1	1
5–10	6	4 (4)		2
	44 (25 deceased)			

Twenty-three of 44 patients had mediastinal involvement. The distribution of these tumors is shown in Table II. There were 18 mediastinal neuroblastomas (6 primary, 12 secondary), 1 ganglioneuroblastoma, and 4 ganglioneuromas.

REPORT OF CASES

Case 1. C.A. No. 363226, a 10 year old Caucasian boy, presented with clinical and roentgenologic evidence of left lower lobe pneumonia. In addition, a right upper lobe density with a well-defined inferior border was considered right upper lobe collapse secondary to pneumonia (not illustrated).

Follow-up examination 2 weeks later showed resolution of the left lower lobe pneumonia and persistence of the right upper lobe abnormality. Rib erosions and broadening of the 4th to 7th ribs were recognized adjacent to the soft tissue density (Fig. 1*A*).

On a somewhat lordotic roentgenogram, the rib erosions and separations were more obvious (Fig. 1*B*). The ill-defined density was localized posteriorly (Fig. 1*C*), and indented the posterior surface of the esophagus (Fig. 2*A*). There were erosions of the vertebral pedicles and widening of the intervertebral foramina at T-3 to T-7 on the right (not illustrated). In spite of the absence of neurologic signs and symptoms, a myelogram demonstrated a complete block at the level of T-7, caused by a right-sided extradural mass (Fig. 2*B*).

The skeletal survey was normal. Vanillylmandelic acid tests were negative on 2 occasions. Laminectomy and subsequent thoracotomy were performed and the "dumbbell" shaped tumor was removed. The intraspinal portion measured 3×2×0.5 cm. The intrathoracic extrapleural mass, which was infiltrating the posterior chest wall, measured 13×8×3.5 cm. after removal. The histologic diagnosis was matured ganglioneurofibroma from a preexistent neuroblastoma. Deforming kyphoscoliosis developed 6 months following surgery. This deformity was due to the surgical intervention alone (Fig. 2*C*).

Case 11. P.S. No. 421432, a 9 month old

Table II

DISTRIBUTION OF MEDIASTINAL INVOLVEMENT BY THE TUMORS

Total No. of Patients	Neuroblastoma		Ganglioneuroblastoma	Ganglioneuroma
	Primary	Secondary		
23	6 (2)	12* (11)	1	4

* The only survivor of this group was 9 days old when diagnosed.
Numbers in brackets refer to death.

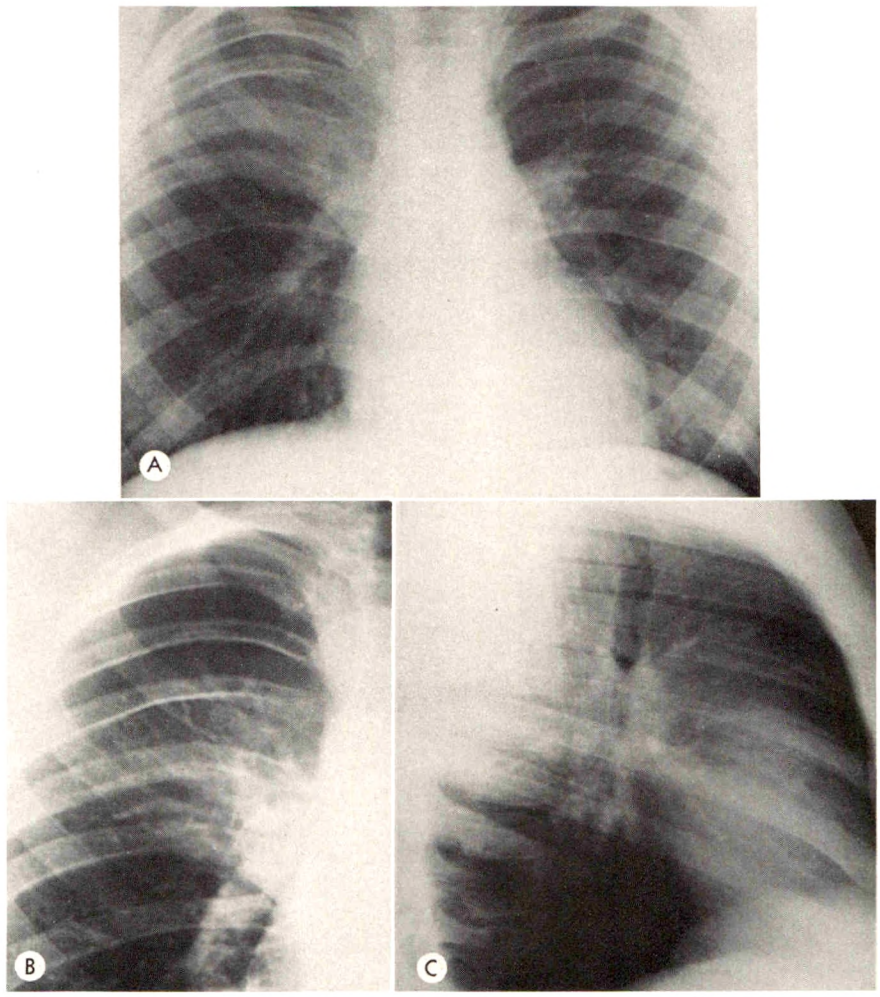

FIG. 1. Case 1. (*A*) Soft tissue density simulating right upper lobe collapse. There are posterior rib deformities. (*B*) On different projection the rib erosions and separations are more obvious. (*C*) The ill-defined density is superimposed on the thoracic spine.

Caucasian boy, had been treated at another hospital for "pneumonia" of 1 month's duration. A chest roentgenogram sent with the patient showed an ill-defined perihilar density on the right. Rib erosion and separation involved the 5th and 6th ribs posteriorly (Fig. 3*A*).

On initial physical examination the child was thought to be normal, but subsequently it became obvious that within the last few days he had lost the ability to stand up. Bone marrow examination and 2 vanillylmandelic acid assays were normal. A myelogram showed an almost complete block at T-6, and, after myelography, the child developed paraplegia and urinary retention. An emergency laminectomy was done. The intraspinal component of the tumor was partially removed and proved to be a neuroblastoma.

The intrathoracic component of the mass was treated with radiation therapy (tumor dose 4,150 r in 36 days), and the mediastinal tumor subsequently disappeared.

Recurrence was suspected and within 1 year of the initial radiation therapy, high-voltage radiation was delivered to the right lung on 2 separate occasions (3,700 r, time not stated, and 1,500 r in 26 days). Chemotherapy was also given.

Radiation pneumonitis, cor pulmonale, and congestive heart failure developed and the patient was digitalized. There was transient cortical blindness which was thought to be the result of respiratory and cardiac arrests. Bone marrow examination and vanillylmandelic acid assay were negative in the postoperative episode.

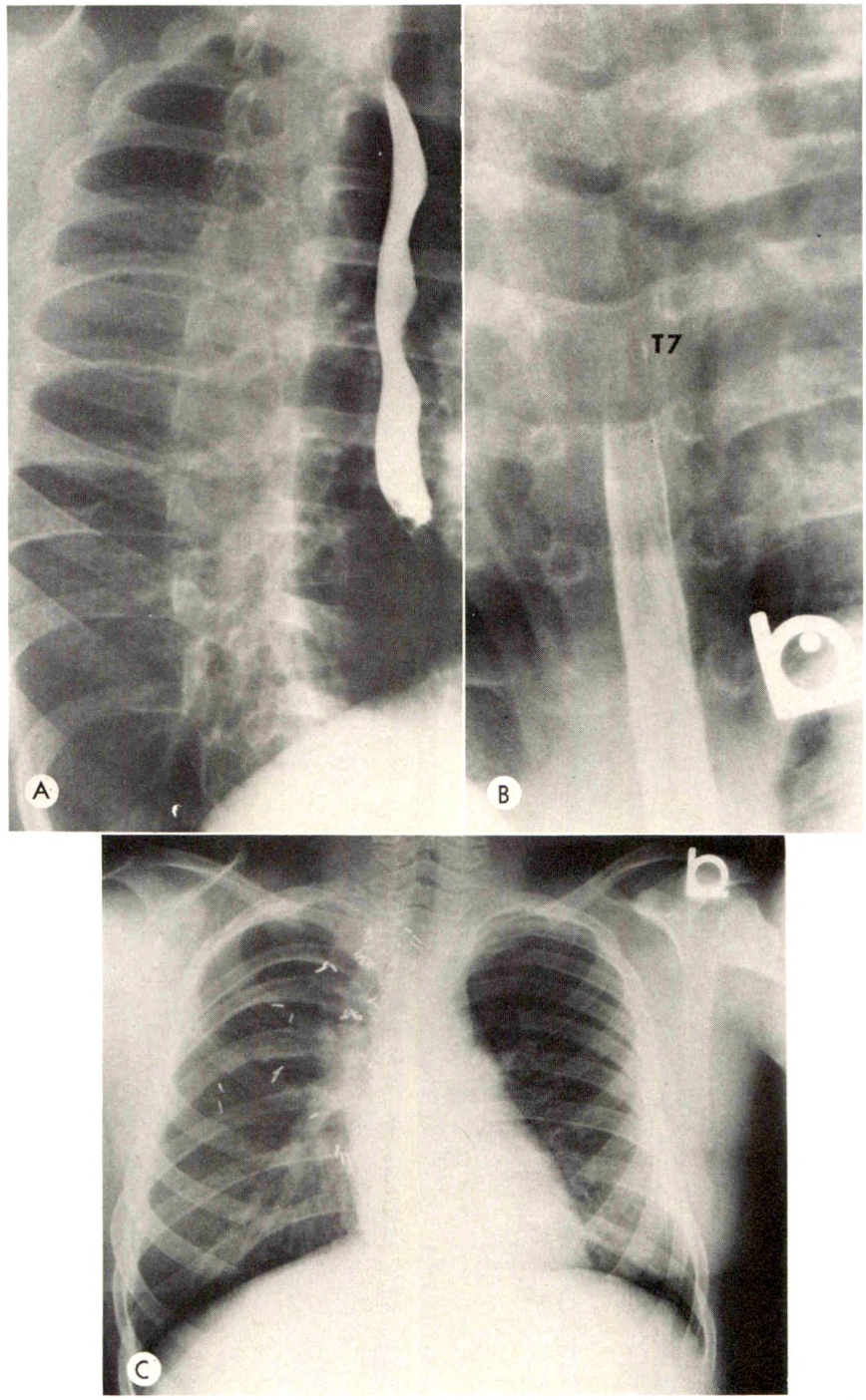

Fig. 2. Case 1. (*A*) Mild posterior indentation of the esophagus by the ill-defined mass. (*B*) There is a complete block at T-7. (*C*) Postoperative changes.

In the 3 years subsequent to the initial treatment, the child had 14 hospital admissions and had severe complications, most of which were considered to be iatrogenic. At the age of 4 years there is no evidence of metastasis or recurrence, but digoxin therapy is being continued, and the child suffers from severe chronic respiratory disease. There are right fibrothorax, mediastinal displacement, and kyphoscoliosis (Fig. 3*B*).

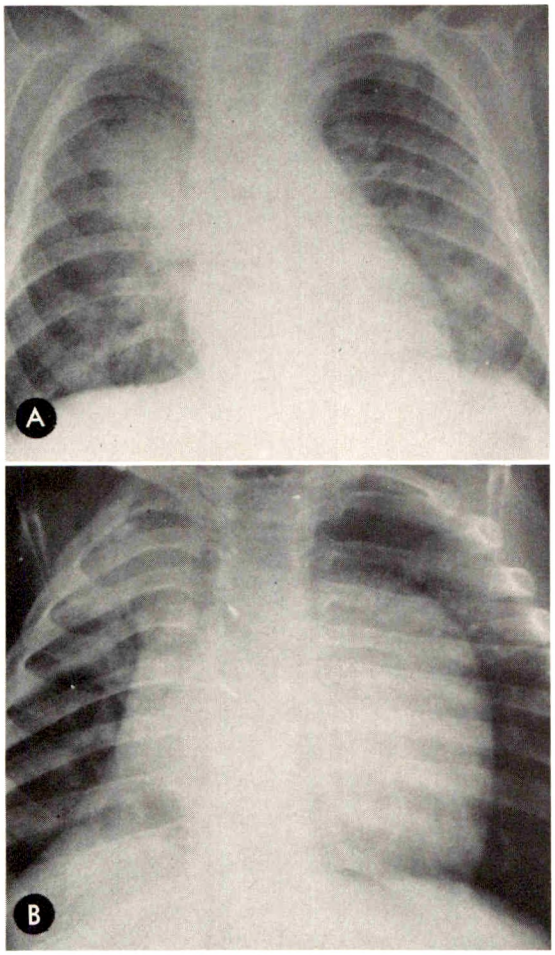

FIG. 3. Case II. (*A*) A soft tissue mass and rib changes. (*B*) Note the thoracic deformity and chronic lung changes at 4 years of age.

CASE III. K.M. No. 387961, a 4 year old Eskimo girl, was referred from another hospital for bone marrow biopsy. She had a history of vomiting and vague abdominal pain of 5 days' duration. On physical examination, non-tender cervical and left axillary lymphadenopathy was found. There was normochromic anemia (hemoglobin 7 grams per cent), and atypical leukocytes were found in the peripheral blood smear. The tuberculin test was positive, but she had had a BCG vaccination 1 year earlier. The chest roentgenogram was suggestive of primary pulmonary tuberculosis since a large left hilar mass was demonstrated.

On admission she was pale with hemoglobin of 5 grams per cent. The chest roentgenogram revealed an obvious left paravertebral mass containing calcifications, and there were separation and erosion of the ribs. The proximal ends of the humeri showed bony metastases (Fig. 4). The rib changes were not appreciated in the other hospital. Skeletal survey demonstrated disseminated bone involvement. Urography was normal. Both the bone marrow and lymph node biopsy showed neuroblastoma.

The patient was treated with chemotherapy and radiation therapy. The mediastinal tumor decreased in size but the patient expired 4 months later.

CASE IV. D.A. No. 404188, a 4 year old Caucasian girl, presented with left-sided chest pain, shortness of breath, and pallor, having been bumped by a cow 3 days prior to admission. Three months earlier she had fallen off a diving board and had suffered a brain concussion.

There was clinical and roentgenologic evidence of left-sided tension hemothorax. In addition, there were rib erosions and separations involving the posterior portions of the 4th to 6th ribs on the same side (Fig. 5*A*).

Thoracentesis yielded 175 ml. of blood. Subsequent examination showed the rib changes more clearly, as well as spotty soft tissue calcifi-

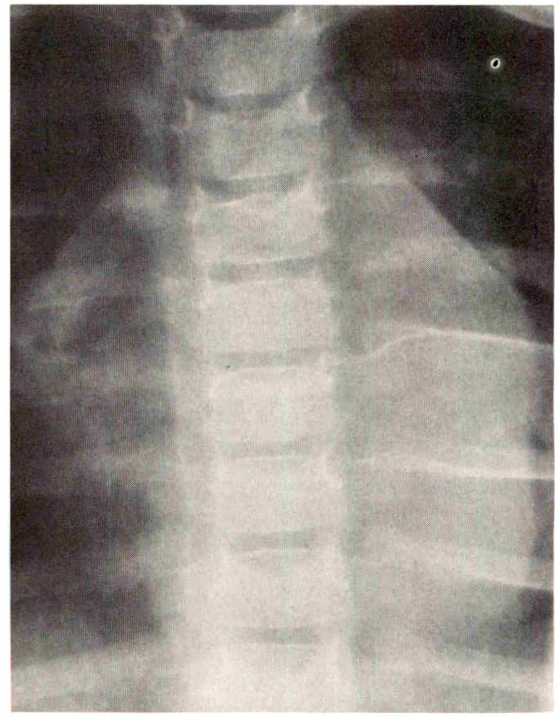

FIG. 4. Case III. There are obvious rib changes adjacent to the paravertebral neuroblastoma.

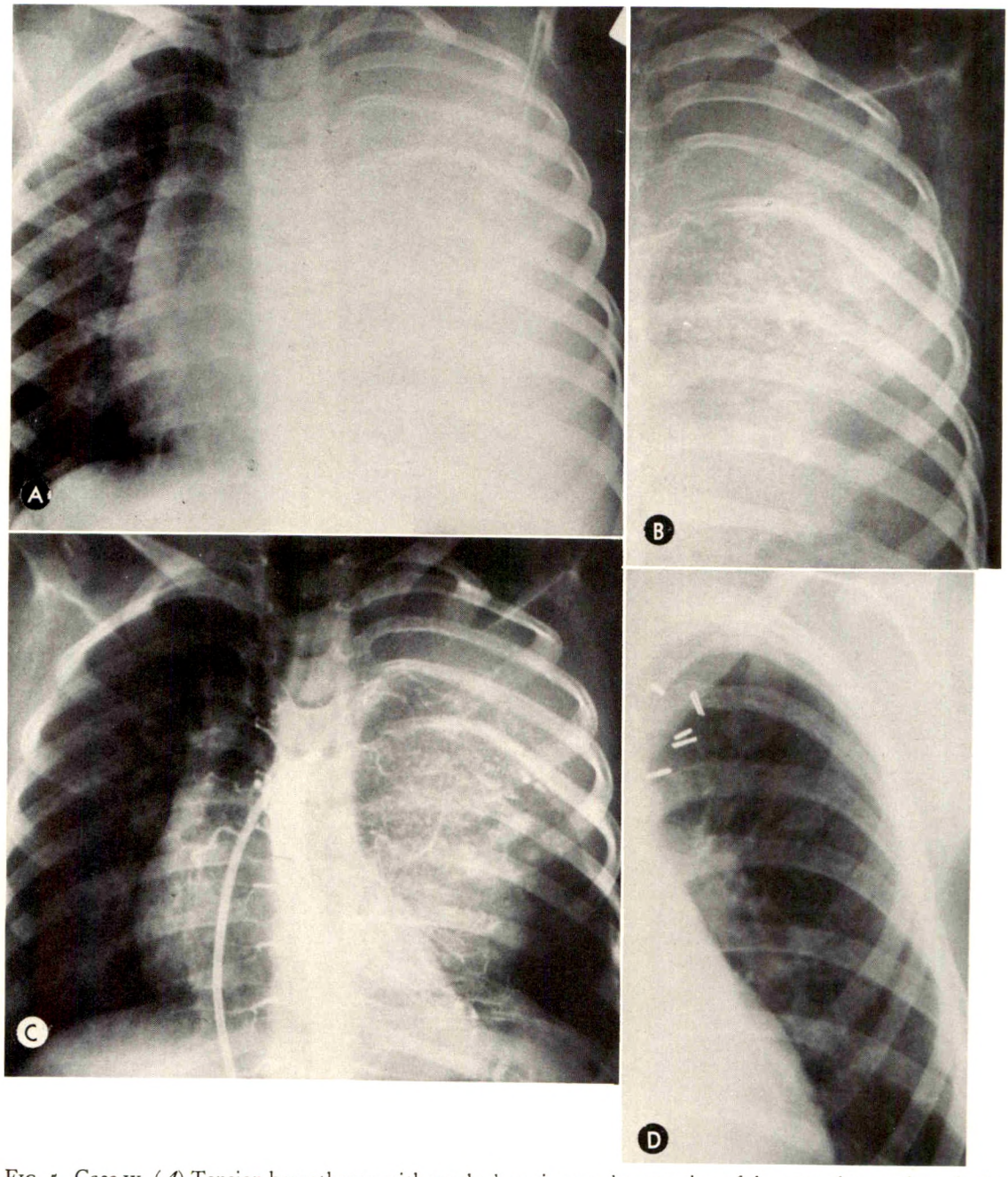

Fig. 5. Case IV. (*A*) Tension hemothorax with marked erosions and separation of the posterior portion of 4th to 6th ribs. (*B*) There are rib changes and the multiple spotty calcifications in the same area. (*C*) Note the displacement of the descending aorta and the tumor vessels. (*D*) There is regression of rib changes 6 months subsequent to therapy.

cations without an obvious mass (Fig. 5*B*). Vertebral erosions were also demonstrated. Bronchography demonstrated external compression of the left main bronchus. The thoracic aorta was displaced by a calcified soft tissue mass which was fed by tumor vessels (Fig. 5*C*). Spinal cord compression by an extradural mass was demonstrated on myelography. The vanillylmandelic acid assay was found to be elevated on 2 occasions. No neurologic signs were recorded during the child's hospital stay. Elective laminectomy was followed by thoracotomy and

TABLE III

ROENTGENOLOGIC FINDINGS, TREATMENT, AND FOLLOW-UP IN PRIMARY NEUROBLASTOMA AND GANGLIONEUROBLASTOMA

Patient Age Sex	Referring Diagnosis	Roentgenologic Findings														Surgery				Therapy				Follow-up and Comments
		Mass		Calcification		Rib				Vertebral Abnormality		Mediastinal Shift		Myelogram		Thoracotomy		Laminectomy		Radiotherapy		Chemotherapy		
						Erosion		Separation																
		Ghost	Obvious	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Abnormal	None	Yes	No	Yes	No	Yes	No	Yes	No	
1 G.G. 1 mo. No. 315329 Male	Disseminated neuroblastoma		+		o		o		o		o	+			o		o		o	+		+		Died at age 6 mo.
2 P.S. 1 mo. No. 292392 Male	Intrathoracic neuroblastoma Postoperative		+		o		o		o		o	+			o	+			o	+			o	6 years survival. Alive with kyphoscoliosis, Horner's syndrome, thoracic hemiatrophy. Preoperative examination from elsewhere
3 P.S. 9 mo. No. 421432 Male	Nonresolving pneumonia	+		+		+		+		+			o	+			o	+		+		+		4 years survival. Alive with kyphoscoliosis, fibrothorax, cor pulmonale
4 J.C. 3 mo. No. 387541 Male	Horner's syndrome		+	+		+		+		+		+		+		+		+		+		+		5 years survival. Alive with kyphoscoliosis, thoracic hemiatrophy
5 L.L. 21 mo. No. 457073 Female	Spastic paraparesis	+		+		+		+		+			o	+		+		+			o	+		2 years survival. Alive with local recurrence within a year of diagnosis. Repeat of laminectomy kyphoscoliosis
6 K.M. 4 yr. No. 387961 Female	? Tuberculosis		+	+		+		+			o		o		o		o		o	+		+		Died at age 4 years, 4 mo. Disseminated disease on admission
7* D.A. 4 yr. No. 404188 Female	Trauma hemothorax	Tension hemothorax		+		+		+		+		Hemothorax		+		+		+			o		o	3 years survival. Alive with kyphoscoliosis. Roentgenologic diagnosis made 7 weeks after admission

* D. A. Ganglioneuroblastoma.

TABLE IV

ROENTGENOLOGIC FINDINGS, TREATMENT, AND FOLLOW-UP IN PATIENTS WITH GANGLIONEUROMA

Patient Age Sex	Referring Diagnosis	Roentgenologic Findings					Surgery		Follow-up
		Mass			Rib Changes				
		"Ghost"	Obvious	Calcification	Erosion	Separation	Thoracotomy	Laminectomy	
B.V. 4.5 yr. No. 256145 Male	"Routine" chest		+	+	+	+	+	o	8 yr. well
S.J. 6.5 yr. No. 245069 Female	Heart murmur		+	o	+	+	+	o	14 mo. well
S.L. 3 yr. No. 276212 Male	Hemophilia		+	+	+	+	o	o	7 yr. (radiation therapy) hemophiliac. Well
C.A. 10 yr.* No. 363226 Male	Pneumonia	+		o	+	+	+	+	1½ yr. kyphoscoliosis

* The only "dumbbell" tumor with vertebral changes and complete block on myelography.

a large "dumbbell" tumor was removed.

The rib changes regressed postoperatively (Fig. 5*D*). Three years after surgery the child has kyphoscoliosis with no evidence of recurrence or metastases.

RESULTS

Primary Mediastinal Neuroblastoma. The findings in 6 primary mediastinal neuroblastomas and 1 ganglioneuroblastoma are summarized in Table III. The age at first hospital admission ranged between 1 month and 4 years, 4 children being less than 1 year old.

Neurologic signs or symptoms, or both, were recognized on admission in 4 out of 7 patients. Four "dumbbell" tumors were demonstrated by myelography. There was no correlation between the degree of cord compression on myelography and the presence or severity of neurologic deficit (Cases 2 and 4). There was an obvious mass recognizable on the chest roentgenograms in 4 patients with primary neuroblastoma (Table III). In 2, the contour of the mass was ill-defined ("ghost" tumor), and 1 child presented with a tension hemothorax which masked the neoplasm (Case 7).

Calcifications within the mass were present in 5 patients. The 2 infants who had no roentgenographic evidence of calcifications were 1 month of age at the time of diagnosis.

Rib erosions and separations were found in 5 patients (Cases 3–7; Table III). There was no rib abnormality in either of the 1 month old babies. The rib erosions and separations occurred in the posterior portion of the ribs adjacent to the soft tissue mass. The costal separation was marked in 4 patients and mild in 1 (Cases 3-7; Table III).

Vertebral changes (erosion of the pedicles and vertebral bodies, enlarged intervertebral foramina) adjacent to the mass were found in 4 children, all of whom had an intraspinal component of the tumor (Cases 4–6, 7; Table III).

Displacement of the mediastinal structures on the chest roentgenogram was present in 3 patients who had large tumors and in 1 child who had a hemothorax (Case 7; Table III).

Five out of 7 children are alive, 6, 4, 5, 2, and 3 years after the initial diagnosis (Table III). Two patients succumbed to the disease within 5 months of recognition. Both of these had disseminated neuroblastoma at the time of admission.

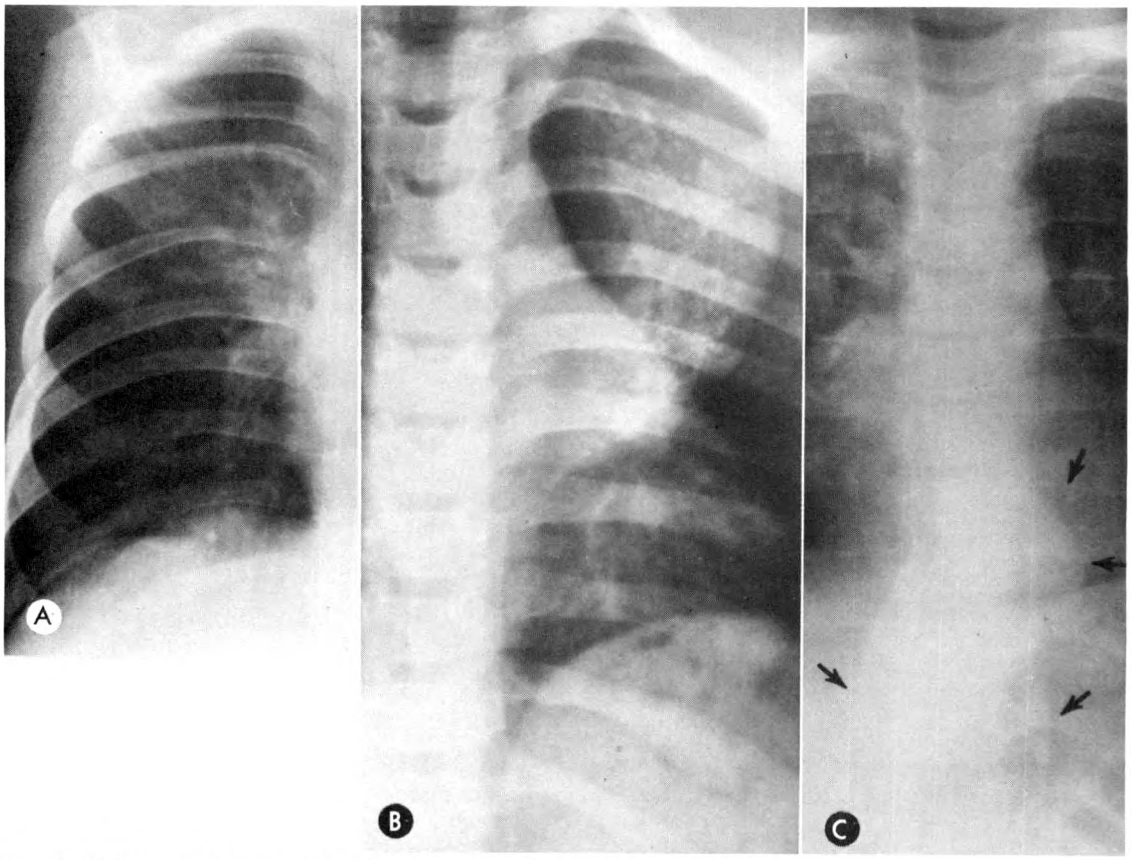

FIG. 6. Differences in roentgenologic appearance of primary neuroblastoma, ganglioneuroma and secondary neuroblastoma involving the mediastinum. (*A*) Note a "ghost" tumor with small calcifications and marked rib erosions and separations in a patient with primary mediastinal neuroblastoma. (*B*) Ganglioneuroma, with a sharp lateral contour, multiple calcifications, and subtle rib changes. (*C*) Metastatic mediastinal neuroblastoma. There is a bilateral triangular paravertebral widening with associated rounded deposits and absence of calcifications and rib changes.

There are significant sequelae of treatment in all survivors. Five children have kyphoscoliosis, 2 who received radiation therapy have thoracic hemiatrophy and 1 has severe fibrothorax and cor pulmonale (Table III; Fig. 3*B*).

Mediastinal Ganglioneuroma. The findings in 4 children with mediastinal ganglioneuroma are summarized in Table IV. The age range is 3 to 10 years. The initial chest roentgenogram showed a clear-cut posterior mediastinal tumor with a sharp border in 3 children. One patient presented with a "ghost" tumor which simulated right upper lobe collapse. The latter proved to be a "dumbbell" shaped mature neuroblastoma (Case 1). In 2 cases there were spotty calcifications in the soft tissue mass. Rib erosions and costal separations in the adjacent ribs were subtle and more difficult to identify than in primary mediastinal neuroblastoma (Fig. 6, *A* and *B*).

Displacement of the mediastinal structures was absent in all patients. The mediastinal tumors were removed in 3 children; 1 patient is a hemophiliac and he received only radiation therapy. There is no change in the size of the soft tissue mass 7 years after the initial diagnosis (Table IV).

Secondary Mediastinal Neuroblastoma. Twelve cases of metastatic mediastinal neuroblastoma were reviewed. In 11 patients there were metastases in the posterior mediastinum in the form of triangular

paravertebral masses; in 5 of these there were associated rounded or diffuse, or both, mediastinal deposits (Fig. 6*C*).

Rib erosions and separations similar to those found in primary mediastinal neuroblastoma were absent.

There were no calcifications in the nontreated secondary mediastinal neuroblastomas. One patient developed calcifications following radiation therapy (when the soft tissue mass regressed); another showed calcifications subsequent to surgery. There was only 1 survivor in this group, an infant 9 days old at the time of initial diagnosis (bony metastases were absent).

DISCUSSION

Patients with primary mediastinal neuroblastoma frequently present with nonspecific symptomatology. Therefore, the diagnosis depends on the awareness of the radiologist.

The roentgenologic appearance of primary mediastinal neuroblastoma is probably the result of the peculiar extrapleural tumor growth, lack of capsule formation, and the incidence of the roentgen-ray beam on a particular projection.[1,5]

Rib erosions and separations are the most important roentgenologic findings in the primary mediastinal neuroblastoma, even when the mass is ill-defined "ghost"-like (Cases 3, 5; Fig. 6). The recognition of rib changes leads to the diagnosis of an extrapleural mass which then may be better visualized with different projections and ideal technique.

In the newborn infant the rib changes, calcifications, and vertebral erosions may not be recognizable (Table III). We suggest the possibility that these changes are directly related to the age of the patient.

Subsequent to treatment (surgery, radiation therapy with or without chemotherapy), rib erosions and separations regress rapidly (Case IV; Fig. 5, *B* and *D*).

The identification of calcification within the soft tissue mass further enhances the diagnosis of primary mediastinal neuroblastoma and differentiates it from other posterior mediastinal tumors of childhood.[7]

Intraspinal extension of the tumor ("dumbbell," "hourglass" tumor) frequently occurs with primary mediastinal neuroblastoma and rarely with ganglioneuroma (Case 1).[4,11] Myelography should be performed when vertebral changes are associated with a posterior mediastinal mass, even in the absence of neurologic deficit Table III).

In ganglioneuroma the mass effect is predominant over the mild rib changes as a rule (Fig. 6).

Secondary deposits invade the extrapleural structures in an entirely different manner. Usually they appear as triangular masses located in the lower posterior mediastinum with their base directed inferiorly.[3] This configuration indicates thoracoabdominal origin. Rounded or diffuse metastases are usually found in the posterior mediastinum and rarely in the middle mediastinum.[9] Rib erosions and separations, as seen with primary mediastinal neuroblastoma, are absent even in cases with extensive bone marrow involvement.

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The authors are grateful to Miss Susan Wilkinson and Mrs. Adele Davidson for their secretarial help and to Miss Philippa Nelson for photography and art work on the scientific exhibit.

REFERENCES

1. Carey, L. S., Ellis, F. H., Good, C. A., and Woolner, L. B. Neurogenic tumors of mediastinum: clinicopathologic study. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *84*, 189–205.
2. Eklöf, O., and Gooding, C. A. Intrathoracic neuroblastoma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 202–207.
3. Eklöf, O., and Gooding, C. A. Paravertebral widening in cases of neuroblastoma. *Brit. J. Radiol.*, 1967, *40*, 358–365.

4. Fagan, C. J., and Swischuk, L. E. Dumbbell neuroblastoma or ganglioneuroma of spinal canal. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *120*, 453–460.
5. Felson, B. Fundamentals of Chest Roentgenology. The Extrapleural Space. W. B. Saunders Company, Philadelphia, 1960, pp. 205–215.
6. Gross, R. E., Forbes, S., and Martin, L. W. Neuroblastoma sympatheticum: study and report of 217 cases. *Pediatrics*, 1959, *23*, 1179–1191.
7. Hamilton, J. P., and Koop, C. E. Ganglioneuroma in children. *Surg., Gynec. & Obst.*, 1965, *121*, 803–812.
8. Hope, J. W., Borns, P. F., and Koop, C. E. Radiologic diagnosis of mediastinal masses in infants and children. *Radiol. Clin. North America*, 1963, *1*, 17–50.
9. Kincaid, O. W., Hodgson, J. R., and Dockerty, M. B. Neuroblastoma: roentgenologic and pathologic study. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *78*, 420–436.
10. Kinnier Wilson, L. M., and Draper, G. J. Neuroblastoma, its natural history and prognosis: study of 487 cases. *Brit. M. J.*, 1974, *3*, 301–307.
11. Lepintre, J., Schweisguth, O., Labrune, M., and Lemerle, J. Les Neuroblastomes en Sablier: Etude de 22 Cas. *Arch. franç. pédiat.*, 1969, *26*, 829–847.
12. Schweisguth, O., Mathey, J., Renault, P., and Binet, J. P. Intrathoracic neurogenic tumors in infants and children: study of forty cases. *Ann. Surg.*, 1959, *150*, 29–41.
13. Sherman, R. S., and Leaming, R. Roentgen findings in neuroblastoma. *Radiology*, 1953, *60*, 837–849.
14. Stella, J. G., Schweisguth, O., and Schlienger, M. Neuroblastoma: study of 144 cases treated in Institut Gustave Roussy over period of 7 years. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *108*, 324–332.
15. Young, L. W., Rubin, P., and Hanson, R. E. Extra-adrenal neuroblastoma high radiocurability and diagnostic accuracy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *108*, 75–91.

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BENIGN, SOLITARY SCHWANNOMAS OF THE LESSER PERITONEAL SAC*

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ABSTRACT:

Two cases of a large schwannoma of the lesser omental sac are described. One arose from the lesser omentum itself. This schwannoma also had roentgenographically visible calcifications, a finding never before reported to have been seen in the abdomen. Emphasis is upon the uniqueness of the site of the tumor.

BENIGN, solitary schwannomas or neurilemomas of the abdomen are rare. In a review of 303 cases of this entity at all sites, Das Gupta *et al.*[4] found no solitary neurilemoma of the gastrointestinal tract. They did, however, describe 2 cases of neurilemoma in the retroperitoneum, at least 1 of which was an abdominal mass.

The following 2 cases are examples of this benign neoplasm as it appears singly in the abdomen. In both instances the mass was discovered in the left upper quadrant of the abdomen on physical examination and further delineated roentgenographically to be in the region of the lesser omental sac. One mass arose from the lesser omentum itself; the other arose from the retroperitoneum.

REPORT OF CASES

Case 1. A 71 year old man was hospitalized for investigation of a large abdominal mass in the left upper quadrant found on routine physical examination. He was asymptomatic except for an occasional tugging feeling in the left upper quadrant. Further investigation, including examination of the upper gastrointestinal tract, barium enema study, and sonography demonstrated a large multiloculated soft tissue mass with scattered calcification. The mass displaced both stomach and colon and appeared to be centered in the region of the lesser omental sac (Fig. 1; and 2).

Laparotomy disclosed a mass 20 cm. in diameter arising from the posterior portion of the lesser omentum and filling the lesser sac. It was well encapsulated and removed *in toto*. Pathological examination demonstrated a benign, encapsulated, cystic schwannoma with calcifications.

Case 11. An 83 year old man was admitted to the hospital because of a 37 pound weight loss, intermittent back and epigastric pain, and nausea and vomiting occurring over a period of 4 months. He noted jaundice and clay-colored stools associated with intermittent pruritus 2 weeks before admission. On physical examination, a large non-tender mass was palpated in the left upper quadrant in the abdomen. Roentgenographic examination of the upper gastrointestinal tract and scans of the liver and spleen

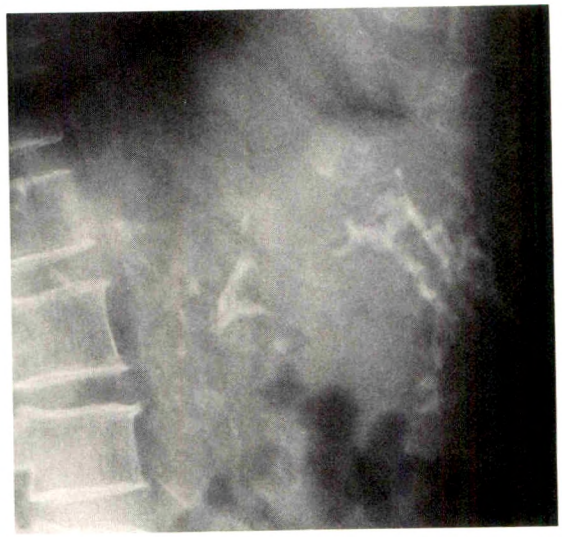

Fig. 1. Case 1. Lateral roentgenogram of the upper abdomen shows scattered calcifications within the abdominal mass, which lies anterior to calcified aorta.

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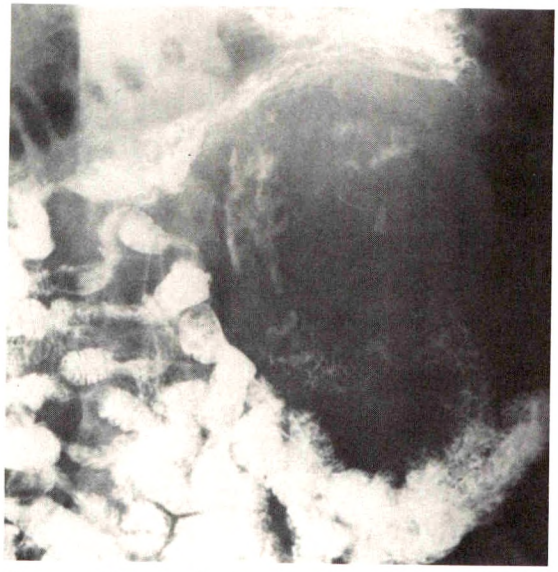

FIG. 2. Case 1. Supine roentgenogram following barium meal demonstrates the mass displacing the body of the stomach superiorly and small bowel inferiorly.

suggested a retrogastric, soft tissue mass (Fig. 3, *A* and *B*). An oral cholecystogram showed poor function of the gallbladder and possible gallstones.

Laparotomy disclosed a mobile, soft neoplasm, 10 cm. in diameter, in the lesser peritoneal sac with a small posterior pedicle arising from the retroperitoneum. The gallbladder was contracted and thick-walled. Large stones were found in the gallbladder as well as in the common and right hepatic ducts. Pathologically the mass was found to be an encapsulated neurilemoma with areas of myxoid degeneration and cyst formation.

DISCUSSION

Benign, solitary neurilemomas (schwannomas) were found in only 13 of 360,672 patients admitted consecutively to a large general hospital and 0.02 per cent of all patients admitted to a large university center over a 19 year period.[7,10] Most often these tumors occur along the course of peripheral nerves.

The actual cell of origin has been a matter of debate since 1803 when Odier first described the enlargement of peripheral nerves and coined the term "neuroma."[9] Most authorities now accept Schwann's cell as the cell of origin. The descriptive terms "neurilemoma" and "schwannoma" are used interchangeably.

In the 303 cases of neurilemoma reported by Das Gupta *et al.*,[4] women (57 per cent) were affected somewhat more often than men (43 per cent). There is no significant age predilection. Approximately 63 per cent of all their patients were between 30 and 60 years of age at the time of diagnosis.

The head and neck, especially the cranial nerves, are by far the most common locations of solitary neurilemomas, accounting for 40 to 50 per cent of all cases. In decreasing frequency the upper extremity, lower extremity, and trunk account for the sites of nearly all remaining cases. Few of these tumors have been reported to be in the abdomen and retroperitoneum.[4–6,11,12,14]

The literature suggests that stomach and small intestine are the most common sites of origin of schwannomas within the abdomen. The rectum is an uncommon site. In their review of the literature, Doyle *et al.*[5] found 192 cases of neurilemoma of the gastrointestinal tract. Of these, 154 were in the stomach, 38 in the small bowel, and none in the colon. They reported a case of a neurilemoma in the cecum, which is the only one at this site we were able to find in the English language literature. Rarely are cases described originating in the appendix, Meckel's diverticulum, liver, or greater omentum.[2–4,5,14]

Neurilemomas may attain a considerable size without causing symptoms, as in our cases. Gastrointestinal bleeding is the most common manifestation of intramural neurilemomas. Partial or complete intestinal obstruction also occurs.

An unusually high correlation exists between neurilemomas and malignancies at distant sites unrelated to peripheral nerves. Forty-nine (more than 16 per cent) of the patients in the series of Das Gupta *et al.*[4] developed unrelated malignancies before as well as after the neurilemoma was discovered. Among them 3 had malignant

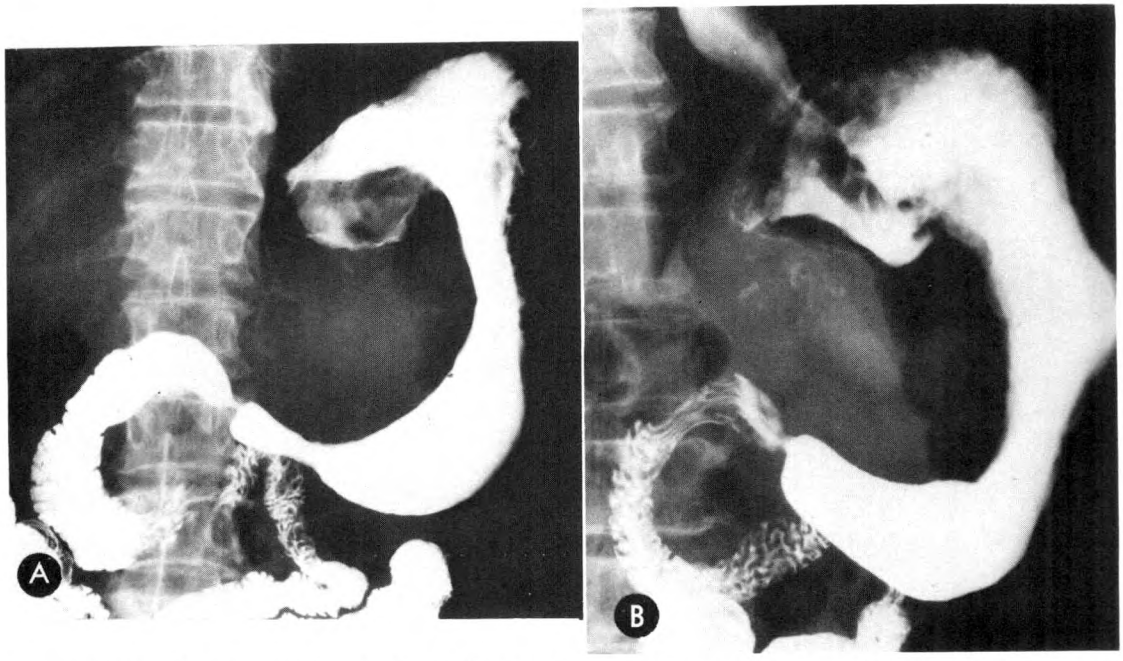

FIG. 3. Case II. Upper gastrointestinal examination. (*A*) Large mass creates a marked extrinsic pressure deformity along the lesser curvature of the stomach. (*B*) Oblique roentgenogram reveals that the mass lies posterior to the stomach in the region of the lesser sac. Scattered calcifications seen posterior are probably in splenic arteries.

melanomas, 12 had malignancies of the breast, 15 of the skin, and 8 of the gastrointestinal tract.

Our 2 cases are unusual in that both schwannomas were confined to the lesser omental sac and both had become quite large while producing only minor symptoms.

The roentgenographic finding of a calcified neurilemoma occurring in the abdomen (Case 1) has not been previously reported. Other calcified intra-abdominal masses are not rare. Pancreatic adenocarcinomas and islet cell tumors may calcify, although rarely. Cystadenomas and cystadenocarcinomas of the pancreas calcify somewhat more frequently.[1,13] Sawyer *et al.*[13] noted that 10 to 18 per cent of pancreatic cystadenomas are calcified; many exhibit a "sunburst" pattern of calcification. Very rarely, phleboliths are seen inside pancreatic and gastric lymphangiomas and hemangiomas.

Gastric leiomyomas commonly have microscopic calcifications, but rarely are the calcifications visible roentgenographically.[8] Fibromas and mucinous adenocarcinomas originating in the stomach likewise occasionally calcify. Hydatid cysts, calcified hematomas, and aneurysms may also be seen in this area.

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REFERENCES

1. BECKER, W. F., WELSH, R. A., and PRATT, H. S. Cystadenoma and cystadenocarcinoma of pancreas. *Ann. Surg.*, 1965, *161*, 845–863.
2. BERTRAND, P., PORTA, J.-P., MEYER, P., and LEQUIN, P. Schwannome du rectum (à propos d'une observation). *J. méd. Lyon*, 1969, *50*, 399–400.
3. CHALKLEY, T., and BRUCE, J. W. Neurofibromatosis of colon, small intestine and mesentery in child. *Am. J. Dis. Child.*, 1942, *64*, 888–894.
4. DAS GUPTA, T. K., BRASFIELD, R. D., STRONG, E. W., and HAJDU, S. I. Benign solitary schwannomas (neurilemomas). *Cancer*, 1969,

24, 355–366.
5. Doyle, J. L., Bryant, F. J., and Volta, P. W. Neurilemoma of cecum. *Texas J. Med.*, 1968, *64*, 42–43.
6. Herrington, J. L., and Edwards, L. W. Massive retroperitoneal neurilemoma, with emphasis on technical problems encountered during surgical removal. *Surgery*, 1965, *57*, 366–369.
7. Jacobs, R. L., and Barmada, R. Neurilemoma. *A.M.A. Arch. Surg.*, 1971, *102*, 181–186.
8. Leigh, T. F. Calcified gastric leiomyoma: report of case. *Radiology*, 1950, *55*, 419–422.
9. Lewis, D., and Hart, D. Tumors of peripheral nerves. *Ann. Surg.*, 1930, *92*, 961–983.
10. Money, R. A. Tumours of peripheral nerves. *Australian & New Zealand J. Surg.*, 1950, *19*, 239–245.
11. Okagaki, T., and Richart, R. M. Neurilemoma of fallopian tube. *Am. J. Obst. & Gynec.*, 1970, *106*, 929.
12. Rauber, G., Bessot, M., Barrucand, D., and Mauuary, G. Schwannome gastrique. *Semaine hôp. Paris*, 1969, *45*, 2529–2530.
13. Sawyer, R. B., Sawyer, K. C., and Spencer, J. R. Proliferative cysts of pancreas. *Am. J. Surg.*, 1968, *116*, 763–767.
14. Simon, G. C. Deux observations de volumineux schwannome bénin du mésentère. *Semaine hôp. Paris*, 1969, *45*, 2541–2546.

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EBSTEIN'S ANOMALY OF TRICUSPID VALVE: CRITICAL REVIEW OF ROENTGENOLOGICAL FEATURES AND ADDITIONAL ANGIOGRAPHIC SIGNS*

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ABSTRACT:

The plain roentgenographic and angiocardiographic features of 15 patients with Ebstein's anomaly of the tricuspid valve were reviewed. Of note on the plain roentgenogram was the fact that the "posterior bulge" seen on the lateral view was demonstrated to be due to either a markedly enlarged right atrium or the posterior displacement of normal left heart structures. The angiocardiographic features described in the literature were reviewed. Two main features seen were the "sail-like" appearance of the anterior leaflet of the tricuspid valve, which sometimes produces a tumor like picture, and the 2 notches seen when the ventricle contracts. One notch corresponds to the true tricuspid annulus. The other represents the site of attachment of the displaced tricuspid valve. The previously undescribed features were noted: (a) all three leaflets could sometimes be identified; (b) fenestrations in the anterior leaflet were demonstrated in 5 cases; and (c) the sinus portion of the ventricle was sometimes filled in a retrograde fashion from the infundibular chamber.

EBSTEIN'S anomaly is an uncommon congenital cardiac defect involving the formation of the tricuspid valve. Ebstein's original description in 1866 included an incisive discussion which encompassed most of the essential anatomic and hemodynamic features recognized currently.[13]

Although the roentgenographic features have received mention in the literature, differences of opinion and inconsistencies exist regarding plain film interpretation.[1,9,10] In addition, little emphasis has been placed on those specific angiographic observations which help to explain the plain film findings, and which correlate well with the anatomic and hemodynamic features of the disease.[4,5,6,11]

We reviewed the roentgenograms and angiographic studies in a group of 15 patients with Ebstein's anomaly studied at the Chaim Sheba Medical Center. We found that by a careful anatomic analysis of the

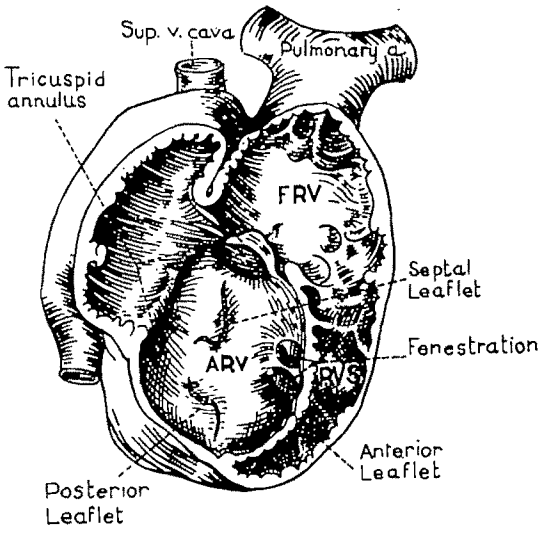

FIG. 1. Drawing depicting the pathological features in Ebstein's anomaly. Note the large displaced anterior leaflet and the smaller, also displaced posterior and septal leaflets of the tricuspid valve. FRV = Functional right ventricle, ARV = Atrialized right ventricle, RVS = Sinus portion of right ventricle.

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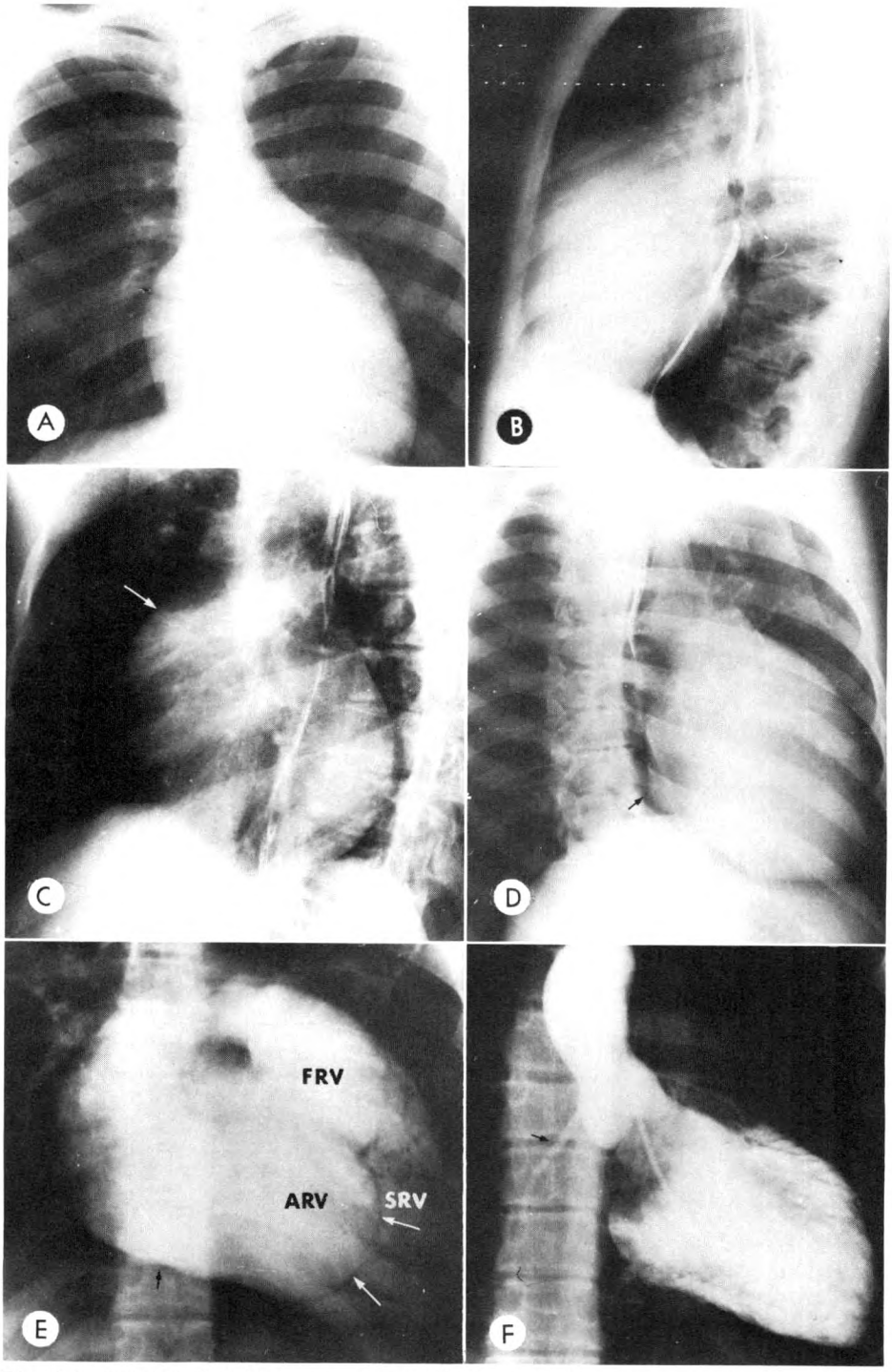

FIG. 2. Ebstein's anomaly. (*A*) Posteroanterior (PA) chest roentgenogram. Moderate cardiac enlargement with narrow vascular pedicle and diminished size of pulmonary vessels. The left heart border is characteristic of Ebstein's anomaly with small aortic knob, concave main pulmonary artery segment and enlarged horizontally inclined infundibular segment. (*B*) Lateral view. Enlarged right heart chambers displace the normal sized left ventricle behind the esophagus. (*C*) Left anterior oblique (LAO) view. The enlarged right heart chambers displace the left ventricle posteriorly to overlap the spine. The arrow indicates the region of the right atrial appendage which is not "squared-off." See explanation in text. (*D*) Right anterior

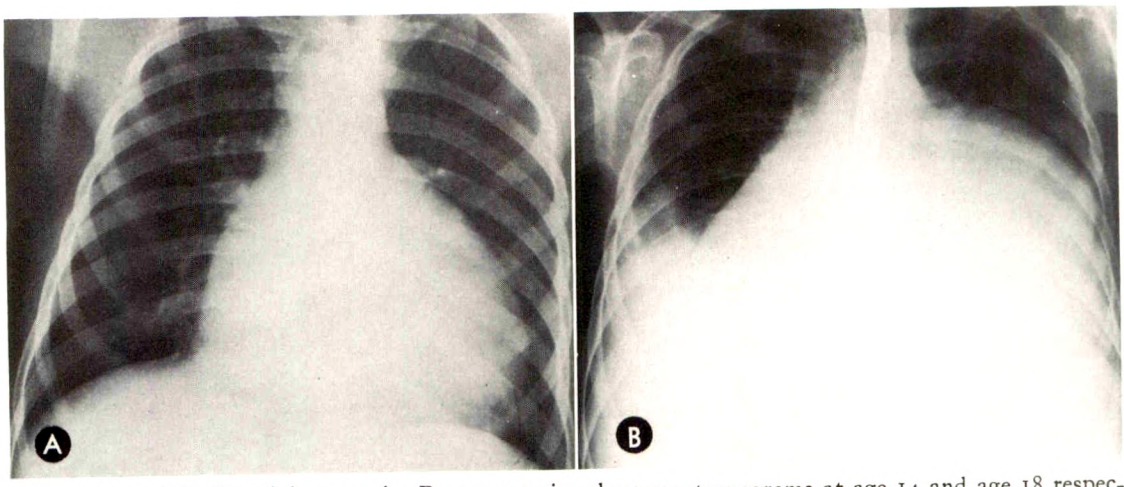

Fig. 3. (*A* and *B*) Ebstein's anomaly. Posteroanterior chest roentgenograms at age 14 and age 18 respectively. In (*A*) the pulmonary vasculature is decreased. The heart is enlarged with a characteristic box-like appearance, a narrow vascular pedicle and concave pulmonary artery segment. In (*B*) the heart size has markedly increased due to pericardial effusion and tricuspid insufficiency, but the box-like appearance is even more marked. Note also dilatation of superior vena cava and pleural effusion due to right heart failure.

angiograms performed in both the anteroposterior and lateral views, several diagnostic features previously not stressed could be recognized.

MATERIAL AND METHOD

Fifteen patients were studied. In each case, physical examination was performed on at least 1 and usually on several occasions. Thirteen lead electrocardiograms and plain chest roentgenograms were performed in each patient. Cardiac catheterization and angiographic studies were performed in all patients. The diagnosis was made by a combination of the features from the above mentioned tests, as well as by necropsy evidence in 5 cases.

CLINICAL AND LABORATORY FINDINGS

There were 9 females and 6 males. Their ages at the time of definitive diagnosis ranged between 4 days and 33 years. Shortness of breath was present in 7 patients and palpitations in 5. Congestive cardiac failure was present in 5 patients. Cyanosis was a feature in 8 patients: of these, 2 also exhibited clubbing.

A systolic thrill was palpable at the left sternal border in 6 patients. The first heart sound was single in 12 patients and split in 3 patients; the second sound was single in 3 patients and split in 12 patients; a third heart sound was audible in 6 patients; and a fourth heart sound in 5 patients. In all patients a systolic murmur was present at

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oblique view. The enlarged infundibulum of the right ventricle is seen anteriorly. The right atrium (arrow) projects posteriorly behind the esophagus. (*E*) Angiocardiogram following injection in superior vena cava. The shallow notch (vertical arrow) indicates the tricuspid annulus. The radiolucent curvilinear shadow (white arrows) represents the displaced anterior tricuspid leaflet. The atrialized portion of the right ventricle (ARV) lies between the true tricuspid annulus and the anterior tricuspid leaflet. The atrialized right ventricle empties directly into the enlarged infundibular chamber or "functional right ventricle" (FRV). The sinus portion of the right ventricle (SRV) can be faintly seen and is filled with radiolucent trabeculations which represent abnormal muscle bands and thickened chordae running between the anterior leaflet and the free wall of the right ventricle. (*F*) Retrograde left ventriculography, anteroposterior view. The right coronary artery (arrow) defines the right atrioventricular junction and, hence, the position of the true tricuspid annulus. Compare with (*E*). FRV = Functional right ventricle, ARV = Atrialized portion of right ventricle, SRV = Sinus portion of right ventricle.

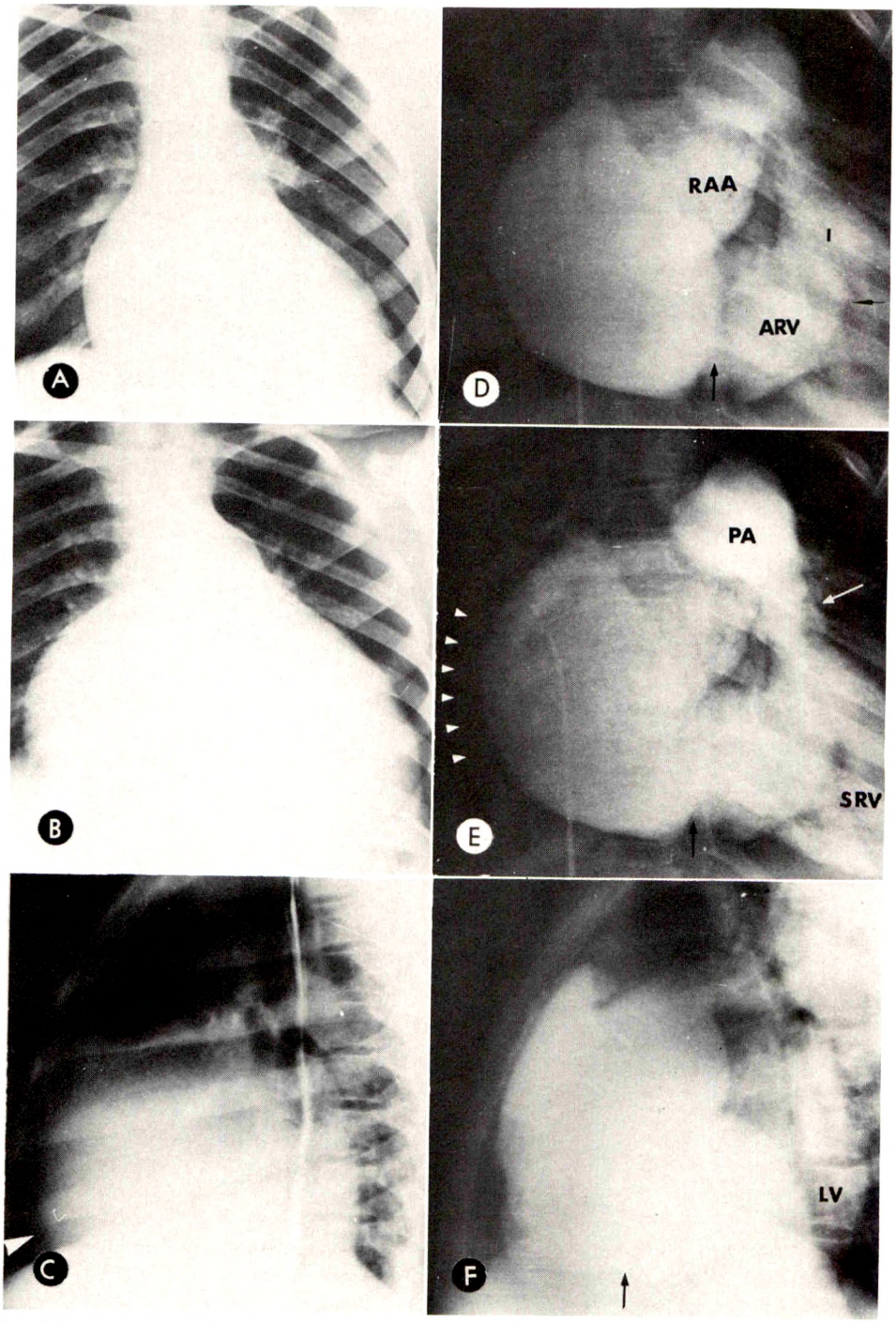


FIG. 4. Ebstein's anomaly with valvular pulmonic stenosis. (*A*) Posteroanterior chest roentgenogram, age 13. Prominent main pulmonary artery segment with right heart enlargement suggests valvular pulmonic stenosis, not "characteristic" of Ebstein's anomaly. (*B*) Posteroanterior chest roentgenogram, age 16. Cardiac size greatly increased due to pericardial effusion. Main pulmonary artery segment still prominent. (*C*) Lateral view, age 16. Markedly enlarged heart bulging behind esophagus. Nevertheless, the lower substernal region is not filled-in (arrowhead). (*D*) Angiocardiogram injection into right atrium, anteroposterior view. Markedly dilated right atrium with enlarged, medially placed right atrial appendage (RAA). Smaller atrialized right ventricular chamber (ARV) empties directly into infundibular portion of right ventricle (I).

the lower left sternal border and in 7 a short diastolic murmur was heard at the left lower sternal border. In 1 other patient a diastolic murmur was audible at the apex. In 1 patient a grade 3/6 pan-systolic murmur was present at the apex. The murmur radiated to the axilla. In 1 patient an ejection systolic murmur, grade 3/6, was heard in the pulmonary area.

The electrocardiograms showed complete right bundle branch block in 9 patients with small QRS complexes in lead V_1 in 8 of these patients. In 4 patients the PR interval was prolonged and in 3 of them right atrial enlargement was present. Supraventricular tachycardia occurred in 2 patients. In 1 patient atrial flutter and in another case paroxysmal ventricular tachycardia were present. In only 1 patient the features of the Wolff-Parkinson-White syndrome were observed.

At catheterization, in 7 patients a right-to-left shunt was documented at the atrial level. In 5 patients the pressure was elevated in the right atrium. The right ventricular end diastolic pressure was elevated in 2 patients. In 5 patients the catheter tip was seen on fluoroscopy to be well to the left of the spine in spite of the fact that an "atrial" pressure tracing was recorded. No serious complications were encountered during catheterization or angiographic studies.

ANATOMIC AND FUNCTIONAL CONSIDERATION

The anatomic features of Ebstein's anomaly have been described in detail by several authors.[2,3,6,7,14] The 2 essential features are: (a) (Fig. 1) downward displacement of the origins of the tricuspid leaflets from the annulus into the right ventricle; and (b) dysplasia of 1 or more of the tricuspid leaflets. The downward displacement of the origin of the leaflets varies in severity, the septal leaflet being displaced to a greater extent than the anterior or posterior leaflets.

Dysplasia also usually affects the leaflets asymmetrically. The septal leaflet is most commonly underdeveloped with thickened, stubby valve tissue fixed to the endocardium by shortened, haphazardly inserted chordae tendineae and papillary muscles. The posterior leaflet may exhibit similar changes or it may fuse with the anterior leaflet. In contrast, the anterior leaflet is usually larger than normal, extending like a sheet across the sinus portion of the right ventricle. The body of this leaflet is usually adherent to the free right ventricular wall through a series of abnormal fibrous and muscular bands.

As a result of the abnormal tricuspid valve structure and location, the right ventricle is divided into 3 portions: (1) a proximal inflow portion, located between the tricuspid annulus and the anterior leaflet (the atrialized portion of the right ventricle); (2) a distal sinus portion, between the anterior leaflet and the right ventricular wall; and (3) an outflow or infundibular portion, located distal to the tricuspid valve leaflets and often called the "functioning" right ventricle.

ROENTGENOGRAPHIC FEATURES

The roentgenographic findings have been

The large notch (vertical arrow) indicates true tricuspid annulus. The anterior tricuspid leaflet is marked by horizontal arrow. (*E*) Later phase. The sinus portion of the right ventricle (SRV) has now filled and is separated from the atrialized right ventricle by the displaced anterior leaflet of the tricuspid valve. The pulmonary valve (white arrow) is thickened and the main pulmonary artery (PA) is dilated. The contrast medium filled right atrium is separated from the apparent heart border (arrowheads) indicating pericardial effusion. (*F*) Lateral view of angiocardiogram. Marked dilatation of right atrium and inferior vena cava. The "posterior bulge" seen in (*C*) is due to the left ventricle (LV) which is displaced posteriorly. The notch due to the true tricuspid annulus is indicated by the vertical arrow. Note that inferiorly the retrosternal space is clear.

RAA = Right atrial appendage, ARV = Atrialized right ventricle, I = Infundibular chamber, PA = Main pulmonary artery, SRV = Sinus portion of right ventricle, LV = Left ventricle.

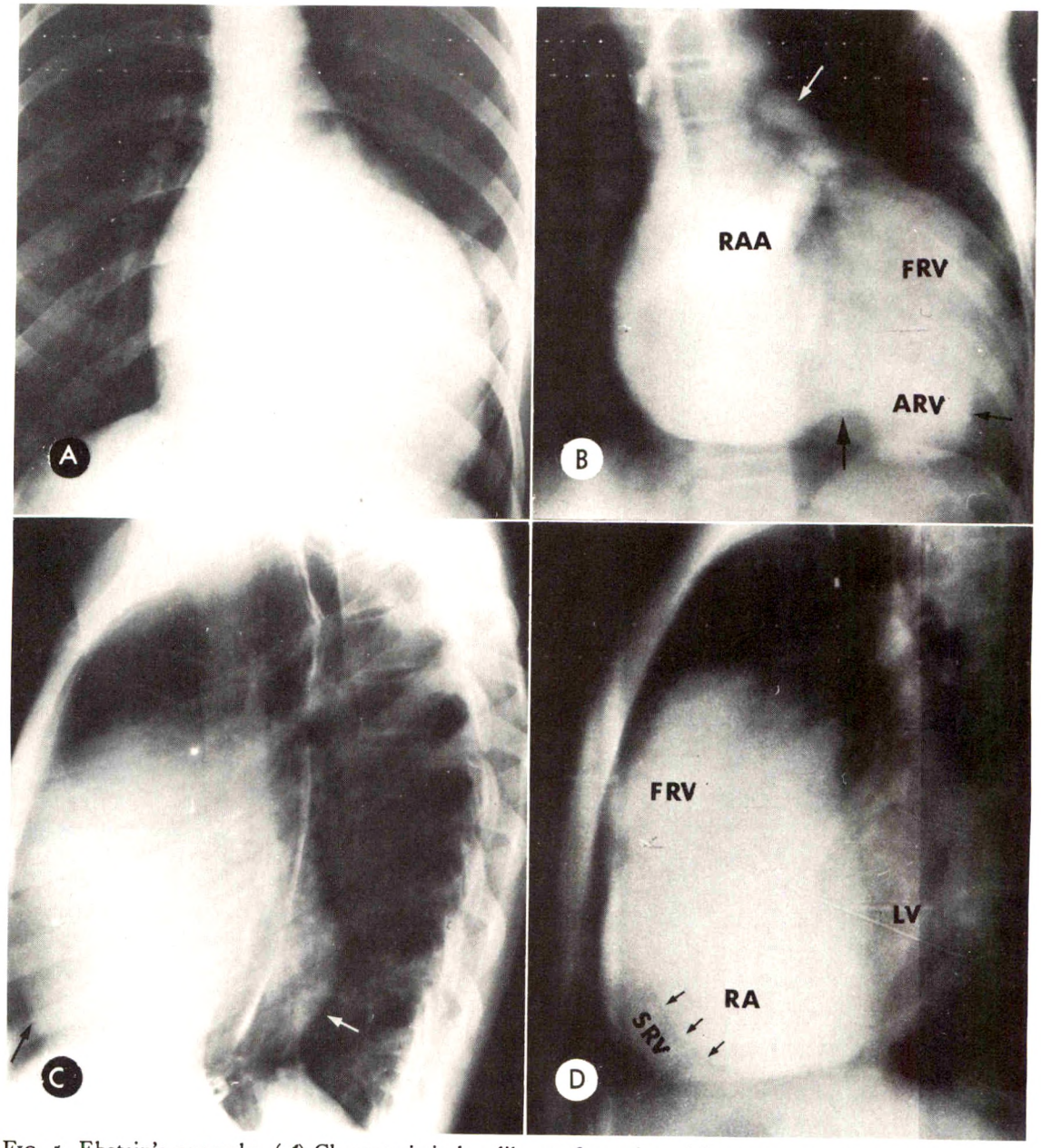


FIG. 5. Ebstein's anomaly. (*A*) Characteristic box-like configuration with narrow vascular pedicle and diminished pulmonary vascularity. (*B*) Angiocardiogram, injection into superior vena cava, frontal view. Enlarged right atrium (RA) with medially placed right atrial appendage (RAA). Large notch (vertical arrow) at site of true tricuspid annulus. Anterior tricuspid leaflet (horizontal arrow) defines limits of atrialized portion of the right ventricle (ARV). The functional right ventricle (FRV) and pulmonary artery (white arrow) have filled but the sinus portion of the right ventricle has not. (*C*) Lateral view of chest with prominent "posterior bulge" (white arrow). The functional right ventricle (compare to FRV in *D*) fills in the upper retrosternal space, but inferiorly the relatively small sinus portion of the right ventricle does not reach the anterior chest wall (black arrow). (*D*) Angiocardiogram, lateral view. The posterior bulge is formed by the left ventricle (LV). The right atrium is superimposed on the right ventricle. The small arrows point to the displaced anterior leaflet of the tricuspid valve.

RA = Right atrium, ARV = Atrialized portion of right ventricle, RAA = Right atrial appendage, LV = Left ventricle, FRV = Functional right ventricle, SRV = Sinus portion of right ventricle.

described by others in detail and vary markedly, depending on the degree of anatomic and hemodynamic abnormality.[1,4,5,10] Heart size ranges from normal to markedly enlarged. In the frontal view the typical findings are a square or box-like cardiac configuration with a narrow vascular pedicle and a small aortic arch segment. The ascending aorta likewise tends to be reduced in size and is not usually visible in the frontal projection.

Previous authors have shown how the enlarged right atrium forms one-half of the box-like cardiac silhouette, while the markedly dilated and horizontally positioned infundibular portion of the right ventricle contributes to the remaining upper corner. In most cases the main pulmonary artery segment is concave and the pulmonary vessels appear normal or decreased in prominence, reflecting the degree of right-to-left shunting and consequent reduction in pulmonary blood flow. This combination of a large box-like heart and decreased pulmonary blood flow has been described as the characteristic plain film findings in Ebstein's anomaly.

Our cases also exhibited these well known findings (Fig. 2*A*; 3, *A* and *B*; 4, *A* and *B*; 5*A*; 6*A*; and 11*A*). A general truism is: The more severe the deformity, the more characteristic the roentgenographic findings. Several of our cases had pericaridal effusion which produced marked apparent cardiomegaly and introduced a more globular configuration, although the basic features were otherwise unchanged (Fig. 3*B*; and 4, *B* and *E*). In our experience, cardiac size and shape tend to remain relatively unchanged in patients who have minimal or no symptoms. However, cardiac enlargement develops in 3 circumstances: cardiac failure; pericardial effusion; and increasing signs of tricuspid insufficiency.

Hanson and Rosenbaum[9] described a "posterior bulge" on the lateral roentgenogram which they attributed to enlargement of the abnormal atrialized portion of the right ventricle. Although we also saw this posterior bulge frequently (Fig. 2*B*; 4*C*; 5*C*; 6*B*; and 8*A*), correlation with lateral angiocardiograms revealed that the atrialized chamber was not responsible for the posterior bulge since it was always situated anterior to the inferior vena cava and was not a posterior border forming structure (Fig. 7, *D* and *E*; and 8*B*). Indeed, we found 2 other structures which were responsible for the posterior bulge: the markedly enlarged right atrium (Fig. 6*D*; and 7*D*) or, alternatively, it was caused by posterior displacement of normal left heart structures (Fig. 4*F*; and 5*D*). When the dilated right atrium is responsible for the posterior bulge in the lateral view, the bulge usually lies below the level of the left atrium (Fig. 6*D*; and 7*E*). However, the dilated right heart structures may cause sufficient posterior displacement of the left atrium or ventricle to produce either a localized or a broad indentation of the esophagus which simulates left atrial (Fig. 8*A*) or combined atrial and ventricular enlargement (Fig. 4, *C* and *F*). In some instances the left ventricle projects behind the esophagus, producing the posterior bulge (Fig. 2*B*; and 5*C*).

The dilated right atrium and inferior vena cava may project behind the esophagus in the right anterior oblique (Fig. 2*D*), as well as in the lateral view (Fig. 6, *B* and *D*). Surprisingly, even though the right atrium is dilated, the squared-off upper right border characteristic of right atrial enlargement in the left anterior oblique view is not commonly seen (Fig. 2*C*). Because the body of the right ventricle is disproportionately small compared to the right atrium, there is a marked clockwise rotation of the heart which particularly affects the right atrium. As a result, the atrial appendage projects farther to the left than is typical of other causes of right atrial enlargement (Fig. 4*D*; 5*B*; 6*C*; and 7*A*) and consequently, it is not a border forming structure in the left anterior oblique view.

Elliott and Hartmann[4] have emphasized

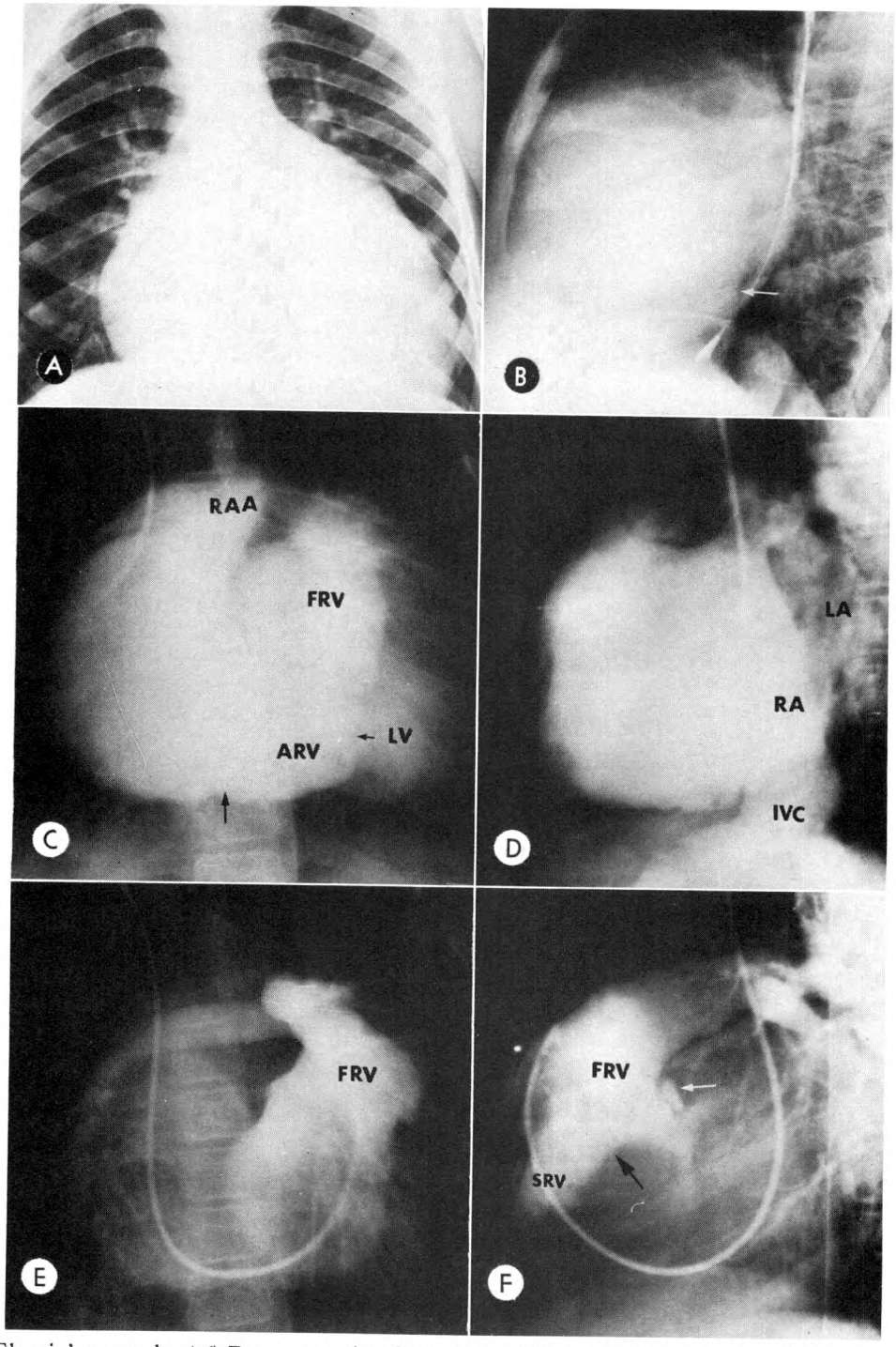


Fig. 6. Ebstein's anomaly. (*A*) Posteroanterior chest roentgenogram. "Square" cardiac silhouette typical of Ebstein's anomaly. Pulmonary vascular pattern is within the range of normal. Superior vena cava shadow is prominent due to elevated right atrial pressure. (*B*) Lateral view. There is a "posterior bulge" behind the esophagus in the region of the left atrium. The inferior vena cava (arrow) is posteriorly located. (*C*) Right atrial injection, anteroposterior view. Marked enlargement of right atrium. The atrialized portion of the right ventricle lies between the tricuspid annulus (vertical arrow) and the displaced anterior leaflet (horizontal arrow). The functional right ventricle (FRV) is large. The left ventricle (LV) has filled via a right-to-left shunt at the atrial level. (*D*) Same injection, lateral view. The enlarged right atrium

the systolic-diastolic variations in size of the infundibular segment of the right ventricle. This segment may be so enlarged that, particularly in diastole, it overlaps the main and small left pulmonary arteries (Fig. 8*F*). The vigorous pulsations of the upper left cardiac border are mirrored by the alternate pulsations of the upper right heart border due to tricuspid insufficiency. These changes are easily observed at fluoroscopy, and may be documented by kymography (Fig. 9). The pulsations of the lower right and left heart borders are usually damped since they reflect the dilated inferior right atrium and the weakly contracting sinus portion of the right ventricle respectively. Other observers have characterized the hearts seen in Ebstein's anomaly to be large and quiet, similar to those seen in cases of pericardial effusion. The alternately pulsating upper borders should assist the fluoroscopist in making this distinction except in those cases of Ebstein's anomaly complicated by superimposed pericardial effusion. Thickening of the anterior pericardial stripe, when present, on the lateral view or at fluoroscopy, may be a helpful sign in identifying superimposed pericardial effusion.

An additional finding which we have observed in several patients with Ebstein's anomaly is a relatively clear lower substernal segment due to hypoplasia of the sinus portion of the right ventricle in conjunction with marked filling-in of the upper retrosternal space, a result of prominence of the infundibular segment of the right ventricle (Fig. 4, *C* and *F*; 5, *C* and *D*; and 8, *A* and *C*). Since the lower sinus portion rarely exhibits dilatation comparable to that of the infundibular portion, this combination of findings on the lateral film may distinguish the more common causes of right heart enlargement which tend to affect all portions of the right ventricle more or less equally.

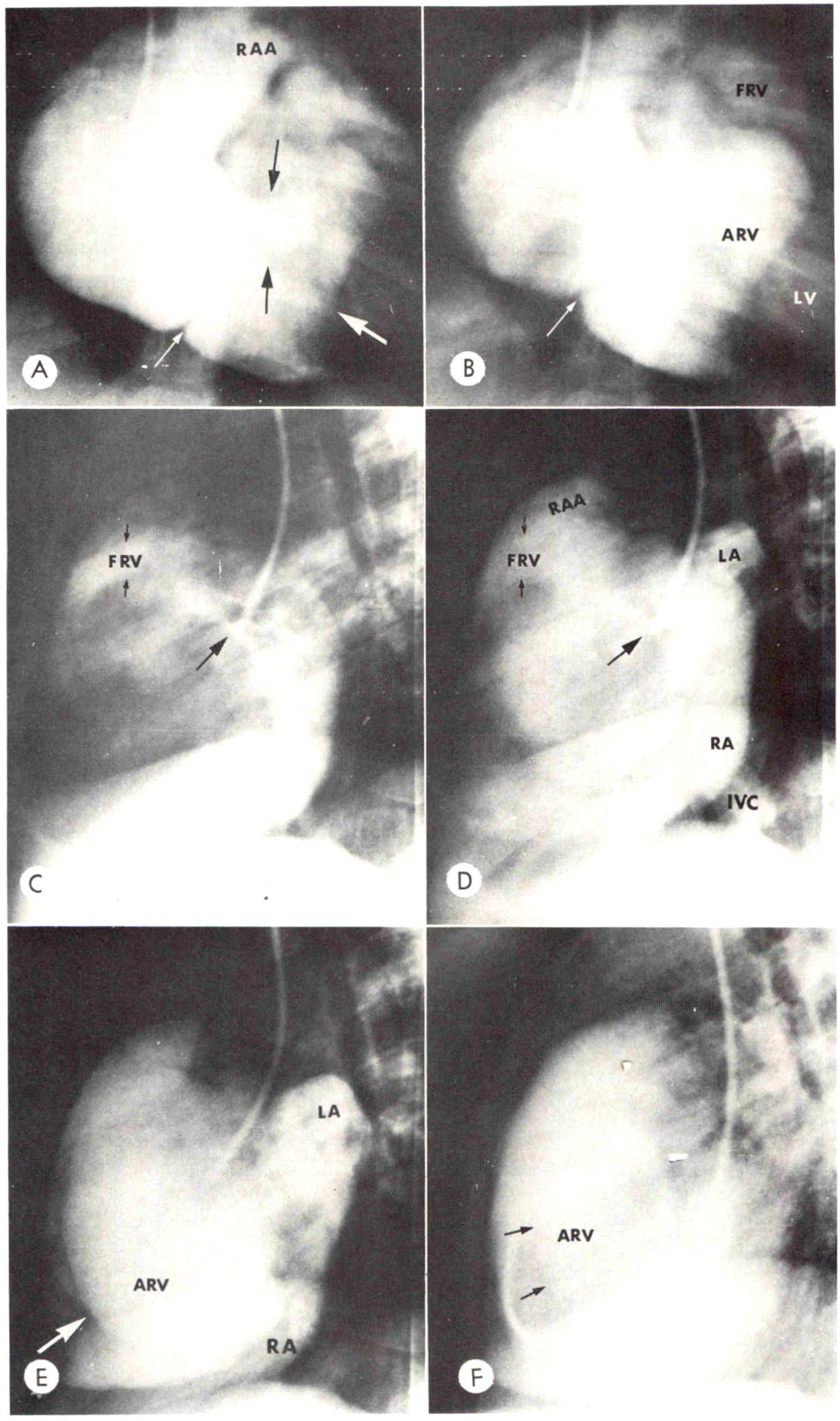
Although the main pulmonary artery segment is characteristically concave, we have observed a case of Ebstein's anomaly with valvular pulmonic stenosis who had poststenotic dilatation of the main pulmonary artery (Fig. 4, *A* and *E*). Additionally, some patients with Ebstein's anomaly have prominence of the superior vena cava in the frontal view (Fig. 3*B*; and 6*A*). The supradiaphragmatic portion of the inferior vena cava is commonly wide due to elevated right atrial pressure and reflux from the right atrium. The enlarged inferior vena cava is better appreciated in the lateral view (Fig. 6*B*).

ANGIOGRAPHIC FEATURES

In the past, the angiographic features of Ebstein's anomaly have not been emphasized. Ellis and co-workers in 1964[6] described the classical angiographic findings in Ebstein's anomaly. Later Elliott and Hartmann[4] discussed in some detail the appearance of the right ventricular infundibulum. Nevertheless, the potential value of angiography has not been stressed. Improved techniques including biplane studies, cine techniques, and the selective injection of contrast medium in the region of the abnormal tricuspid valve or in the infundibular chamber, have increased the value of angiocardiography, particularly since cor-

overlies the right ventricle. Reflux into the widened and posteriorly located inferior vena cava. The left atrium (LA) has opacified, although the site of the atrial shunt is not visible. (*E*) Injection into the functional right ventricle, anteroposterior view. The pulmonary valve and pulmonary vessels can be seen with clarity. There is reflux filling of the atrialized chamber and the right atrium. The anterior leaflet of the tricuspid valve is better defined. The left ventricle is not opacified. (*F*) Same injection, lateral view. The functional portion of the right ventricle (FRV) and the pulmonary valve and main pulmonary artery are defined best in this view. The non-opacified atrialized chamber bulges into the sinus portion simulating a tumor. The black arrow defines the anterior leaflet spreading like a sail across the body of the right ventricle. The white arrow points to a deformity on the septal leaflet.

RAA = Right atrial appendage, FRV = Functional right ventricle, RA = Right atrium, LA = Left atrium, ARV = Atrialized right ventricle, LV = Left ventricle, IVC = Inferior vena cava.


RAA
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rective surgical procedures may be contemplated.

We would like to emphasize some observations based on our material concerning the anatomy of the right atrium and ventricle and the various patterns of filling and emptying of these chambers seen in biplane studies. We have found the lateral view to be an important adjunct and indeed it may be critical in establishing the diagnosis.

Since the septal and posterior leaflets are commonly dysplastic, small, and adherent to adjacent endocardium, they are difficult to define angiographically (Fig. 1; 6*F*; and 7*A*). However, the anterior leaflet billows across the cavity of the right ventricle to virtually obliterate the sinus portion of the ventricle where the leaflet is fixed to the endocardium by abnormal fibrous and muscular attachments (Fig. 2*E*; and 11*C*).

The most valuable angiographic finding, in our experience, is the appearance of the atrialized chamber with its distal limits marked by the smooth contour of the displaced anterior tricuspid leaflet which has a "sail-like" appearance. This is best seen during the early phases of the study before the sinus portion is opacified in both the frontal and lateral projections. Later, a smooth radiolucent arc representing the displaced anterior leaflet is well defined in at least 1 projection (Fig. 2*E*; 4, *D* and *E*; 5, *B* and *D*; 6, *C* and *F*; 7, *A*, *B*, *E* and *F*; 8, *C*, *E* and *F*; and 11*C*). The size and shape of the atrialized chamber vary considerably; in addition, the functional right ventricle may be deformed, small, or occasionally large (Fig. 2*E*; 6*E*; and 7*B*). When the ventricle contracts, 2 notches may be identified; 1 corresponding to the true tricuspid annulus and the other representing the site of attachment of the displaced tricuspid valve. These 2 notches are seen in both frontal and lateral projections (Fig. 4, *E* and *F*; 5*B*; 6*C*; 7, *A* and *B*; and 8, *B–F*). They may be appreciated more readily during cineangiography than serial filming. Cine techniques are also useful to confirm that the atrialized portion actually contracts synchronously with the remainder of the right ventricle.[6] In the presence of marked tricuspid insufficiency, large reciprocal volume changes may occur between the functional right ventricle and right atrium. During the cardiac cycle an extreme degree of volume variation also occurs in the right ventricular infundibulum. The dilated infundibulum may rise above the pulmonary trunk during diastole (Fig. 8*F*). Additionally, the right atrial appendage and the right ventricular infundibulum exhibit reciprocal expansion

Fig. 7. Ebstein's anomaly. (*A*) Angiocardiogram, injection into right atrium, anteroposterior view, systole. Markedly enlarged right atrium with medially displaced right atrial appendage (RAA). All three leaflets of the tricuspid valve can be seen in this exposure. The posterior leaflet (small white arrow) is only slightly displaced from the notch indicating the true tricuspid annulus. The deformed septal leaflet (between black arrows) is not often seen on angiocardiography. The anterior leaflet (large white arrow) is well defined. (*B*) Later diastolic phase. The notch (white arrow) is more evident. The atrialized right ventricle (ARV) is larger while the right atrium is smaller. The functional right ventricle (FRV) is small. The left ventricle (LV) has filled via a right-to-left shunt at the atrial level. (*C*) Lateral view angiocardiogram, beginning injection. Filling of right atrium with streaming of contrast medium into the functional right ventricle (FRV) directly, between the septal and anterior tricuspid leaflets. (*D*) Later phase, same injection. The beginning of right-to-left shunt at the atrial level is now apparent. Further filling of right atrium (RA) and functional right ventricle (FRV). Reflux into inferior vena cava and early filling of atrialized chamber. (*E*) Later phase, same injection. Now the atrialized chamber is opacified and the displaced anterior leaflet is well defined (white arrow). The left atrium (LA) has become completely opacified. (*F*) Injection into functional ventricle, lateral view. The entire right heart has filled. Arrows indicate the displaced anterior leaflet.

RAA = Right atrial appendage, LA = Left atrium, ARV = Atrialized right ventricle, FRV = Functional right ventricle, IVC = Inferior vena cava, LV = Left ventricle.

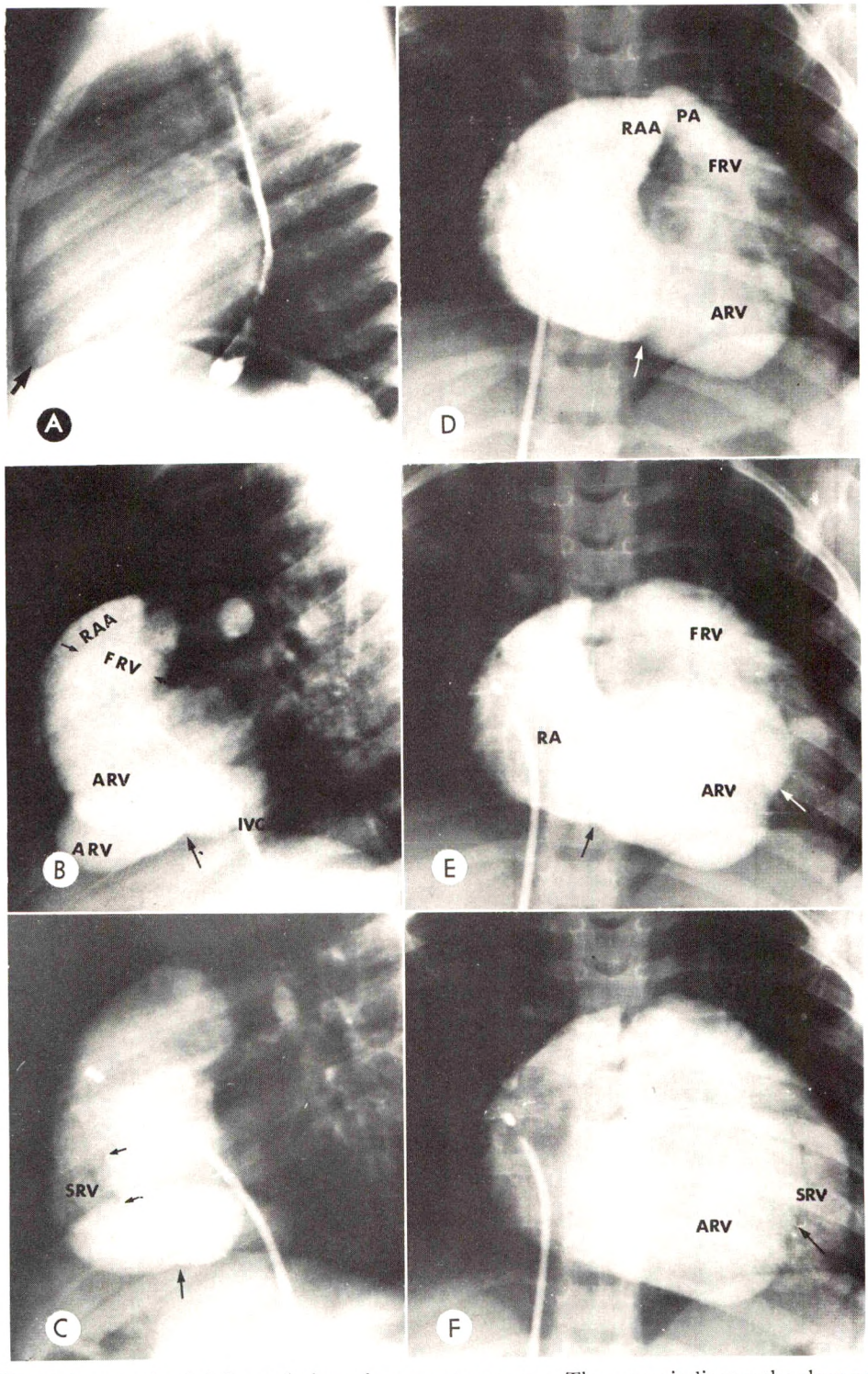

FIG. 8. Ebstein's anomaly. (*A*) Lateral view, chest roentgenogram. The arrow indicates the clear area in the inferior retrosternal space. There is an indentation on the esophagus in the region of the left atrium. (*B*) Angiocardiogram, injection into right atrium, lateral view during atrial diastole. The right atrial appendage (RAA) partially overlaps the functional right ventricular chamber (FRV between small arrows). The tricuspid annulus is indicated by the inferior notch (arrow at the bottom) the sinus portion of the

(Fig. 8, *D* and *F*), the "teeter-totter" phenomenon.[4]

Right-to-left shunting through a patent foramen ovale or a true atrial septal defect is best seen following right atrial or superior vena caval injections, since overlap of the right ventricle is avoided in the early phases of the series. Although shunting may be identified on the frontal view (Fig. 6*C*; and 7*B*), the lateral view affords a better demonstration of the shunt when there is extreme dilatation of right heart structures and clockwise rotation of the heart (Fig. 6*D*; and 7*E*).

An interesting and previously undescribed aspect of Ebstein's anomaly concerns the emptying of the atrialized chamber. When fenestrations exist in the anterior leaflet, there is direct communication between the atrialized and the sinus portions of the right ventricle (Fig. 11*B*). More commonly, however, the sinus portion is initially bypassed and the atrialized chamber empties directly into the infundibular chamber through an opening between the abnormally inserted anterior and septal leaflets (Fig. 7, *C* and *D*). This opening may be so small as to produce functional obstruction to the emptying of the atrialized chamber. After the infundibular functional chamber fills there is retrograde flow into the sinus portion (Fig. 4, *D* and *E*; and 8, *D–F*).

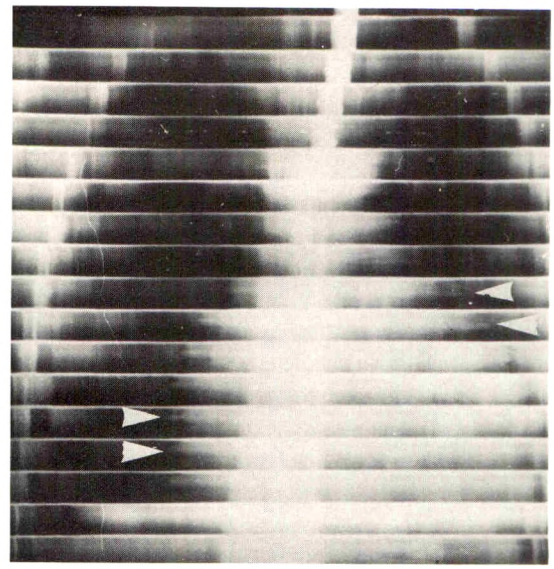

Fig. 9. Ebstein's anomaly. Anteroposterior kymogram. Same patient as in Figure 8. The reciprocal pulsations of the infundibular chamber (left-sided arrowheads) and the right atrium (right-sided arrowheads) are evident. The lower left heart border is quiet since it reflects the weakly contracting sinus portion of the right ventricle. In this case the lower right heart border pulsates vigorously. More commonly, the dilated inferior portion of the right atrium does not change very much between systole and diastole.

The sinus portion of the ventricle will vary in size depending on the degree of displacement of the anterior leaflet and its

right ventricle is not yet opacified. Note that the ARV lies anterior to the IVC and is not a posterior border forming structure. (*C*) Later phase, same injection during ventricular diastole. The sinus portion of the right ventricle (SRV) is now opacified. The horizontal arrows indicate the anterior tricuspid leaflet. (*D*) Angiocardiogram, anteroposterior view. Early frame during ventricular systole. Note medial position of right atrial appendage (RAA). The right atrium empties into the atrialized portion of the right ventricle (ARV) and then directly into the functional chamber (FRV). The arrow indicates the tricuspid annulus. (*E*) Later phase, same injection, ventricular diastole. Both the atrialized and the functional portions of the right ventricle have expanded while the right atrium has contracted. The functional chamber overlaps the pulmonary artery. The reciprocal changes in size between the right atrial appendage and the infundibular chamber create the "teeter-totter" phenomenon (compare with *D*). The white arrow indicates the displaced anterior tricuspid leaflet. The black arrow identifies the tricuspid annulus. The sinus portion of the right ventricle is beginning to fill. (*F*) Still later phase of same injection in ventricular diastole. The dilated functional right ventricle overlaps the small main pulmonary artery. The sinus portion of the right ventricle has filled completely. The anterior tricuspid leaflet (arrow) separates the atrialized portion from the sinus portion of the right ventricle. Compare frames (*D*, *E* and *F*) for sequence of filling of the 3 right ventricular chambers and also for the mirrored alternate pulsation of the dilated infundibulum of the right ventricle and right atrial appendage.

RAA = Right atrial appendage, FRV = Functional right ventricle, RA = Right atrium, SRV = Sinus portion of right ventricle, ARV = Atrialized right ventricle, PA = Pulmonary artery, IVC = Inferior vena cava.

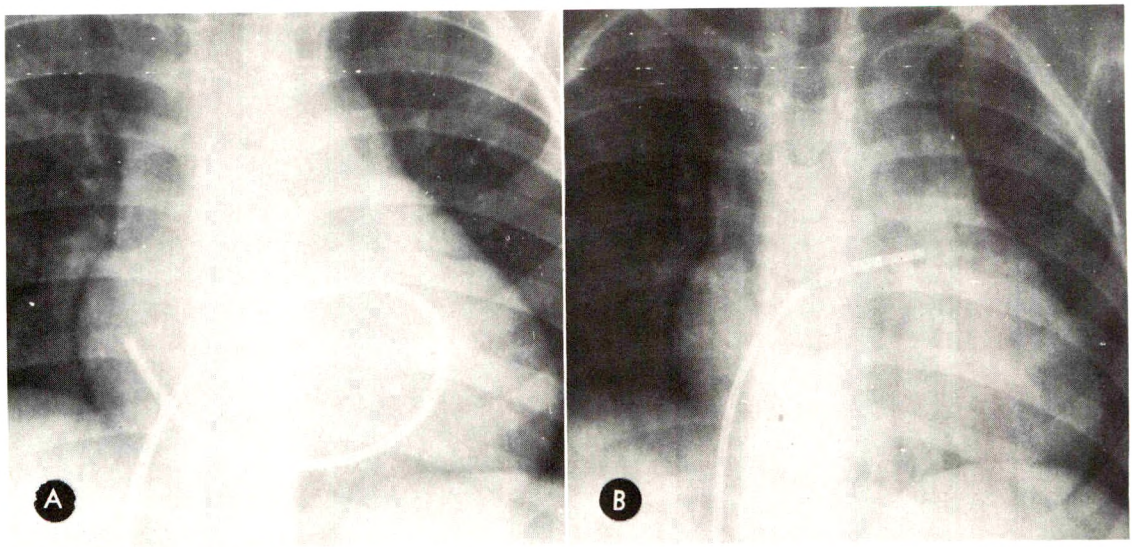

Fig. 10. Ebstein's anomaly. (*A*) The cardiac catheter is looped in the right atrium and the atrialized portion of the right ventricle. The curved portion is against the anterior tricuspid valve leaflet. (*B*) The tip of the catheter is against the septal leaflet of the tricuspid valve. What appears to be a dilated pulmonary artery segment is due to a prominent thymus.

fixation to the free wall of the right ventricle. The size and character of the sinus portion are best seen in the lateral view. When the sinus portion is obliterated by muscular and fibrous bands, it tends to be small in size, especially when compared to the infundibulum (Fig. 2*E*; 5, *B* and *D*; and 6*F*). Thus, on the lateral view, there is a discrepancy between the filling in of the lower versus the upper retrosternal space such that a clear space may exist in front of the sinus portion while the dilated infundibulum impinges upon the sternum anteriorly. This is appreciated both in lateral plain roentgenograms and angiocardiograms (Fig. 5, *C* and *D*).

When a selective injection is made into the functional chamber, the anterior leaflet, with unopacified blood behind it in the atrialized chamber, may bulge into the contrast medium filled sinus portion, particularly when this segment is relatively well formed. This appearance simulates a right atrial tumor bulging into the right ventricle during the early phases of the injection (Fig. 6*F*).

Dilatation of the main pulmonary artery suggests coexistent pulmonic valve stenosis (Fig. 4, *A* and *B*). Since it may be difficult to pass the catheter across the pulmonic valve, the best way to demonstrate the anatomy of the valve is by injection into the distal portion of the ventricle in order to achieve satisfactory visualization without overlap of the right atrium (Fig. 6, *E* and *F*). Apart from 1 case of pulmonic stenosis (Fig. 4, *D* and *E*) and those cases in which right-to-left shunting was present through either a patent foramen ovale or an atrial septal defect, no other associated congenital lesions were present in our cases.

The pulmonary vessels tend to be normal or reduced in size in the presence of right-to-left shunting. The size of the central vessels is also reduced consistent with that of the peripheral vessels so that confusion with the pattern of pulmonary hypertension is not common. Transit time through the lungs is markedly prolonged, not as a result of increased pulmonary resistance or left heart failure but because of the marked tricuspid insufficiency and right heart failure. Visualization of the left heart structures may be poor in the levocardiogram because of the prolonged circulation time.

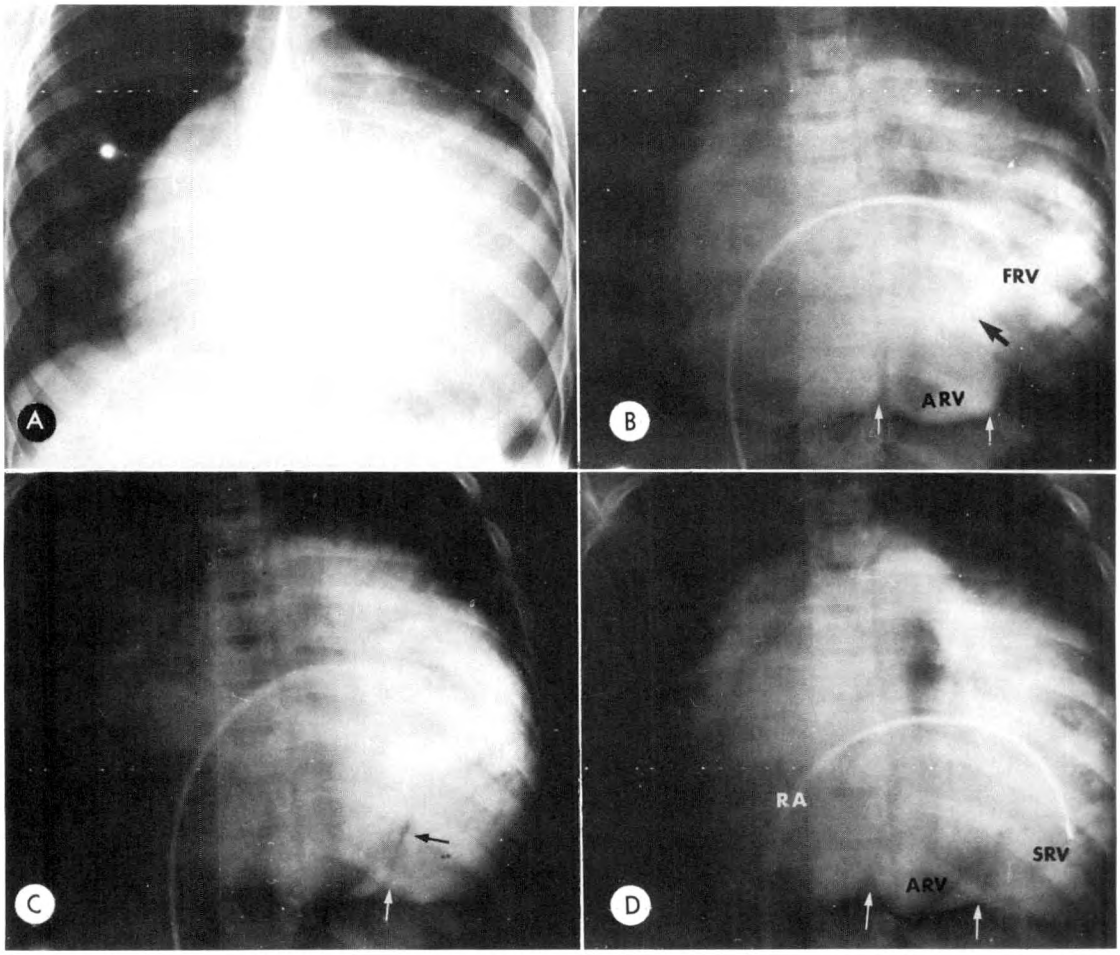

Fig. 11. Ebstein's anomaly. (*A*) Posteroanterior chest roentgenogram. "Characteristic" appearance of box-like cardiac silhouette, narrow vascular pedicle and decreased pulmonary vasculature. (*B*) Angiocardiogram. The catheter begins injection in the functional right ventricular chamber (FRV), and the dye regurgitates in the form of a jet (black arrow) through a fenestrated anterior leaflet into the atrialized right ventricle (ARV). The atrialized right ventricle (ARV) lies between the 2 arrows; the right arrow indicates the notch of the true tricuspid annulus and the left arrow the site of attachment of the displaced anterior leaflet. (*C*) Later phase, same injection. The displaced tricuspid leaflet (arrows) is defined from both sides as the sinus portion fills. There are additional lucent shadows in the sinus portion caused by abnormal muscular bands and chordae running from the free wall and septum to the displaced anterior leaflet. (*D*) Later phase, same injection. The tip of the catheter now lies in the sinus portion of the right ventricle (SRV).

RA = Right atrium, FRV = Functional right ventricle, ARV = Atrialized right ventricle, SRV = Sinus portion of right ventricle.

Pericardial effusion may coexist with Ebstein's anomaly and may be demonstrated angiographically by apparent increase in the right atrial wall thickness (Fig. 4*E*). Since the differential diagnosis includes pericardial effusion, it is important to demonstrate the other anatomic features of Ebstein's anomaly. We have never seen the classic angiographic signs of pericardial tamponade or constrictive pericarditis in our cases of Ebstein's anomaly or in those described in the literature.

In cases in which the murmur of tricuspid insufficiency simulates that of mitral insufficiency, left ventriculography may be the first procedure performed (Fig. 2*F*).

This procedure usually demonstrates a normal or small left ventricle and aorta with the ascending aorta rotated medially due to right heart enlargement. In addition, the position of the right coronary artery in the atrioventricular groove (Fig. 2*F*) may be observed. This defines the position of the true tricuspid annulus. Subsequent comparison with the right heart study facilitates recognition of the displaced leaflets and the double notch sign.[5]

DISCUSSION

Characteristic clinical, electrocardiographic and roentgenographic findings are usually sufficient to establish the diagnosis of Ebstein's anomaly. Careful cardiac catheterization and angiocardiography are of value to confirm the clinical impression and to exclude other surgically correctable cardiac lesions. Earlier reports of catheterization studies of patients with Ebstein's anomaly stressed the susceptibility of these patients to arrhythmias. Indeed, several deaths were reported secondary to those investigations. However, more recent reports of large series of patients and our own experience with 15 patients indicate that complications can be avoided.[5,8,13] Nevertheless, premedication with antiarrhythmic drugs may be indicated. Certainly today, with increased awareness of the risks of catheterization and the immediate availability of cardiac defibrillators in the catheterization laboratory, the safety of the procedure is such that catheterization and angiocardiography are warranted in symptomatic individuals in order to firmly establish the diagnosis of Ebstein's anomaly.

In order to demonstrate the specific anatomic and hemodynamic abnormalities in a given patient, multiple injections of contrast material may be required. Right atrial or caval injection may demonstrate the anatomy of the right heart chambers, the right-to-left shunt, and pericardial effusion. However, injection in the right ventricle may be necessary to see the displaced tricuspid leaflets and to estimate the degree of tricuspid insufficiency. The pulmonary valve may be seen best following injection into the infundibular chamber.

We have found biplane studies to be most useful in defining the abnormal anatomic structures. Often the lateral view demonstrates the double notch, the character of the atrialized chamber, the anatomy and filling of the sinus portion of the ventricle, the right-to-left shunt, and the pulmonic valve better than the frontal view. Our experience with cineangiography of Ebstein's disease is limited but there is no doubt that the hemodynamic features of this condition are better appreciated by cine rather than serial film methods. Precise knowledge of the anatomy of the right ventricle is a necessary prerequisite to any attempt at surgical correction.

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REFERENCES

1. Amplatz, K., Lester, R. G., Schiebler, G. L., Adams, P., Jr., and Anderson, R. C. Roentgenologic features of Ebstein's anomaly of tricuspid valve. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *81*, 788–794.
2. Becker, A. E., Becker, M. J., and Edwards, J. E. Pathologic spectrum of dysplasia of tricuspid valve: features in common with Ebstein's malformation. *A.M.A. Arch. Path.*, 1971, *91*, 167–178.
3. Edwards, J. E. Pathologic features of Ebstein's malformation of tricuspid valve. *Proc. Staff Meet. Mayo Clin.*, 1953, *28*, 89–94.
4. Elliott, L. P., and Hartmann, A. F., Jr. Right ventricular infundibulum in Ebstein's anomaly of tricuspid valve. *Radiology*, 1967, *89*, 649–700.
5. Ellis, K., Griffiths, S. P., Burris, J. O., Ramsay, G. C., and Fleming, R. J. Ebstein's anomaly of tricuspid valve: angiographic considerations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 1338–1352.
6. Fabian, C. E., Mundt, W. P., and Abrams, H. L. Ebstein's anomaly: direct demonstra-

tion of contractile synchrony between two parts of right ventricle. *Invest. Radiol.*, 1966, *1*, 63–68.

7. From, A. H. L., Mazzitello, W. F., Judd, A. S., and Edwards, J. E. Ebstein's malformation of tricuspid valve associated with valvular stenosis and cor triatriatum. *Chest*, 1973, *64*, 248–251.
8. Genton, E., and Blount, S. G., Jr. Spectrum of Ebstein's anomaly. *Am. Heart J.*, 1967, *73*, 395–425.
9. Hanson, D. J., and Rosenbaum, H. D. Posterior cardiac prominence in Ebstein's anomaly simulating mitral disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 1331–1337.
10. Hipona, F. A., and Arthachinta, S. Ebstein's anomaly of tricuspid valve: report of 16 cases and review of literature. *Prog. Cardiovasc. Dis.*, 1965, *7*, 434–448.
11. Kistin, A. D., Evans, J. M., and Brigulio, A. E. Ebstein's anomaly of tricuspid valve: angiocardiographic diagnosis. *Am. Heart J.*, 1955, *50*, 634–640.
12. Schiebler, G. L., Adams, P., Jr., Anderson, R. C., Amplatz, K., and Lester, R. G. Clinical study of twenty-three cases of Ebstein's anomaly of tricuspid valve. *Circulation*, 1959, *19*, 165–187.
13. Schiebler, G. L., Gravenstein, J. S., and Van Mierop, L. H. S. Ebstein's anomaly of tricuspid valve: translation of original description with comments. *Am. J. Cardiol.*, 1968, *22*, 867–873.
14. Watson, H. Natural history of Ebstein's anomaly of tricuspid valve in childhood and adolescence: international co-operative study of 505 cases. *Brit. Heart J.*, 1974, *36*, 417–427.

CONGENITAL AVALVULAR PULMONARY ARTERY AND INFANTILE LOBAR EMPHYSEMA*

A DIAGNOSTIC CORRELATION

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ABSTRACT:

Some cardiovascular lesions may result in tracheobronchial obstruction and many of these will commonly be associated with infantile lobar emphysema (ILE). By contrast a relatively smaller percentage of cases of ILE results from vascular compression.

We offer a new term, "congenital avalvular pulmonary artery (CAPA)," to describe the complex of the absent pulmonary valve and the concomitant marked pulmonary artery dilatation because CAPA is more descriptively inclusive of the abnormality of the artery beyond the valve.

The diagnostic correlation of CAPA and ILE is of considerable importance since the initial presentation of CAPA is usually as ILE and the recognition of a triangular or rounded hilar density should raise suspicion for CAPA even before clinical signs become evident.

Differential diagnosis must include other causes of ILE including infantile lobar emphysema with bronchial atresia.

CONGENITAL absence of the pulmonary valve or congenital avalvular pulmonary artery (CAPA) is a well recognized entity and its relationship to simple and complex cardiovascular anomalies is well documented.[17,21,23,27] Similarly well documented are the multiple etiologies of infantile lobar emphysema (ILE).[10,14,20,25,30] Commonly, ILE may herald the presence of CAPA. It is the purpose of this paper to correlate the diagnostic aspects of CAPA and ILE from the perspective of a review of the literature and 3 case reports.

REPORT OF CASES

CASE 1. M.S. (Strong Memorial Hospital (SMH) #65-50-33), a girl infant of a gravida 2, para 2, 19 year old mother, was delivered by footling breech at Rochester General Hospital and weighed 3,280 grams. Dyspnea and cyanosis were evident and systolic and diastolic murmurs were auscultated. An anteroposterior chest roentgenogram (Fig. 1*A*) 45 minutes after birth showed a shift of the heart toward the left, diffuse, hazy aveolar and interstitial opacification of the right lung, and a central angular density at the right hilum. Digitalization was begun at 2 hours of age, but was discontinued because of arrhythmia with occasional 2:1 heart block on the third day of life.

Because of suspected cardiac disease the infant was transferred at 9 days of age to Strong Memorial Hospital (SMH) and on admission tachycardia (pulse: 168 per minute) and dyspnea (respiration: 60 per minute) with mild substernal retractions were present. Breath sounds were poorly heard on the left and were coarse on the right. A Grade III/VI diastolic murmur was heard in the left second interspace and a Grade IV/VI systolic murmur was heard over the entire percordium but loudest at the apex. There was no first heart sound but a single second sound was heard at the left lower sternal border. The systolic blood pressure was 86 mm. of Hg in both arms. An anteroposterior chest roentgenogram (Fig. 1*B*) clearly demonstrated emphysema of the right middle lobe and secondary right upper and lower lobe atelectasis. A central right hilar density was present.

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Over the next few days there were periodic bouts of respiratory distress, particularly with feeding. On the tenth day of life a right thoracotomy was performed and the upper and lower lobes were found to be compressed and displaced by the overexpanded right middle lobe. The middle lobe showed minimal air exchange and was resected. Although the specimen showed gross and microscopic emphysema, normal cartilaginous development was noted in the resected bronchial segment. The immediate postoperative course was characterized by improvement in the respiratory studies.

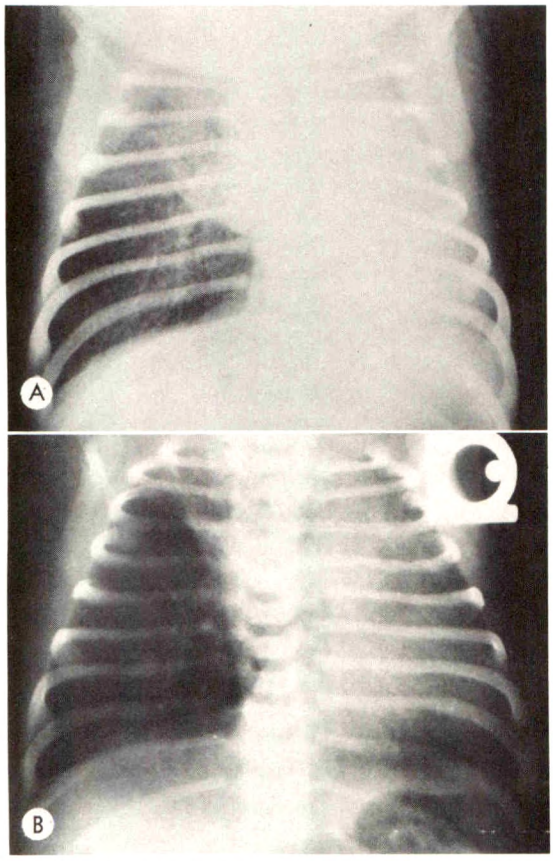

Fig. 1. Case 1. (*A*) Anteroposterior chest roentgenogram at 45 minutes of life. Heart and mediastinum are shifted to the left. A generalized hazy opacification (aveolar and interstitial) of the right lung and a large angular density at the center of the right hilum are present. (*B*) Anteroposterior chest roentgenogram at 9 days of age. Retained fluid of the right lung field has cleared. Cardiac and mediastinal displacement persist. Right middle lobe emphysema and right upper and lower lobe atelectasis are evident. The central hilar density is still present.

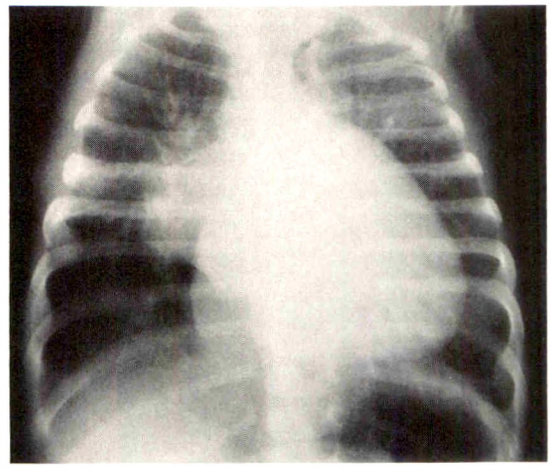

Fig. 2. Case 1. Anteroposterior chest roentgenogram approximately 2 weeks post right middle lobectomy. Note the massive right pulmonary artery, the enlarged heart, and central vascular engorgement. Right lower lobe emphysema is now present.

On the 23rd day of life the infant became dusky and showed substernal retractions and dyspnea (respiration: 60 per minute). A chest roentgenogram (Fig. 2) demonstrated right lower lobe emphysema and a large central hilar density apparently representing an aneurysmally dilated right pulmonary artery.

Cardiac catheterization revealed an oxygen step-up in the right ventricle and systemic pressures. Right atrial pressure was normal. Angiocardiography showed dilated main and right pulmonary arteries (Fig. 3*A*), ventricular septal defect (VSD), and pulmonary insufficiency. The hugely dilated pulmonary artery compressed the right lower lobe bronchus as well as the right atrium. Intermittent tachypnea and cyanosis followed, and during another episode of respiratory distress the patient succumbed at age 73 days.

At postmortem examination (limited to the heart and lungs) an overexpanded right lower lobe was noted with pleural surface blebs and intra-alveolar hemorrhage. Although chronic inflammatory changes in the right main bronchus were noted, there was no evidence of bronchial cartilage abnormality. In the heart there was a small VSD high in position with enlarged right cardiac chambers and normal left chambers. Miniscule tags of tissue were present, apparently remnants of pulmonary valve leaflets (Fig. 3*B*) and the main and right pulmonary arteries were markedly dilated.

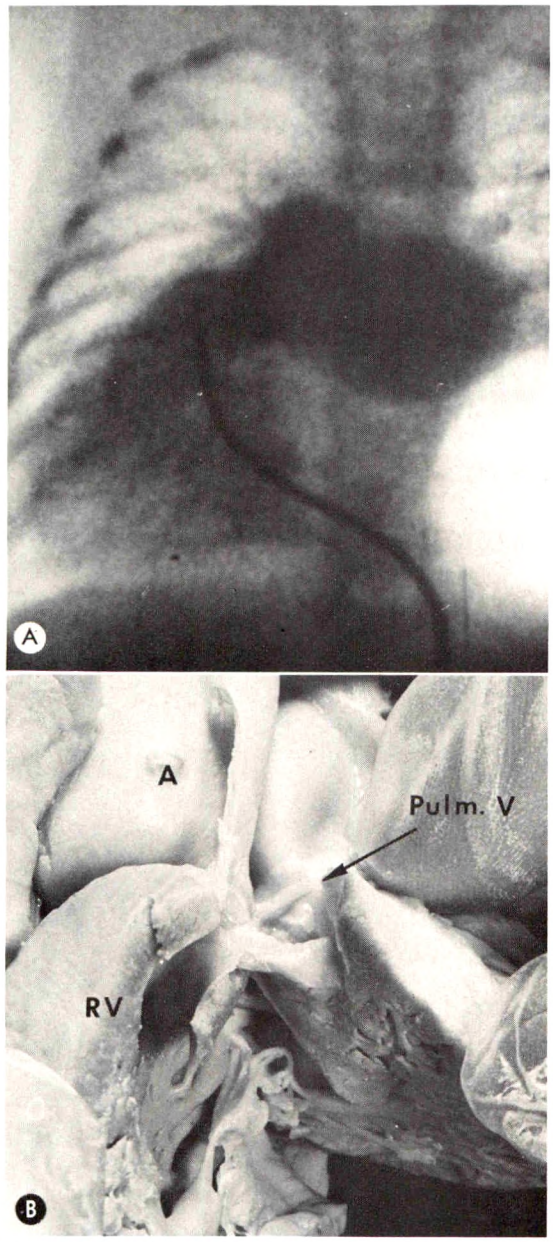

FIG. 3. Case 1. (*A*) Anteroposterior view of frame from cine angiocardiogram at 23 days of life. Note marked dilatation of main and right pulmonary arteries. (*B*) Postmortem photograph of heart sectioned to reveal the pulmonic valve. The pulmonary valve leaflets are replaced by a thin band of tissue with no commissurae. Note also the thickened right ventricular wall.

These are the findings of congenital avalvular pulmonary artery (CAPA).

Comment. The neonatal presentation of a unilateral opaque lung associated with cardiac and mediastinal contralateral displacement is an often observed phenomenon of infantile lobar emphysema in the neonate.[12] Its occurrence in this infant indicates that the phenomenon may occur both in the intrinsic or idiopathic form of ILE as well as in the extrinsic compression form, specifically CAPA in this case.

Despite the removal of the emphysematous right middle lobe and transient abatement of tachypnea, the aneurysmal dilatation of the main and right pulmonary arteries compressed the residual lower lobe bronchus and caused development of emphysema of the lower lobe.

CASE II. M.R. (SMH #84-27-15.) This 4,400 gm. male infant was vaginally delivered at term following a normal pregnancy of his gravida 4, para 2 with 1 abortion, 30 year old mother.

During the second day of life the infant developed tachypnea and a Grade III/VI to and fro murmur along the right upper sternal border. An electrocardiogram revealed abnormally notched p-waves. Anteroposterior and lateral chest roentgenograms (Fig. 4, *A* and *B*) revealed apparent dextrocardia, a very enlarged left pulmonary artery, and marked hyperaeration of the left upper lobe. His pulse was 160–180 per minute, and acrocyanosis of the hands and feet was noted. Blood pressure in all extremities was normal (90/60).

On the fifth day of life cardiac catheterization and cardiac angiocardiography showed normal ventricular and great vessel relationships in the dextroposed heart. A small VSD, high in position with simultaneous filling of the aorta and pulmonary arteries and with aneurysmal dilatation of the left pulmonary artery was present (Fig. 5, *A* and *B*). There was an approximate 50 mm. Hg gradient between the right ventricle and pulmonary artery. These findings were consistent with tetralogy of Fallot with congenital avalvular pulmonary artery (CAPA). Soon thereafter, the infant began experiencing periodic cyanotic episodes. Propanalol reduced the frequency of these episodes without affecting their severity.

Five days following the infant's discharge, at 4 weeks of age, he was again hospitalized with increasing, severe and frequent cyanotic spells. These were controlled with changes in his Propanalol dosage. Throughout this interval the chest roentgenograms were not significantly changed although there was a suggestion of

slight decrease in the left upper lobe hyperaeration.

At $4\frac{1}{2}$ months of age, following several hospital admissions for control of the cyanotic spells, the infant suffered another severe cyanotic episode and was pronounced dead on arrival at the emergency department.

At autopsy, the pulmonary annulus was stenosed with absence of valve leaflets. The aorta overrode a high posterior membranous VSD subadjacent to the posterior mitral leaflet.

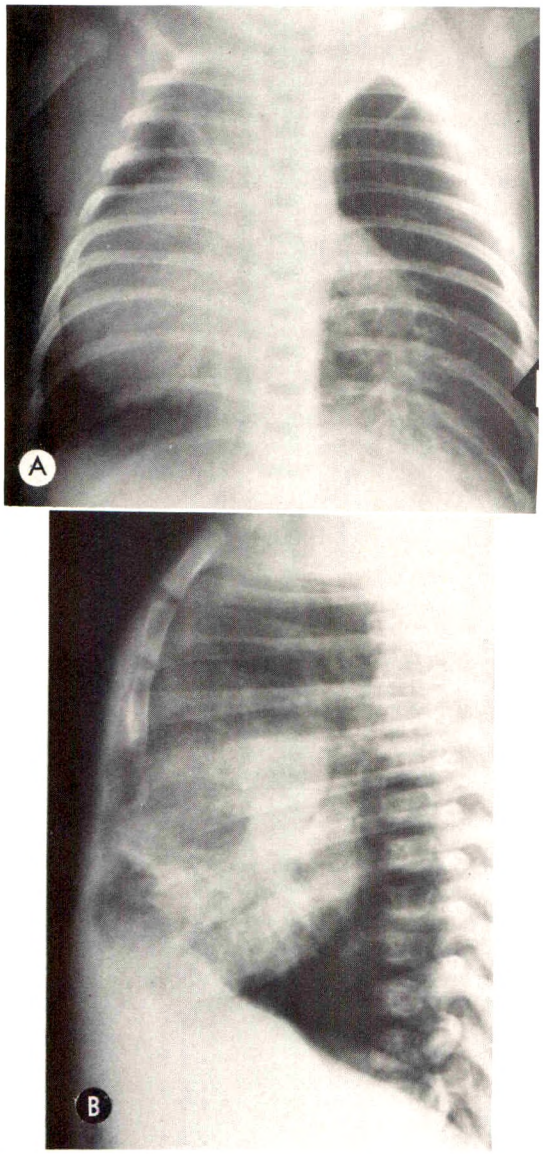

FIG. 4. Case II. (*A*) Anteroposterior and (*B*) left lateral chest roentgenograms at 48 hours of age. Dextrocardia, huge left pulmonary artery and left upper lobe emphysema are demonstrated.

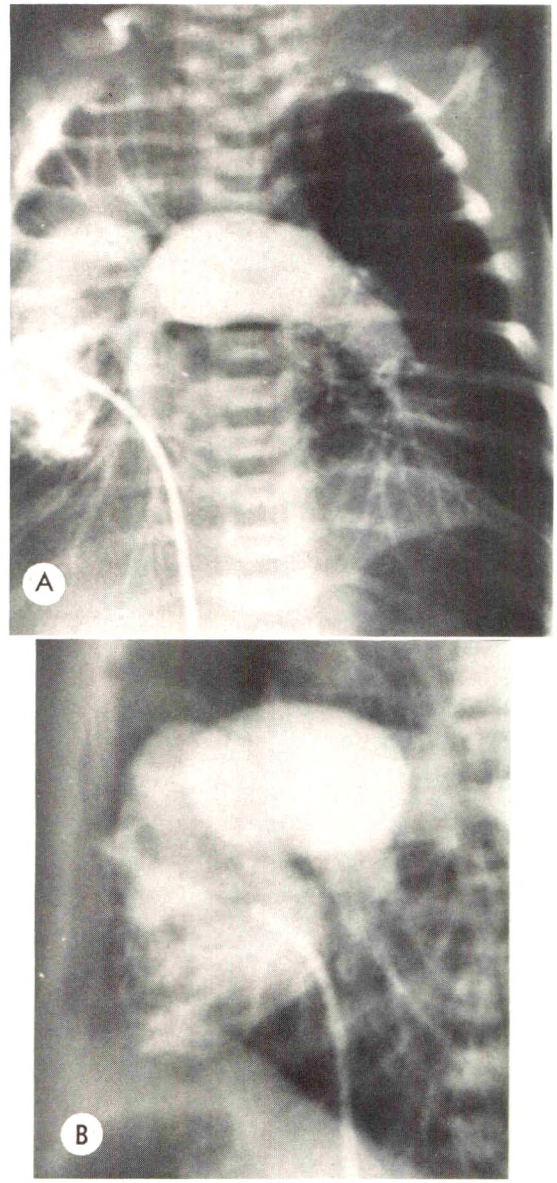

FIG. 5. Case II. (*A*) Anteroposterior and (*B*) lateral roentgenograms from cardiac angiogram show dilated main and left pulmonary arteries. There are hyperaeration and apparent hypovascularization of the left upper lobe.

The pulmonary artery was dilated (4 cm. in circumference). The media showed a marked decrease in elastic fibers. The right ventricular wall was thickened at the infundibulum; however, there was no stenosis. Hyperinflation of the left upper lobe secondary to compression of the left main bronchus was noted, and the cartilage of the left upper lobe bronchus was normal both grossly and microscopically.

Comment. We do not know why this patient had such a dextroposed heart, since it does not appear that the lobar emphysema and the dilated pulmonary artery were severe enough to account for dextrocardia. We note that Contro and associates'[8] first case was almost identical to this patient and apparently was also a dextroposition type of dextrocardia (dextrocardia without chamber inversion).

The roentgenographic appearance of left upper lobe emphysema and a triangular density in the left hilum may also be caused by bronchial atresia, bronchocele, and left upper lobe hyperaeration,[13] and consequently that complex may be an important differential diagnostic consideration.

CASE III. J.P. (SMH #78-87-71.) This 3,384 gm. male infant, the term product of an uneventful pregnancy and delivery of a gravida 2, para 2, 36 year old, woman, was born at a hospital in Watertown, New York. A Grade III/VI systolic heart murmur heard best at the left sternal border was discovered soon after birth. An electrocardiogram obtained at that time revealed extreme right axis deviation.

At the time of the first well baby examination the murmur was louder and the infant was referred to SMH. The infant was small, moderately well nourished, and without cyanosis. A Grade III/VI systolic ejection murmur was heard at the left sternal border with radiation to the left axilla and to the back and there was an absent pulmonary component of the second heart sound. The liver was palpable to 3 cm. below the right costal margin. An electrocardiogram demonstrated an axis of −70 to −75 degrees with a Wolff-Parkinson-White conduction defect. The heart was normal in size, and pulmonary vascularity was normal as evidenced by the chest roentgenogram. The impression was that he had a "precyanotic" form of tetralogy of Fallot.

Subsequently, at 5 months of age, he was mildly cyanotic at rest and a to and fro murmur was present. There was normal atrioventricular conduction. A chest roentgenogram obtained during his hospital admission at 9 months of age, revealed moderate cardiomegaly with mild upper lung congestion. The right lower lung appeared hypovascular.

At 12 months of age he developed left upper lobe pneumonia, right upper lobe atelectasis, and hyperaeration of the right middle and lower lobes. He appeared chronically ill and was below the third percentile for head circumference and height. He was treated for pneumonia which cleared, and 3 weeks later chest roentgenograms with barium in the esophagus (Fig. 6, *A* and *B*) showed hyperlucency of the right lower lobe and right middle lobe, and an enlarged right hilar shadow was present.

Cardiac catheterization and angiography demonstrated a VSD with 50 per cent left to right shunt, and right ventricular pressure at systemic levels. No pulmonary valve leaflets were evident, and aneurysmal dilatation of the proximal right pulmonary artery as well as mild left pulmonary artery dilatation were present (Fig. 6, *C* and *D*). Marked narrowing of the main pulmonary artery just proximal to the bifurcation and significant narrowing of the pulmonary outflow tract at the pulmonary annulus were seen.

For the next 2 years he fared rather poorly with frequent "bronchitic" and wheezing episodes requiring hospitalizations. He was cyanotic but had not been in clinical congestive failure during these episodes. At 2½ years of age he was transferred to SMH for therapy of a lower respiratory tract infection. Hyperaeration of the right middle lobe, an enlarged right pulmonary artery and cardiomegaly were evident on an admission chest roentgenogram.

He was discharged shortly thereafter only to be readmitted 1 month later for a second cardiac catheterization. At this time a bidirectional VSD was present. Angiography prior to total intracardiac repair of the tetralogy of Fallot revealed an adequate pulmonary infundibulum and there was no significant change as compared to the first catheterization.

At surgery, his main pulmonary artery was normal but the right pulmonary artery was 3 times normal size. The right ventricle was hypertrophied and a 1.6 cm. VSD was present. There were small "nubbins" of tissue between which was a small commissure in the region of the pulmonary valve, and the annulus was 1 cm. at its widest point. He withstood the surgery well and prior to discharge there was a noticeable decrease of the right middle lobar emphysema. He is currently being followed as an outpatient by the pediatric cardiology group.

Comment. Why this child failed to manifest lobar emphysema earlier in infancy is somewhat of an enigma and one may only speculate as to the possibilities. Perhaps the pulmonary annular narrowing and the more distal main pulmonary artery stenosis delayed a hemodynamic

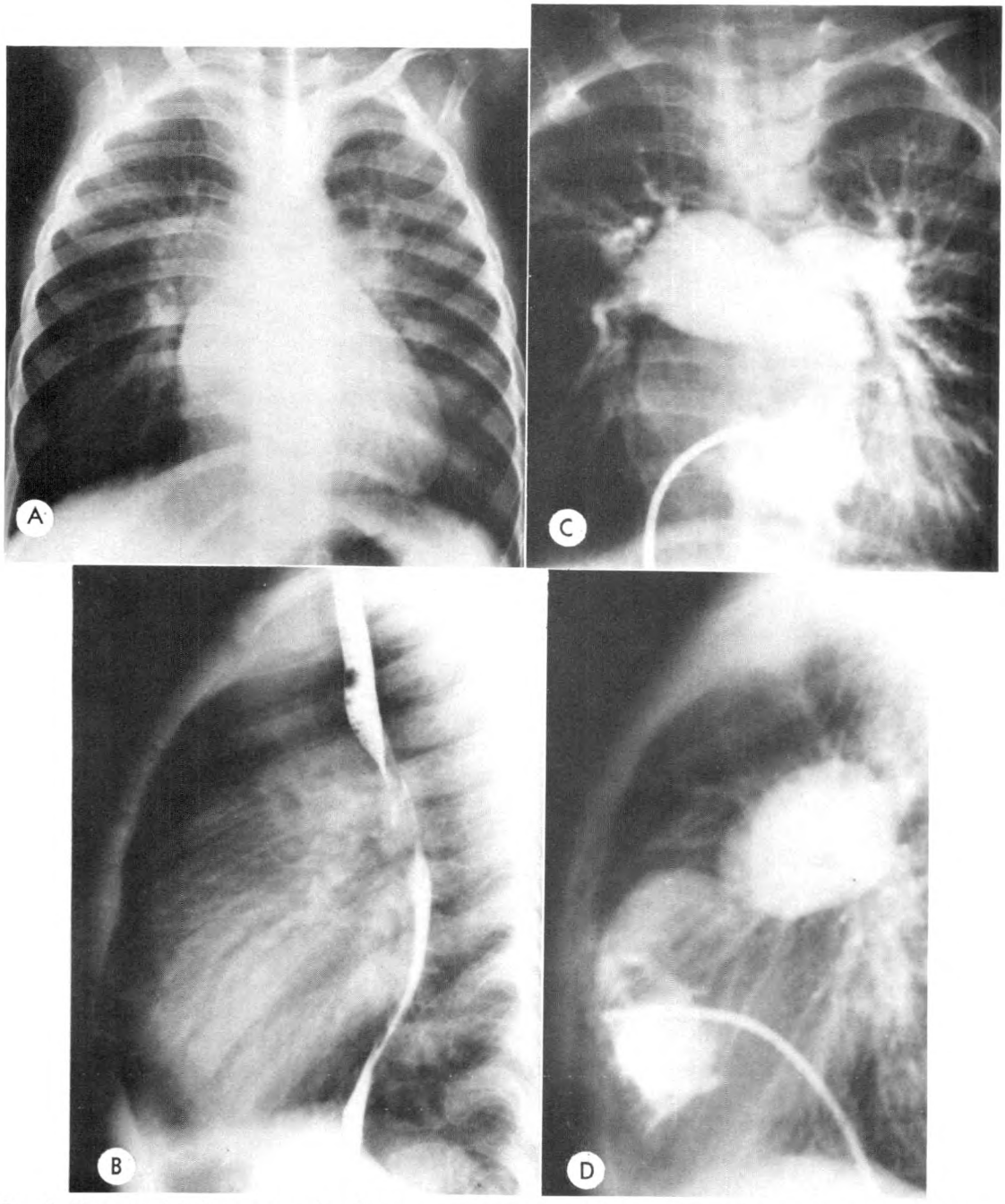

Fig. 6. Case III. (*A*) Anteroposterior chest roentgenogram with barium in esophagus at 12 months of age. There is hyperlucency of the right lower lobe and right middle lobe. An enlarged right hilar vascular shadow is present. There is vascular congestion in the left upper lobe without emphysema. (*B*) Left lateral chest roentgenogram with barium in esophagus. Note indentation upon and displacement of the esophagus by the pulmonary arteries and the heart. (*C*) Anteroposterior view of cardiac angiogram (12 months of age). Note dilated pulmonary arteries. (*D*) Left lateral view of cardiac angiogram (12 months of age). Note the absence of the pulmonary valve leaflets along with the huge central pulmonary arteries.

state which would have otherwise intervened. This would have resulted in the usual early manifestation of pulmonary artery dilatation and subsequent compression of the bronchus. Another possibility is that miniscule valve leaflets despite their anatomic minuteness, might have been enough of an impediment to flow to temporarily forestall the more usual early pulmonary artery dilatation. Progression from a normal appearance of aeration to one of frank emphysema seems to be more frequent with extrinsic causations of infantile lobar emphysema (ILE).

DISCUSSION

Congenital avalvular pulmonary artery (CAPA): Absence of the pulmonary valve leaflets was first reported in 1847 by Chevers[6] and was subsequently definitively described by Kurtz *et al.*[17] in 1927. Since that time there have been over 70 cases reported in the literature.[9,21,23,27,34] This entity occurs either as an isolated defect or, more commonly, associated with other cardiovascular anomalies, usually ventricular septal defect or tetralogy of Fallot.[2,19,34] In 1965 Ruttenberg *et al.*[27] reviewed 38 cases of absence of the pulmonary valve leaflets, 27 of which had associated VSD with 20 of the 27 also having tetralogy of Fallot.

Other associated cardiovascular anomalies that have been described with absence of the pulmonary valve include patent ductus arteriosus,[23] single coronary artery,[4] common atrioventricular canal, tricuspid atresia, mitral atresia, double-outlet right ventricle,[9] and aneurysm of the sinus of Valsalva.[7]

Embryologically, CAPA may be due to an unequal division of the truncus arteriosus as suggested by Campeau *et al.*[5] Simonds[28] earlier suggested that maldirection of the aortic septum resulted in improper division of the endocardial cushion, the precursor of the pulmonary cusps. VSD, overriding aorta, and pulmonary stenosis may also result from abnormal position of the aortic septum. This cardiovascular developmental malformation which occurs approximately in the 7th week of gestation (the period of truncal division), does not appear to be directly the cause for either the pulmonary artery dilatation or the bronchial obstruction.

Miller *et al.*[19] suggested an associated muscular weakness at the base of the pulmonary artery to account for the dilatation seen in infants. Subsequently, Ruttenberg *et al.*[27] reported 2 cases of CAPA with tetralogy of Fallot with 1 of these patients having a mosaic pattern of the elastic fibers of the pulmonary trunk. Elastic fiber disruption or absent pulmonary artery media —as in our third case—has also been described. This appears to support the possibility of primary arterial wall weakness to account in part for the aneurysmal dilatation of the pulmonary artery.

The mechanism of primary arterial weakness *versus* a hemodynamic etiology is an intriguing problem. Barger *et al.*[1] destroyed the pulmonary valves in dogs and subsequently found no decrease in work tolerance or arterial dilatation up to 3 months following surgery. This observation appears to indicate that acquired pulmonary insufficiency alone does not contribute to pulmonary artery dilatation. Others have suggested that a rather precarious situation exists whereby isolated pulmonary valve insufficiency alone may not result in cardiac decompensation. However, in complicated conditions with prolonged increase of pressure, flow increase resulting from left to right shunting or complex cardiac disease may well result in cardiac failure.[11] However, the neonate with absence of the pulmonary valve does develop aneurysmal dilatation of the artery. This perhaps is related to the high pulmonary resistance *in utero* and at birth combined with high volume secondary to VSD, PDA, or both, contributing to right ventricular strain, with ultimate cardiac decompensation. The presence of infundibular stenosis is also a factor and may contribute to poststenotic dilatation of the pulmonary artery.[33]

We have chosen to label the entity of absence of the pulmonary valve and the concomitant pulmonary artery dilatation as congenital avalvular pulmonary artery

(CAPA). This more aptly describes the valvular malformation and the pulmonary arterial changes that invariably are present or develop.

Infantile lobar emphysema and CAPA: Since the first case of infantile lobar emphysema was noted by Bartholinus in 1687 (cited by Koontz[16] in 1925) scores of such cases have been reported.[10,14,20,24,30] Fourteen per cent of these patients have associated cardiovascular anomalies, usually ventricular septal defect or patient ductus arteriosus.[20] A few patients present concomitantly with complex cardiovascular disease, which includes congenital avalvular pulmonary artery (CAPA), and nearly half of these have tetralogy of Fallot. All of our cases have tetralogy of Fallot.

Several authors during the past 2 decades have addressed themselves to the question of the etiology of infantile lobar emphysema (ILE). Almost half of the reported cases to date have been classified as being idiopathic. Half of Hendren and McKee's[14] 113 cases, and more than a third (38 per cent) of Stovin's[30] 45 cases fall into this grouping. Other authors, Campbell[3] and Lincoln *et al.*,[18] have found a greater than expected incidence of bronchial dysplasia. One hundred per cent of Campbell's 12 cases and 78 per cent of Lincoln *et al.'s* 28 patients had pathologically demonstrated bronchial abnormality. Campbell found either small amounts of bronchial cartilage abnormal for the patient's age or increased bronchial flaccidity. He suggested, as did Jones *et al.*[15] and Stovin,[30] that with more proximal resection of the involved bronchial branches a greater number of cases of ILE may be found to have abnormal bronchial cartilage.

Third in order of frequency in most series[14,20] is the category of intrinsic bronchial obstruction, a manifestation of mucus plugging or intrabronchial mucosal hypertrophy.

Finally, as demonstrated by our 3 cases, extrinsic bronchial compression has been defined as a mechanism for bronchial obstruction. Eight per cent of patients with ILE will have extrinsic lesions according to Hendren and McKee[14] and Murray.[20]

Extrinsic compression of the tracheobronchial tree is usually the result of cardiac abnormality or vascular anomaly. Specific areas of the tracheal and bronchial tree are particularly susceptible to compression by abnormally large or aberrant adjacent vascular structures and these have been well described.[22,29] If compression occurs early enough *in utero*, such as would be the case with the dilated main pulmonary artery of CAPA, and prior to development of cartilage in the tracheobronchial tree at about the 12th week of fetal development,[32] then it is conceivable that a combination of extrinsic compression and intrinsic cartilaginous abnormality could exist. In CAPA the extrinsic compression is commonly at the trachea or either main bronchus.[22]

Those congenital heart lesions causing pulmonary artery enlargement are particularly associated with development of lobar emphysema, predominantly right middle lobe and left upper lobe. In addition to these is the vascular sling (anomalous origin of the left pulmonary artery from the right side of the main pulmonary artery). From this anomalous origin the vessel passes anterior to the right bronchus and courses behind the trachea and in front of the esophagus.[24] Robertson and James[26] reported aneurysmal dilatation of a right pulmonary vein in a 9 month old infant in association with lobar emphysema. Lincoln *et al.*[18] mention a 2 week old infant with left upper lobar emphysema secondary to a dilated azygos vein, the azygos continuation of the left inferior vena cava, arching over and compressing the left main bronchus.

Contro *et al.*[8] described 2 cases of pulmonary artery aneurysm encircling the right main bronchus and trachea as compressing lesions. Although the authors do not mention pulmonary valve leaflets, we wonder if these might be CAPA and we are especially intrigued by the similarity of presentation of Contro and co-worker's first case and our second case.

The association of ILE and CAPA is

probably more frequent than the literature indicates, since this presentation or complication may have inadvertently not been mentioned. For example, note Figure 2*C* in the paper of Pernot *et al.*[23] in which right lobar emphysema is evident. We suspect that many cases of CAPA in infancy will have either clinical or roentgenologic manifestations of lobar emphysema and submit that it is important that this be recognized since the management of the patient may be significantly affected by its presence.

Differential diagnosis includes other causes of ILE, but the presence of a large central hilar density should raise suspicion for CAPA. There can be a striking similarity of ILE secondary to CAPA and ILE associated with bronchial atresia,[31] since both may show an ipsilateral hilar mass density. The patients with bronchial atresia are usually asymptomatic in early childhood but presentation in infancy has been reported.[31] When they present, as usual, in mid or late childhood or as adults, bronchography should be a part of their work-up, and no bronchial branch filling takes place beyond the atresia. Bronchography is seldom necessary and is probably contraindicated in lobar emphysema of infancy but if employed should show a patent bronchial tree.

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We thank Drs. Chloe Alexson and Elliot Lipchik for their advice and Mrs. Margaret Kowaluk and Mrs. Victoria Bell for their assistance in the preparation of this manuscript.

REFERENCES

1. BARGER, A. C., ROE, B. B., and RICHARDSON, G. S. Relation of valvular lesions and of exercise to auricular pressure, work tolerance, and to development of chronic, congestive failure in dogs. *Am. J. Physiol.*, 1952, *169*, 384–399.
2. BOVE, E. L., SHAHER, R. M., ALLEY, R., and MCKNEALLY, M. Tetralogy of Fallot with absent pulmonary artery: report of two cases presenting with obstructive lung disease. *J. Pediat.*, 1973, *81*, 339–343.
3. CAMPBELL, P. E. Congenital lobar emphysema. *Australian Paediat. J.*, 1969, *5*, 226–233.
4. CAMPEAU, L., GILBERT, G., and AERICHIDE, N. Absence of pulmonary valve: report of two cases associated with other congenital lesions. *Am. J. Cardiol.*, 1961, *8*, 113–124.
5. CAMPBEAU, L. A., RUBLE, P. E., and COOKSEY, W. B. Congenital absence of pulmonary valve. *Circulation*, 1957, *15*, 397–404.
6. CHEVERS, N. Recherches sur les maladies de l'artere pulmonaire. *Arch. Gen. Med.*, 1847, *15*, 488–508.
7. CHILDERS, R. W., and MCCREA, P. C. Absence of pulmonary valve: case occurring in Marfan syndrome. *Circulation*, 1964, *29*, 598–603.
8. CONTRO, S., MILLER, R. A., WHITE, H., and POTTS, W. J. Bronchial obstruction due to pulmonary artery anomalies. II. Pulmonary artery aneurysm. *Circulation*, 1968, *17*, 424–427.
9. D'CRUZ, I. A., LENDRUM, B. L., and NOVAK, G. Congenital absence of pulmonary valve. *Am. Heart J.*, 1964, *68*, 728–740.
10. FISCHER, H. W., LUCIDO, J. L., and LYNXWILER, C. P. Lobar emphysema. *J.A.M.A.*, 1958, *166*, 340–345.
11. FOWLER, N. O., MANNIX, E. P., and NOBLE, W. Some effects of partial pulmonary valvectomy. *Circulation Res.*, 1956, *4*, 8–11.
12. FRANKEN, E. A., JR., and BUEHL, I. Infantile lobar emphysema: report of two cases with unusual roentgenographic manifestation. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *98*, 354–357.
13. HANNA, E. A., VATTANAPAT, S., and DERRICK, J. R. Congenital lobar emphysema with bronchial atresia. *Ann. Thoracic Surg.*, 1969, *7*, 357–361.
14. HENDREN, W. H., and MCKEE, D. M. Lobar emphysema of infancy. *J. Pediat. Surg.*, 1966, *1*, 24–39.
15. JONES, J. C., ALMOND, C. H., SNYDER, H. M., MEYER, B. W., and PATRICK, J. R. Lobar emphysema and congenital heart disease of infancy. *J. Thoracic & Cardiovasc. Surg.*, 1965, *49*, 1–10.
16. KOONTZ, A. R. Congenital cysts of lung. *Bull. Johns Hopkins Hosp.*, 1925, *37*, 340–361.
17. KURTZ, C. M., SPRAGUE, H. B., and WHITE, P. D. Congenital heart disease: interventricular septal defects with associated anomalies in series of three cases examined post mortem, and living patient fifty-eight years old with cyanosis and clubbing of fingers. *Am. Heart J.*, 1927, *3*, 77–90.
18. LINCOLN, J. C. R., STARK, J., SUBRAMANIAN, S., ABERDEEN, E., BONHAM-CARTER, R. E., BERRY, C. L., and WATERSTON, D. J. Congeni-

tal lobar emphysema. *Ann. Surg.*, 1971, *173*, 55–62.

19. Miller, R. A., Lev, M., and Paul, M. H. Congenital absence of pulmonary valve: clinical syndrome of tetralogy of Fallot with pulmonary regurgitation. *Circulation*, 1962, *26*, 266–278.
20. Murray, G. F. Congenital lobar emphysema. *Surg., Gynec. & Obst.*, 1967, *124*, 611–625.
21. Osman, M. Z., Meng, C. C. L., and Girdany, B. R. Congenital absence of pulmonary valve: report of eight cases with review of literature. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *106*, 58–69.
22. Park, C. D., Waldhausen, J. A., Friedman, S., Aberdeen, E., and Johnson, J. Tracheal compression by great arteries in mediastinum. *A.M.A. Arch. Surg.*, 1971, *103*, 626–632.
23. Pernot, C., Hoeffel, J. C., Henry, M., Worms, A. M., Stehlin, H., and Louis, J. P. Radiological patterns of congenital absence of pulmonary valve in infants. *Radiology*, 1972, *102*, 619–622.
24. Potts, W. J., Holinger, P. H., and Rosenblum, A. H. Anomalous left pulmonary artery causing obstruction to right main bronchus: report of case. *J.A.M.A.*, 1954, *155*, 1409–1411.
25. Raynor, A. C., Capp, M. P., and Sealy, W. C. Lobar emphysema of infancy. *Ann. Thoracic Surg.*, 1967, *4*, 374–385.
26. Robertson, R., and James, E. S. Congenital lobar emphysema. *Pediatrics*, 1951, *8*, 795–804.
27. Ruttenberg, H. D., Cary, L. S., Adams, P., Jr., and Edwards, J. E. Absence of pulmonary valve in tetralogy of Fallot. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *91*, 500–510.
28. Simonds, J. P. Congenital malformation of aortic and pulmonary valves. *Am. J. M. Sc.*, 1923, *166*, 584–595.
29. Stanger, P., Lucas, R. V., Jr., and Edwards, J. E. Anatomic factors causing respiratory distress in acyanotic congenital cardiac disease: special reference to bronchial obstruction. *Pediatrics*, 1959, *43*, 760–769.
30. Stovin, P. G. I. Congenital lobar emphysema. *Thorax*, 1959, *14*, 254–262.
31. Strunge, P. Infantile lobar emphysema with lobar agenesis and congenital heart disease. *Acta pediat. scandinav.*, 1972, *61*, 209–212.
32. Vanpeperstraete, F. Cartilaginous skeleton of bronchial tree. Chapter X. Foetal studies. *Adv. Anat., Embryol., & Cell Biol.*, 1973, *48*, 55–62.
33. Waldhausen, J. A., Friedman, S., Nicodemus, H., Miller, W. W., Rashkind, W., and Johnson, J. Absence of pulmonary valve in patients with tetralogy of Fallot. *J. Thoracic & Cardiovasc. Surg.*, 1969, *57*, 669–674.
34. Wyler, F., Rutishauser, M., Olafsson, A., and Kaufmann, H. J. Congenital absence of pulmonary valve in tetralogy of Fallot and origin of left pulmonary artery from aortic arch. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 505–508.

TRANSARTERIAL ELECTROCOAGULATION THERAPY OF A PSEUDOANEURYSM IN THE HEAD OF THE PANCREAS*

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ABSTRACT:

Because chemical and transcatheter embolizations for control of bleeding were inappropriate, electrocoagulation through a guidewire was used for arterial occlusion. The power was alternating current. The effectiveness of the procedure was confirmed by arteriograms done immediately and 5 months later.

NONOPERATIVE treatment of gastrointestinal bleeding utilizing angiographic techniques has become a frequent roentgenologic procedure. Vasopressin infusion and embolic occlusion of vessels are techniques which can effectively control hemorrhage in a wide variety of circumstances, but both techniques generally have an effect on vessels adjacent to the artery responsible for the hemorrhage. Recently we encountered a clinical situation in which neither vasopressin infusion nor transcatheter embolization appeared to be the appropriate method of treatment. As an alternative, a guidewire was advanced into the artery responsible for the hemorrhage and a clinical alternating current electrocoagulation unit was employed to superselectively occlude that artery.

REPORT OF A CASE

A 58 year old man with a 12 year history of pancreatitis was admitted to the surgical service of Westminister Hospital on July 31 with complaints of weakness, fatigue, and abdominal pain of 2 years' duration, accentuated in the past 4 months and much worse in the last 2 weeks. Black, tarry stools had been noted for 2 months. On admission, hemoglobin was 3 grams per cent with a hematocrit of 11, and stool samples were positive for occult blood. Endoscopic examinations and barium studies of the upper and lower gastrointestinal tract were negative on 2 occasions. Symptomatic therapy with packed red blood cells and whole blood transfusions was initiated.

On September 17, a diagnostic laparotomy did not uncover the source of bleeding but a hard mass in the head of the pancreas was noted. Occult bleeding had stopped temporarily but recurred by October 7. Further endoscopic and barium studies were again unrewarding. On October 18, an arteriogram identified a pseudoaneurysm (17 mm. in diameter) in the head of the pancreas as the probable source of the chronic recurrent hemorrhage. On October 27, a pancreaticoduodenectomy was undertaken, but extensive fibrosis prevented removal of the pancreatic head. Active hemorrhage through the ampulla of Vater was observed. Postoperatively, bile peritonitis due to duodenal perforation necessitated another surgical procedure and 2 duodenal drainage tubes were brought out through the anterior abdominal wall. The proximal loop of jejunum was necrotic and required resection. Fresh blood was returned from the duodenal tube daily indicating continuing hemorrhage from the pseudoaneurysm. By November 21, the patient had received a total of 64 units of blood as transfusions. Arteriography was repeated with the intention of occluding the pseudoaneurysm with Gelfoam emboli. The pseudoaneurysm had doubled in diameter since the earlier arteriogram and now measured 35 mm. in diameter (Fig. 1*B*). The artery leading to the pseudoaneurysm was clearly identified (Fig. 1*A*), but could not be entered superselectively. Because of the acute angle which the artery to the pseudoaneurysm made with the gastroduodenal ar-

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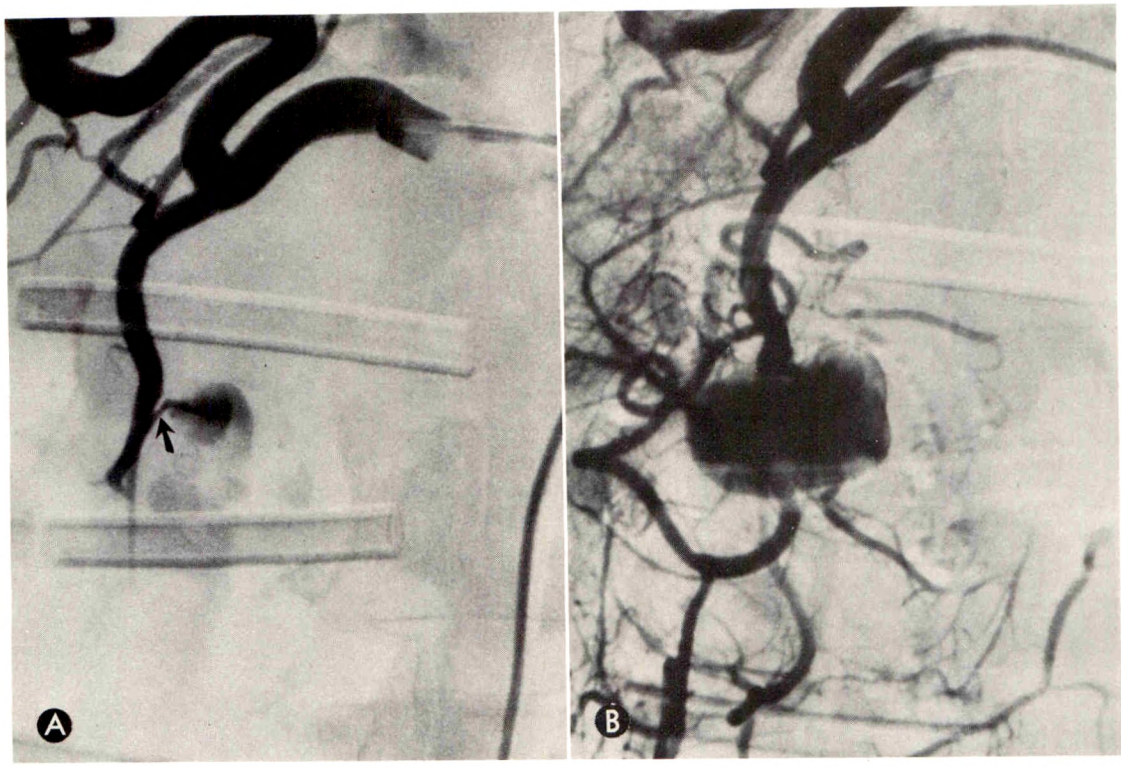

Fig. 1. (*A*) Common hepatic artery injection, early arterial phase. The gastroduodenal artery is filled with contrast material and there is clear delineation of the artery leading to the pseudoaneurysm in the head of the pancreas (arrow). (*B*) Gastroduodenal artery injection, mid-arterial phase. Dense opacification of the pseudoaneurysm in the head of the pancreas. The pseudoaneurysm had doubled in diameter to 35 mm. since the arteriogram of 34 days earlier (not shown).

tery, and because of the recent postoperative perforation of the duodenum and necrosis of the jejunum, it was feared that embolization of the gastroduodenal artery could result in significant duodenal ischemia due to distal branch occlusions. A guidewire, 0.038 inches in diameter with a "J" shaped tip (3 mm. radius), was advanced through the catheter in the gastroduodenal artery and the tip of the guidewire was positioned in the artery leading to the pseudoaneurysm. A reference plate was attached to the patient's back (Fig. 2). A Valley-Lab clinical electrocoagulation unit (model SSE-2J) on a coagulation setting of 2.5 was touched to the guidewire protruding from the femoral artery for 3 seconds. The guidewire was then removed and a contrast medium injection through the catheter demonstrated persistent patency of the pseudoaneurysm. This procedure was repeated at a setting of 4 and was again unsuccessful. A setting of 7 was then used for $2\frac{1}{2}$ seconds and resulted in a stabbing pain of mild to

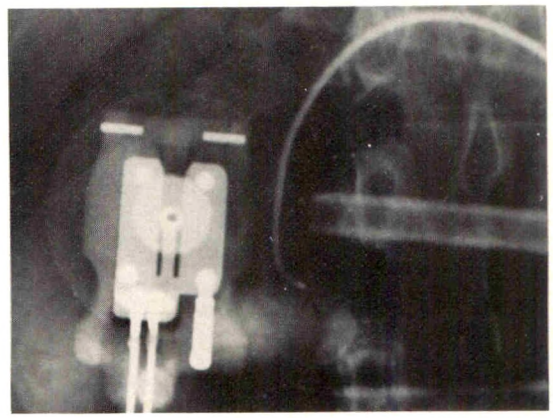

Fig. 2. The catheter used for arteriography remains in the gastroduodenal artery. A "J" shaped guidewire, 0.038 inches in diameter, has been passed through the catheter, and the tip of the guidewire is positioned in the artery leading to the pseudoaneurysm. Five mm. of the guidewire protrude from the tip of the catheter. A reference plate has been attached to the right side of the patient's back.

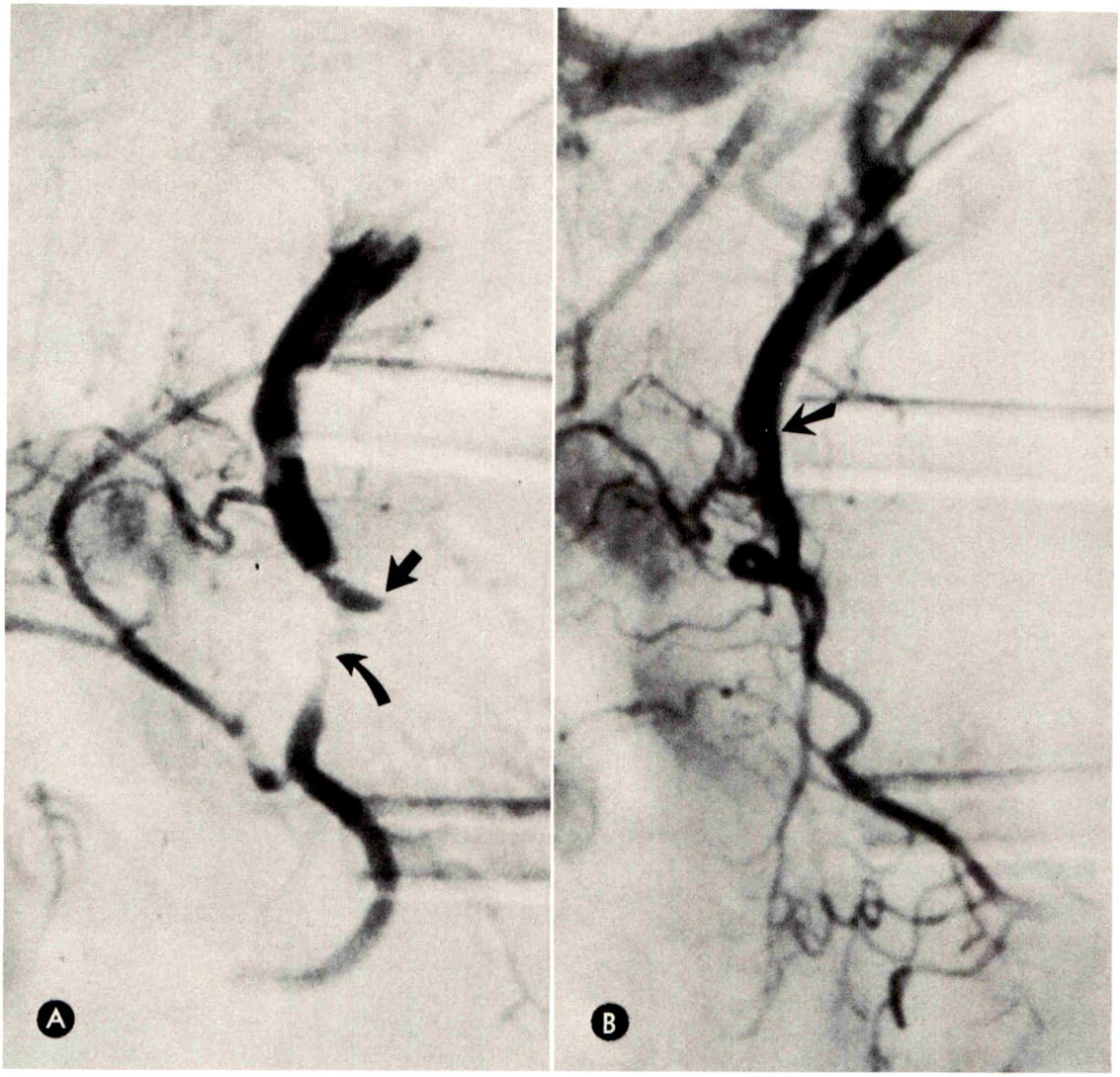

FIG. 3. (*A*) Gastroduodenal artery injection, mid-arterial phase following electrocoagulation. The artery leading to the pseudoaneurysm is occluded (straight arrow). The gastroduodenal artery distal to the site of electrocoagulation is markedly narrow and irregular (curved arrow). No opacification of the pseudoaneurysm occurred. (*B*) Gastroduodenal artery injection, mid-arterial phase, 30 minutes after the previous study shown in (*A*). The gastroduodenal artery has been completely occluded at the site shown earlier to be narrowed. Retrograde thrombosis has occurred to the level of origin of the posterior superior pancreaticoduodenal artery (arrow).

moderate severity in the middle of the patient's back. The catheter and guidewire were left in place for 20 minutes. The guidewire was then easily withdrawn and a contrast medium injection demonstrated complete occlusion of the artery leading to the pseudoaneurysm and marked irregularity and compromise of the gastroduodenal artery opposite the origin of the occluded vessel (Fig. 3*A*). A repeat gastroduodenal artery study ½ hour later revealed complete occlusion of the gastroduodenal artery with retrograde thrombosis to the origin of the posterior superior pancreaticoduodenal branch (Fig. 3*B*). A superior mesenteric arteriogram was then performed demonstrating excellent fill of both the proximal gastroduodenal artery and the portion of that vessel distal to the occlusion through the pancreaticoduodenal branches. Excellent fill of duodenal branches was apparent on the superior mesenteric arteri-

ogram (Fig. 4). Since this procedure, no blood loss into the gastrointestinal tract has occurred. The length of follow-up is presently 9 months. The patient was discharged from the hospital 12 weeks after the procedure. A follow-up arteriogram 5 months after the procedure demonstrated continued occlusion of the gastroduodenal artery. An aneurysm of the proximal portion of the gastroduodenal artery has developed (Fig. 5). Superior mesenteric branches continue to provide good blood flow to the duodenum (not shown).

DISCUSSION

This patient presented with the unusual problem of chronic recurrent hemorrhage from a pseudoaneurysm in the head of the pancreas. The pseudoaneurysm presumably had eroded into the pancreatic ductal system, as bleeding into the gastrointestinal tract was occurring through the ampulla of Vater. A number of similar cases has been reported,[2] but to our knowledge no previous report of non-surgical management of this type of lesion has appeared.

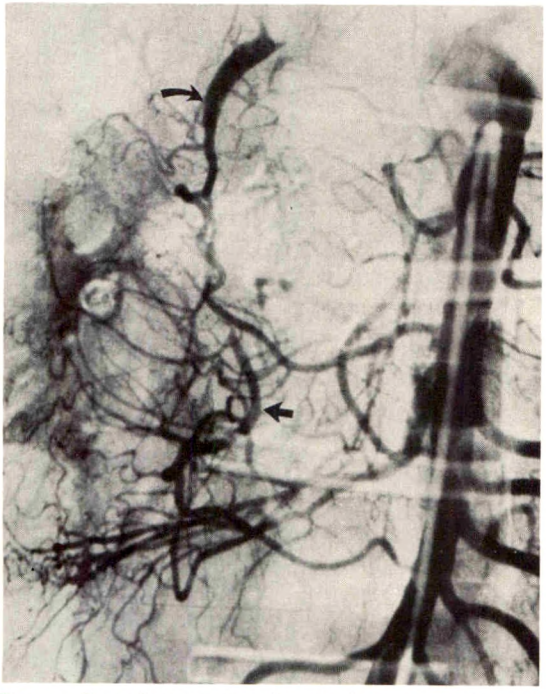

FIG. 4. Superior mesenteric arteriogram performed after the studies shown in Figure 3, mid-arterial phase. Extensive collateral flow to both the proximal (curved arrow) and distal (straight arrow) portions of the gastroduodenal artery. The length of gastroduodenal artery that had been occluded measured 3 cm. Duodenal blood supply is completely intact. No opacification of the pseudoaneurysm occurred.

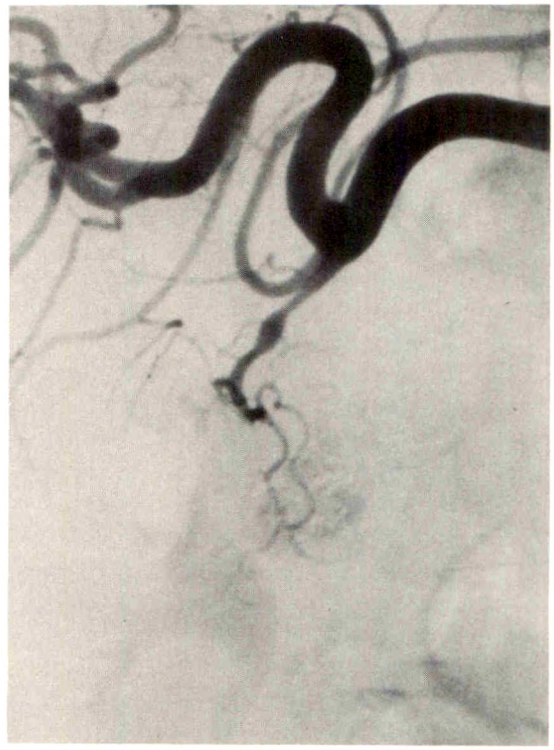

FIG. 5. Hepatic artery injection 20 weeks after the occlusion of the artery to the pseudoaneurysm. The gastroduodenal artery has remained occluded in its mid-portion. An aneurysm of the proximal gastroduodenal artery has developed.

Embolization of the gastroduodenal artery has been shown to produce ischemic changes in the mucosa[1] and because of the recent mobilization of the duodenum in our patient, followed by a postoperative perforation of the duodenum and necrosis of jejunum, we had reason to suspect preexisting compromise of the duodenal vascular supply. The acute angle that the artery to the pseudoaneurysm made with the gastroduodenal artery (Fig. 1*A*) made it likely that a large volume of emboli would be required to occlude the vessel. For these reasons, transcatheter embolic occlusion of the pseudoaneurysm was considered unsafe. Transarterial electrocoagulation of the artery leading to the pseudoaneurysm provided the selectivity of vascular occlu-

sion required in this case. A clinical alternating current electrocoagulation unit was immediately obtainable. Direct current electrothrombosis is at present a technique that has been more extensively investigated[3] but a satisfactory power source was not readily available. Preliminary experimental studies of our own using alternating current coagulators have pointed out several factors which contributed to the success of the procedure and several dangers inherent in the method, some of which have been previously reported.[3]

Early experiments on dogs have prompted us to change our technique of electrocoagulation. An ammeter has been connected in series with the clinical electrocoagulation unit. Coagulation is initiated on the lowest setting on the unit for 5 seconds. If the milliamperage (mA) reaches a peak value and then decreases within those 5 seconds, the coagulation is repeated twice more at that setting for 5 seconds each with a 15 second interval between each coagulation. The guidewire is then withdrawn and an arteriogram is performed. If the mA does not decrease from its peak in the first 5 seconds, the setting is increased in steps until coagulation in the first 5 seconds results in mA reaching a peak and then falling. At that setting, the coagulation is performed a total of 3 times, as described, before performing an arteriogram to check on the result. Effective mA has been in the 250 to 500 mA range. Further increases in either mA or time can result in perforation of the vessel. It has also become apparent that using our present catheters (external diameter of 1.6 to 2.2 mm.) and guidewires (usually 1.0 mm. in diameter) there is a severe limitation in the size of the vessels that can be safely and reproduceably occluded. Vessels the same size or smaller than the catheter can be readily and safely occluded, but in these vessels the catheter or guidewire is virtually in a wedged position. The results in vessels larger than the catheter are variable, but more often than not, attempts at occluding them are unsuccessful.

An important element in the safety of the procedure may be the internal regulatory circuitry in the coagulator units. A voltage limiting circuit, when present, can prevent a significant rise in voltage with increases in the resistance through which the current is passing. If peak milliamperage is restricted to 500 milliamperes and coagulation is performed at the lowest setting that results in mA reaching a peak and then decreasing, early studies suggest that the technique is effective and safe in small arteries. Engineering appraisals, *in vivo* animal studies and *in vitro* analyses, are presently underway to define thoroughly the factors that would establish this technique as a safe and effective clinical method of effecting transcatheter occlusions of vessels.

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We would like to express our sincere appreciation for the help afforded by Mr. Ken Cheung.

REFERENCES

1. Bookstein, J. J., Chlosta, E. M., Foley, D., and Walter, J. F. Transcatheter hemostasis of gastrointestinal bleeding using modified autogenous clot. *Radiology*, 1974, *113*, 277–285.
2. Harris, R. D., Anderson, J. E., and Coel, M. N. Aneurysms of small pancreatic arteries: cause of upper abdominal pain and intestinal bleeding. *Radiology*, 1975, *115*, 17–20.
3. Phillips, J. F. Transcatheter electrocoagulation of blood vessels. *Invest. Radiol.*, 1973, *8*, 295–304.

VOL. 125, NO. 2

ARTERIOGRAPHIC MANIFESTATIONS OF ABDOMINAL SITUS INVERSUS*

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ABSTRACT:

The arteriographic findings are described and the embryology is discussed in a rare case of situs inversus of the abdominal viscera combined with reversed rotation of the bowel. The arteriogram correctly localized the precise site of hemorrhage in this perplexing case, and a complex surgical problem was simplified.

LOCALIZATION of the source of uncomplicated acute lower gastrointestinal hemorrhage remains a difficult and frequently frustrating diagnostic dilemma. When the case is complicated by abnormal intestinal rotation, the situation may be baffling at laparotomy.

The case described below combines these 2 diagnostic problems. A woman with situs inversus of the abdominal viscera combined with reversed rotation of the bowel presented with acute lower intestinal bleeding. Emergency preoperative angiography accurately localized the site of hemorrhage and the abnormal location of the cecum in this rare case.

REPORT OF A CASE

A.C., a 73 year old female, was admitted to the University of Virginia Hospital on May 30, 1974 with a 1 day history of acute rectal bleeding. She denied abdominal pain or emesis.

The patient's previous medical history was significant. She had experienced several episodes of minor lower gastrointestinal bleeding in the past. Roentgenograms of the gastrointestinal tract performed in 1971 demonstrated multiple diverticula of the sigmoid colon. Situs inversus of the abdominal viscera was also shown. The stomach was on the right and the entire small bowel was located in the right side of the abdomen (Fig. 1). The colon occupied a position in the left side of the abdomen (Fig. 2). The heart was located in its normal position on the left. The bleeding subsided spontaneously after each episode and she did well until the afternoon of admission.

Physical examination on admission revealed an elderly, pale, restless woman with a heart rate of 96 and a supine blood pressure of 130/60 mm. Hg. Hyperactive bowel sounds were present. Shortly after admission, however, she became markedly hypotensive with a blood pressure of 90/0 mm. Hg.

A gastric aspirate contained no blood. Proc-

FIG. 1. Upper gastrointestinal series shows the stomach and the small bowel on the right.

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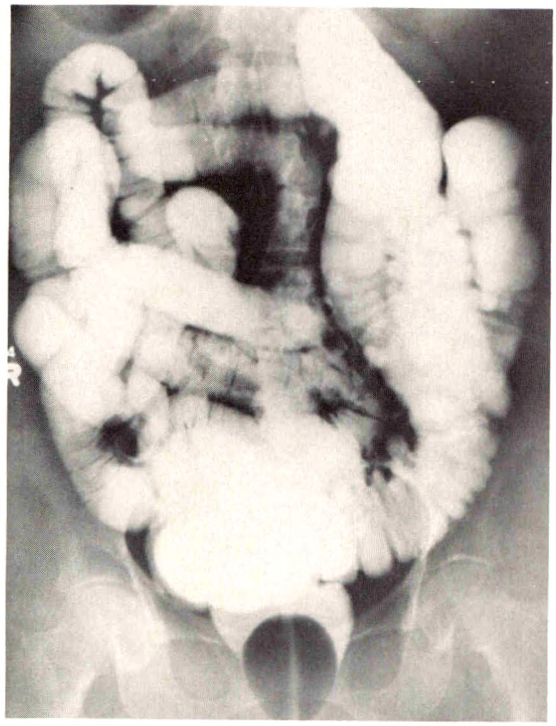

FIG. 2. Barium enema examination demonstrates the entire colon located in the left side of the abdomen.

toscopy demonstrated dark blood in the rectum but failed to reveal the source of hemorrhage. The hematocrit was 34 per cent on admission. Despite the rapid transfusion of 3,000 ml. of blood, the hematocrit dropped to 30 per cent.

An emergency percutaneous angiogram was performed. Selective injection of the superior mesenteric artery (Fig. 3) demonstrated that the jejunal and ileal branches originated from the right side of the artery. The right colic and ileocolic vessels branched from the left side of the superior mesenteric artery and supplied the cecum and ascending colon in the left lower quadrant. Massive extravasation of contrast material was seen at the junction of the cecum and ascending colon. The venous phase showed the liver in the left upper quadrant (Fig. 4).

At celiotomy a blood-filled colon was found but no active bleeding site was identified. Exploration of the abdomen revealed that the liver and gallbladder were located in the left upper quadrant and abdominal situs inversus was present. The entire colon was on a free mesentery. Marked diverticulosis was noted in the sigmoid colon. A few scattered diverticulae were noted in the ascending colon. A hemicolectomy was performed.

Gross and histologic study of the colon revealed diverticulosis of the terminal ileum and ascending colon.

DISCUSSION

Situs inversus has intrigued physicians and confused the unwary since this rare condition was first described by Aristotle.[1] Seventy-six of the 1,551,047 patients who registered at the Mayo Clinic from 1910 through 1947 were found to have complete situs inversus.[3] Isolated abdominal situs inversus is an even rarer condition.

The colon in our patient is on the left side and is associated with situs inversus of the abdominal viscera. The liver and entire colon are on the left and the stomach is on the right. A review of the literature has revealed only 2 other cases. Van Meurs[5] reported a similar case and described it as an

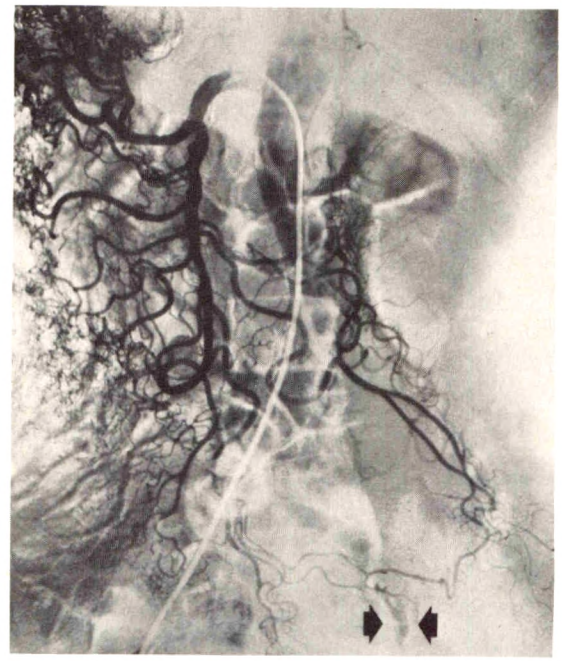

FIG. 3. Subtraction film of the selective superior mesenteric arteriogram shows the branches supplying the small bowel originating from the right side of the artery. The right colic and ileocolic vessels branch from the left side of the artery. Extravasation of contrast material is seen at the junction of the cecum and ascending colon (arrows).

example of nonrotation. McIntosh and Donovan[4] also described a similar case and called the anomaly malrotation with situs abdominalis partialis.

The problem of anomalies of the intestinal tract is a complex and intriguing subject. A knowledge of the embryology is necessary to understand the vagaries of intestinal rotation. There are conflicting reports as to what actually occurs during the first and second stages of intestinal rotation because direct proof is as yet almost impossible to obtain. These conflicting opinions account for the varied terminology used in describing these cases.

The fetal alimentary tract is a straight tube suspended by a dorsal mesentery at about the fourth week of life. By the fifth week it can be divided into the foregut, midgut, and hindgut, according to the blood supply it receives. These areas are supplied by celiac axis, the superior mesenteric, and inferior mesenteric arteries, respectively. The midgut grows rapidly, forming a loop which extends into the umbilical sac. The midgut is divided into prearterial and postarterial segments according to its relationship to what is to become the superior mesenteric artery.

The first stage of rotation is generally accepted as the period of umbilical cord herniation. Authorities differ on the sequence of rotation. However, all authorities agree that the total rotation in the first 2 stages is 270° counterclockwise.

The first stage of rotation occurs between the fifth and tenth weeks of embryonic life. Estrada[2] subdivides this stage into the substage of deviation and the substage of realignment. During each substage the midgut, which is supplied by the superior mesenteric artery, rotates 90° counterclockwise, making a total rotation of 180° in the first stage.

The second stage occurs during the tenth week and lasts only a short period of time. The prearterial segment leaves the umbilical sac first and enters to the right of the artery. The hindgut is pushed by the small bowel to the left, forming the descending colon. When the cecum and terminal ileum return to the abdominal cavity, they lie anterior to the artery. There is an additional 90° of counterclockwise rotation during this stage. The superior mesenteric artery thus comes to lie anterior to the third portion of the duodenum and posterior to the transverse colon completing the 270° of counterclockwise rotation. The third stage is the stage of descent and fixation of the colon.

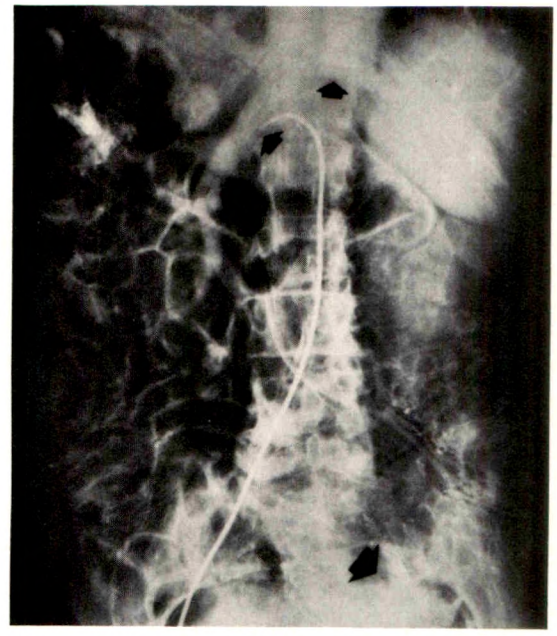

Fig. 4. Subtraction technique film of venous phase of superior mesenteric arteriogram shows the mesenteric vein entering the portal vein (small arrows). The liver is in the left upper quadrant. The large arrow shows extravasated contrast medium in the proximal ascending colon.

In understanding our complex case, according to Estrada,[2] "the important thing is not which side of the abdominal cavity the colon is on, but the relationship of the colon to the stomach and liver."

The colon is on the left side and associated with situs inversus of the abdominal viscera. The liver and entire colon are on the left and the stomach is on the right. Estrada[2] feels this rare anomaly is best termed situs inversus with reversed rota-

tion. This occurs during the second stage. The substage of deviation occurs normally with a 90° counterclockwise rotation. However, during the substage of realignment a 180° clockwise rotation of the midgut occurs. The net rotation is 90° clockwise. In the second stage the prearterial segment is reduced first from the umbilical sac. The small intestine is forced to the same side as the stomach. The cecum and ascending colon enter the abdomen and lie on the same side as the liver, which is on the left in abdominal situs inversus, and the terminal ileum enters the cecum from the left side.

The mesenteric vessels form the axis of intestinal rotation in the embryo. Thus, the vascular architecture reflects the position of the abdominal viscera. By examining the position and orientation of the mesenteric vessels, the localization of the organs can be correctly identified and the type of intestinal rotation can be predicted. The arteriogram correctly localized the abdominal viscera in our case. The branches of the superior mesenteric artery were reversed. The vessels supplying the small bowel branched from the right side of the superior mesenteric artery and supplied the small bowel on the right side of the abdomen. The vessels supplying the colon originated from the left side of the artery. The venous phase is important because it correctly localized the position of the liver in the left upper quadrant of the abdomen. By tracing the course of the superior mesenteric artery, the site of hemorrhage was easily localized in the ascending colon.

The arteriographic studies confirmed the findings demonstrated on the previous barium studies and localized the site of hemorrhage. The angiogram was then utilized as a road map at laparotomy to direct the surgeon to the precise site of hemorrhage. A complex and potentially dangerous surgical problem was simplified by arteriography.

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REFERENCES

1. Aristotle: De generatione animalium, quoted by Schmutzer, K. J., and Linde, L. M. Situs inversus totalis associated with complex cardiovascular anomalies. *Am. Heart J.*, 1958, *56*, 761.
2. Estrada, R. L. Anomalies of Intestinal Rotation and Fixation. Charles C Thomas, Publisher, Springfield, Ill., 1958.
3. Mayo, C. W., and Rice, R. G. Situs inversus totalis. *Arch. Surg.*, 1949, *58*, 724–730.
4. McIntosh, R., and Donovan, E. J. Disturbances of rotation of intestinal tract. *Am. J. Dis. Child.*, 1939, *57*, 116–166.
5. Van Meurs, D. P. Ileocolic intussusception associated with nonrotation of midgut, congenital heart disease, congenital hydrocele. *Brit. J. Surg.*, 1946, *34*, 91–93.

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TECHNIQUE AND COMPLICATIONS OF TRANSUMBILICAL CATHETERIZATION OF THE PORTAL VEIN AND ITS TRIBUTARIES*

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ABSTRACT:

In 120 patients who had 127 transumbilical catheterizations of the portal vein, 33 of pancreatic veins, and 15 for other portal tributaries, the surgical success rate was 91 per cent over-all, increasing to 95 per cent during the last year. Catheterization was made with the same technique as is used for arteriography. The most important complication was thrombosis of the portal vein, giving slight transient symptoms in 2 patients. Other complications were wound infection, suture granuloma, herniation in the linea alba, bleeding from the operative wound, and subintimal injections. As the technique may yield good results in studies of drug resorption, flow studies, detection of metastases, and investigation of pancreatic veins, it seems justified in suitable patients despite the complication rate. None of the complications produced lasting disability.

THE transumbilical approach to the portal vein system has been used for: portography of cysts, abscesses, and metastases; flow measurements in the splanchnic area; decompression in portal hypertension; infusion of cytostatics and antibiotics; resorption studies; sampling of metabolites and hormones; and pancreatic phlebography. As we have routinely investigated the portal vein and especially its tributaries for 3 years, our technique and the complications will be presented and analyzed.

MATERIAL

Seventy-seven males and 43 females, of an age range between 16–86 (mean 58) years, were examined with portography 127 times. In 48 cases tributaries of the portal vein were also catheterized. All but 6 patients had been or were to be treated for malignant disease, mostly carcinoma of the rectum or sigmoid, and the usual question was whether or not the liver contained metastases.

METHOD

Operative technique. The umbilical cord in 106 of the 120 cases was exposed extraperitoneally by a supra-umbilical midline incision under local or general anesthesia and reopened with a probe. In 14 patients the peritoneal cavity was opened in order to perform the umbilical vein cannulation. A soft PVC catheter (infant-feeding tube, 8 F, Argyle, England) with end-hole was introduced. The umbilical vein was then sutured to the linea alba in order to avoid dislodgement of the cord into the abdomen.

Roentgen technique. The interval between the operation and the roentgenological examination was 0–3 days (mean 2 days). Recannulation was made after 1 and 2 years, respectively, in 2 patients. Under fluoroscopic control a 160 guidewire with a flexible and precurved tip was introduced into the portal system *via* the infant-feeding tube. The tube was withdrawn and replaced by a thin-walled Ödman-Ledin radiopaque catheter (ID/OD 1.4/2.2) with the tip curved about 80 degrees 3–4 cm. from the tip. Generally the catheter had 2 side-holes close to the tip, and in several cases there was also a side-hole about 6 cm. from the tip of the convex surface of the catheter to allow easier flow of contrast medium into the left branch of the portal

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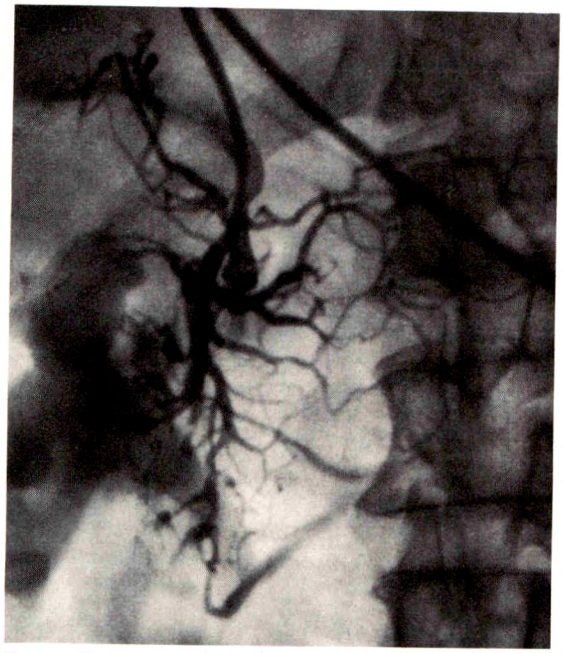

FIG. 1. Demonstration of the veins of the head of the pancreas and some duodenal veins. Note the varying caliber of the duodenal veins: these are normal.

vein. After introduction it was easy in most cases to rotate the curved guidewire or the tip of the catheter into the main portal vein and then to advance it to a suitable site. In supine position, portograms were obtained regularly in anteroposterior projection; often roentgenograms in prone position in anteroposterior and lateral projections were added. Elema-Schönander cut film changers were used and the series was 5/5, 8/4, and 3/6. Forty ml. of contrast medium (Isopaque Cerebral, Nyco, Norway) were injected at a rate of about 10 ml./sec. using a high pressure syringe.

In most patients the catheter was then exchanged for one without side-holes but with a curved tip for catheterization of pancreatic (Fig. 1), gastroduodenal (Fig. 2), jejunal, ileal and colon veins (Fig. 3). For further details regarding this technique see Göthlin *et al.*, 1974.[2] In these patients 10 ml. of contrast medium were injected by hand. Finally, a new catheter was introduced and left with its tip in the main portal vein for metabolic studies.

RESULTS

In 134 attempts to cannulate the umbilical vein, 122 succeeded (91 per cent). During the last year the failure rate was 5 per cent. In 5 patients it was impossible to identify the original lumen of the umbilical vein, and in 4 patients the probe perforated the vein wall without causing complication. In 2 patients the infant-feeding tube slipped out of the umbilical vein and could not be replaced, and in 1 case the infant-feeding tube had no end-hole and thus could not be exchanged to permit other studies. In several cases the portograms in the anteroposterior projection did not demonstrate the veins of the left lobe sufficiently and the patient was then changed to a prone position and the examination repeated. Sometimes selective catheterization of the left branch was necessary. In most cases, when pancreatic veins were to be catheterized, the study was successful (Fig. 1).

COMPLICATIONS (Table 1)

Wound infection was encountered in 7 patients. All healed within 10 days. There were no signs of systemic infection in any patient.

Suture granuloma was encountered in 7 patients.

FIG. 2. Normal gastroduodenal veins.

Herniation in the linea alba occurred in 6 patients, of whom 2 had symptoms requiring repair of the hernia.

Bleeding from the operative wound occurred in 4 patients. In 2 of them tamponade stopped the bleeding while in the other 2 subcutaneous ligation of bleeding vessels was carried out. One patient had pronounced melena of unknown cause for 1 day after the portography. Since then the patient has been well. There was no significant bleeding after catheter withdrawal, which was made stepwise during half an hour.

Thrombosis occurred in the main portal vein in 1 patient and in another patient in the right branch of the portal vein:

Case 1. Female, age 60, with carcinoma of the large bowel. At the first portography (Fig. 4*A*), there is extensive thrombosis of the left portal branch. Heparin therapy was carried out for 6 days. For 3 days the patient experienced slight upper abdominal pain, and an elevation of the body temperature to a maximum 38.1° C was registered. For the same period there was a slight elevation of alkaline phosphatase and transaminase activity. Bilirubin was normal. After 3 days the patient felt well and portography (Fig. 4*B*) revealed improved blood flow to the left lobe of the liver. Follow-up revealed entirely normal liver function.

Case 11. Male, age 64, with large bowel carcinoma. At portography a small thrombus in the main right portal branch (Fig. 5) was revealed. The patient experienced no pain but had a fever to 38.0° C. On 1 occasion there was a slight elevation of alkaline phosphatase while other liver function tests were normal. Treatment with heparin was given for 1 week. At follow-up the liver function was normal.

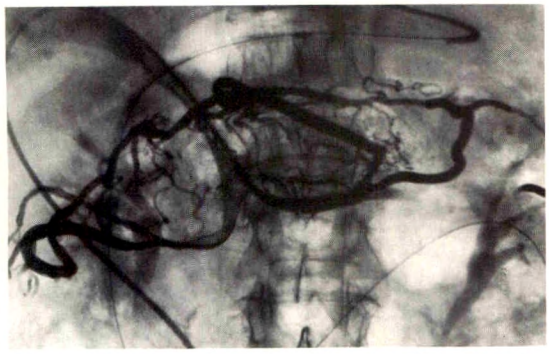

Fig. 3. Normal veins of the transverse colon. Besides the transumbilical portal catheter, a gastric tube has been inserted.

Fig. 4. (*A*) Thrombosis in the stem and the branches of the left portal vein. (*B*) Two days later there is resolution of the thrombus although remnants are left.

In the patients where thrombosis within the left branch was suspected or evident, the patients felt well and there were no abnormalities in the liver function tests (bilirubin, alkaline phosphatase, lactic dehydrogenase and serum glutamic oxalacetic transaminase).

Lack of filling with contrast medium in parts of the left hepatic lobe (Fig. 6; and 7) occurred in 8 patients, and most of these were considered to have thrombosis. Subintimal injection and extravasation were seen in 12 cases. In 8 patients this occurred in the main portal vein or its left branch. The most extensive subintimal injection is demonstrated in Figure 8).

Discussion

Successful cannulation of the umbilical

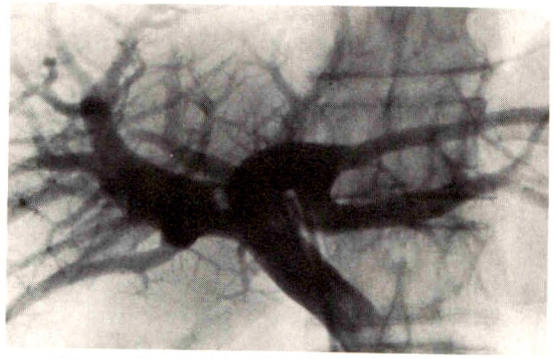

Fig. 5. Small thrombus in the stem of the right portal branch.

cord was made in 91 per cent of the cases in our series. Several authors do not state their rate of success. However, Lavoie *et al.*[5] in 1966, Braastad *et al.*[1] in 1970, and Kessler *et al.*[3] in 1973 had about the same rate as we have had. White *et al.*[10] in 1966 and Watts and Douglas[9] in 1971 reported a lower rate due evidently to the composition of their patient sample. Several of the authors state that the failure rate decreases with increasing experience, and we can confirm this opinion. In the latter half of our series, failure was unusual. The time factors must also be borne in mind: if a surgeon tries long enough the likelihood of success will increase, but such factors as the duration of the anesthesia and the danger of perforating the umbilical cord must be considered.

No serious complication in connection with the cannulation occurred. One reason may be that our patients were comparatively healthy. Perforation of the umbilical vein wall without sequelae has been reported by most authors and in our material we have the same experience. To perforate the portal vein, on the other hand, may be fatal, as reported by Watts and Douglas[9] in 1971 and Kessler *et al.*[3] in 1973. Wound infections, bleeding at the cannulation site, and hernias in the linea alba are other less serious complications reported, although repair of the hernias has been necessary as reported by Piccone *et al.*[6] in 1968 and Kessler *et al.*[3] In our series also, 2 patients out of 6 required hernia repair.

Table I

Complications	Number of Patients	
Wound infection	7	
Suture granuloma	7	
Herniation in the linea alba	6	
Bleeding from the operation wound	4	
Melena	1	
Thrombosis	10	
Main portal vein		1
Right branch		1
Left branch		8
Subintimal injection or extravasation	12	
Main portal vein		6
Right branch		0
Left branch		2
Umbilical vein		2
Pancreatic veins		2

The principal hazard of transumbilical catheterization is thrombosis, especially if the catheter is allowed to stay in the portal vein for a considerable time, despite the fact that Storer and Akin[8] in 1966, Piccone *et al.*[7] in 1967, and Kiessling *et al.*[4] in 1972 encountered no problems using heparin treatment. Occurrence of thrombosis has been reported by Kessler *et al.*[3] and Wiechel and associates[11] in 1974, and complete recovery followed. In our cases it is worth noting that the thrombosis in Case 11 (Fig. 5) was

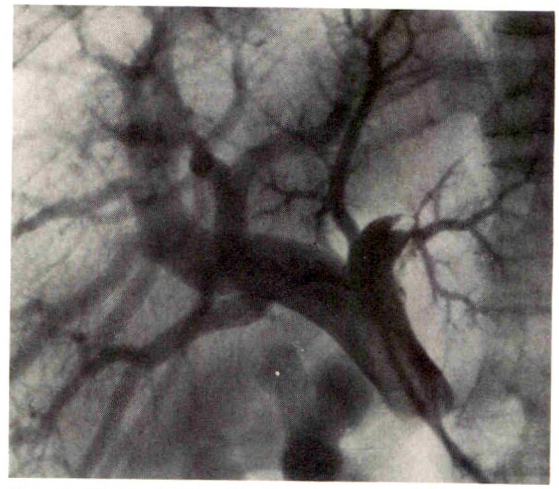

Fig. 6. Thrombosis of 2 branches to the left lobe of the liver with complete obstruction of 1 of them.

very small and that the fever and transient increase of alkaline phosphatase may have been caused by something else. In Case 1 the thrombus began to dissolve during heparin treatment (Fig. 4*B*). The remaining cases with incomplete filling of parts of the left lobe of the liver (Fig. 6; and 7) with contrast medium may in some cases be due to layering. It is important to examine the patient in a prone position as well, or to make a selective injection of the left portal branch to confirm the diagnosis. Selective examination has been advocated by Piccone *et al.*,[7] among others. In the cases with a definite thrombosis, however, no subjective or objective symptoms were evident. The left branch of the portal vein is most susceptible to thrombosis where the umbilical vein enters it and where there may be some rough surfaces following cannulation. Experience indicates that one should be aware of the peril of thrombus formation but that it is not a contraindication to the examination.

We have found it useful to apply the same technique in catheterization of the portal vein and its tributaries as in arteriography and venography. The Ödman-Ledin catheters and flexible guidewires are easy to manipulate and in most cases the desired sites are possible to reach, *e.g.*, pancreatic veins for phlebography and sampling of blood.[2] The 2 episodes of extravasation that have occurred in connection with this examination did not give the patients any symptoms.

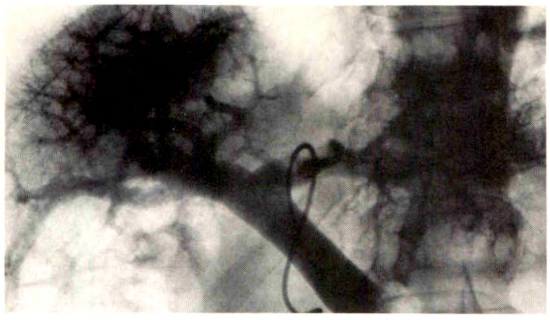

Fig. 7. Thrombosis of the main left branch of the portal vein. The flow of contrast medium to the left lobe of the liver is slower than to the right.

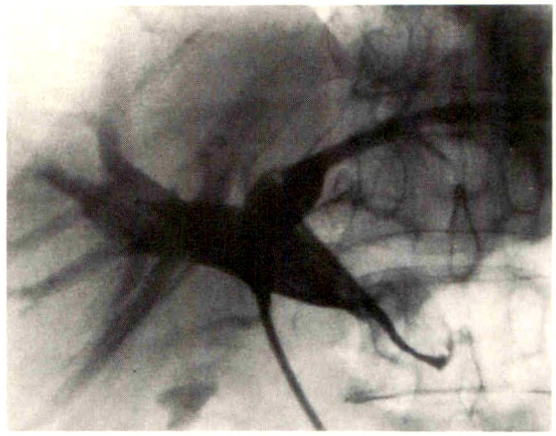

Fig. 8. Extravasation of contrast medium in the main portal vein. This extravasation was the most extensive found.

Recannulation 1 to 2 years after the first approach was made in 2 of our patients without difficulties. Kessler *et al.*[3] made repeat cannulations in 24 patients.

In removing the catheter from the umbilical vein we recommend stepwise withdrawal as has been advocated by Braastad *et al.*[1] and Wiechel and associates.[11] We have had no bleeding from the umbilical vein after catheter withdrawal.

Finally, we believe that access to the portal system *via* the umbilical vein is reasonable. The technique is safe and is to be preferred in hospitals where the transhepatic technique has not been or cannot be developed. With good technique the only complication to be considered is thrombosis and this resolves without sequelae.

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REFERENCES

1. Braastad, F. W., Wukasch, D. C., and Jordan, P. H. Practicality of umbilical vein cannulation in patients with and without portal hypertension. *A.M.A. Arch. Surg.*, 1970, *101*, 32–35.
2. Göthlin, J., Lunderquist, A., and Tylén, U. Selective phlebography of pancreas. *Acta radiol.* (Diag.), 1974, *15*, 474–480.
3. Kessler, R. E., Tice, D. A., and Zimmon, D. S. Value, complications and limitations of um-

bilical vein catheterization. *Surg., Gynec. & Obst.*, 1973, *136*, 529–535.

4. Kiessling, J., Rieche, K., Wirbatz, W., Mateeu, B., and Ziegenbein, B. Transumbilikale Blutuntersuchungen am Menschen aus der Vena portae. *Acta hepatogastroenterol.*, 1972, *19*, 148–156.
5. Lavoie, P., Jacob, M., Leduc, J., Legare, A., and Vialett, A. Umbilicoportal approach for study of splanchnic circulation: technical, radiological and hemodynamic considerations. *Canad. J. Surg.*, 1966, *9*, 338–341.
6. Piccone, V. A., Bonnano, P., and Leveen, H. H. Clinical and research uses of reopened adult umbilical vein. *Surgery*, 1968, *63*, 29–37.
7. Piccone, V. A., Leveen, H. H., White, J. J., Skinner, G. B., and Maclean, L. D. Transumbilical portal hepatography: significant adjunct in investigation of liver disease. *Surgery*, 1967, *61*, 333–346.
8. Storer, E. H., and Akin, T. J. Chemotherapy of hepatic neoplasms via umbilical-portal vein. *Am. J. Surg.*, 1966, *111*, 56–58.
9. Watts, J. M., and Douglas, M. C. Clinical applications and complications of umbilical vein cannulation. *Brit. J. Surg.*, 1971, *58*, 61–66.
10. White, J. J., Skinner, G. B., and Maclean, L. D. Hepatoportography via umbilical vein: superior approach to diagnosis in liver disease. *Canad. M. J.*, 1966, *95*, 997–1003.
11. Wiechel, K. L., Erwald, R., Kulling, K. G., Lundbergh, P., Marions, O., and Strandell, T. Multiple catheterization technique of portal venous system for regional blood flow studies. *Surg., Gynec. & Obst.*, 1974, *138*, 406–412.

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EXTERNAL ILIAC ARTERY BLOOD FLOW IN PATIENTS WITH ARTERIOSCLEROSIS OBLITERANS*

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ABSTRACT:

Blood flow in the left external iliac artery was measured by means of a densitometric technique in 21 patients with arteriosclerosis of the lower extremities and in 21 controls. Normal flow volumes were (mean ± S.D.) 604 ± 104 ml./min. for males and 478 ± ml./min. for females. Flow volume was significantly lower in the patients with ischemia of the legs. There was no correlation between flow volume through the artery and the degree of stenosis at its narrowest point. The diameter of the iliac artery and the flow volume were significantly larger in males than in females; however, flow velocity was significantly larger in females.

ACCURATE assessment of the circulation of the lower extremities is essential in reconstructive vascular surgery. The clinical examination will indicate the severity of the ischemia and routine angiography will supply morphological information. Not infrequently, however, in the presence of several lesions of different severity along the arterial tree, more accurate information regarding the volume of flow through the different segments would be helpful in determining the correct plan of action.

A recent modification of cineangiodensitometry has enabled us to study flow volume and velocity through segments of the arterial tree in patients.[5] This study is performed at the time of routine angiography and does not necessitate a separate catheterization.

The present report describes the results of flow measurements through the external iliac arteries of 21 normal subjects and 21 patients with clinical arteriosclerosis obliterans of the lower extremities.

MATERIAL AND METHOD

Our method has been previously described.[5] It consists of cineangiographic recording of the artery in question during injection of a bolus of contrast material, and a densitometric evaluation of the washout of this material at 2 selected sites along the vessel using an optic electronic system. This densitometric analysis yields 2 indicator dilution curves. The time interval between these 2 curves corresponds to the transit time of the bolus between the 2 points along the vessel.

By measuring the diameter of the vessel and the distance between the 2 points, flow volume and velocity can be derived:

$$\text{Flow volume} = \text{Volume}/\text{Time} = \pi r^2 L/T$$
$$\text{Flow velocity} = L/T$$

Where r is the radius of the vessel, L is the distance between the 2 points and T is the transit time.

This densitometric system for flow measurement has been shown to be both accurate and reproducible *in vitro*,[5] with an error of ± 5 per cent.

The examination is performed by catheterization of the femoral artery using the Seldinger technique. The tip of the catheter is advanced above the bifurcation of the aorta. Twelve cc. of dye are injected and a cineangiographic recording of the left external iliac artery is performed. The

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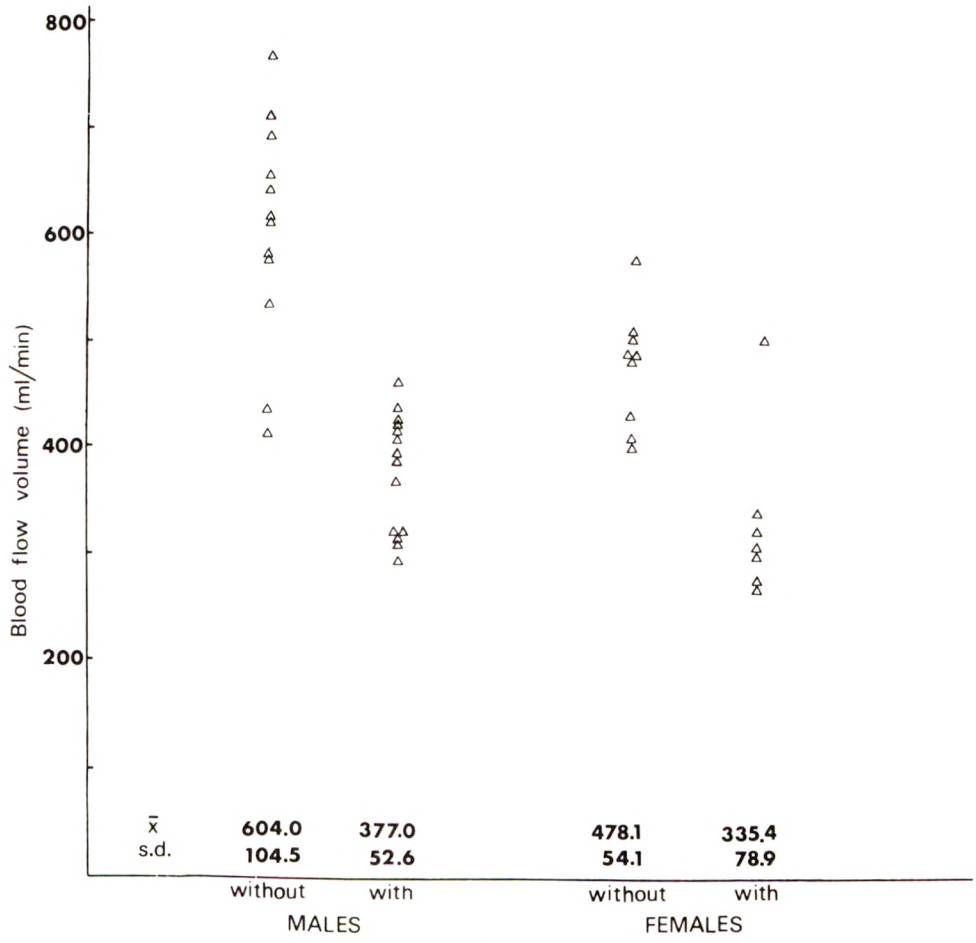

Fig. 1. Distribution of blood flow volume in external iliac artery in males and females over the age of 50, with and without clinical arteriosclerosis obliterans of the lower extremities.

planned angiography is then completed with the same catheter.

Twenty-one subjects over the age of 50 (12 males and 9 females) with no symptom or sign of vascular disease of the lower extremities were studied during abdominal angiography performed for evaluation of non-acute conditions. These 21 patients were used as controls ("normals").

Twenty-one patients over 50 (14 males and 7 females) with moderate to severe intermittent claudication due to arteriosclerosis obliterans were studied during aortography performed as part of the preoperative evaluations ("patients").

Statistical methods: Comparison of mean blood flow volume was performed by the Mann-Whitney non-parametric test because of unequal variances in the various groups.[6] Comparisons of mean diameter and flow velocity were performed using the *t* test.

RESULTS

The value for blood flow volume (ml./min.) for the 21 normals and 21 patients is given in Figure 1. The mean blood flow volume for the normal males was 604 (ml./min.) with a s.d. of 104.5 and 90 per cent tolerance limits for 90 per cent of the population 353–855. For the normal females the respective values were 478 ± 54.1 for the mean and s.d., and 336–620 for the tolerance limits. For the male patients the

Table I

INDIVIDUAL VALUES FOR DIAMETER OF EXTERNAL ILIAC ARTERY, DIAMETER AT ITS NARROWEST POINT, AND BLOOD FLOW VELOCITY IN PATIENTS

Case No.	Sex	Age	Diameter (mm.)	Narrowest Point (mm.)	Flow Volume (ml./min.)
1.	M	52	6.5	4.8	323
2.	M	54	6.7	6.1	298
3.	M	56	6.1	5.1	415
4.	M	57	7.3	5.1	410
5.	M	58	8.2	3.9	420
6.	M	58	7.0	4.6	394
7.	M	64	7.3	5.6	389
8.	M	64	5.8	5.0	317
9.	M	65	7.2	3.8	323
10.	M	66	6.2	3.6	462
11.	M	67	8.2	5.9	436
12.	M	68	5.6	3.1	370
13.	M	71	6.0	4.9	315
14.	M	71	5.2	5.2	406
15.	F	54	5.0	3.9	310
16.	F	55	4.8	4.1	281
17.	F	60	5.6	4.9	346
18.	F	61	5.7	5.3	323
19.	F	61	6.2	4.1	506
20.	F	64	4.7	3.9	305
21.	F	69	4.1	3.5	277

mean and s.d. were 377 ± 52.6 and for the female patients 335 ± 78.9. The mean blood flow volume for the patients was significantly lower than for the normals. (For males p (Mann-Whitney) <0.0001, and for females p (Mann-Whitney) <0.01.) The mean blood flow volume for females was significantly lower than for males in both normals (p (Mann-Whitney) <0.01) and in patients (p (Mann-Whitney) $=0.04$.)

Individual values of flow volumes, diameter of the external iliac artery, and the diameter of the vessel at its narrowest point in the 21 patients are given in Table I. The correlation between the degree of narrowing and flow volume is plotted in Figure 2. The absence of correlation between these 2 variables is striking ($r=0.014$).

The mean diameter and blood flow velocity (in cm./sec.) for the 21 normals is given in Table II. For the diameter in males the mean and s.d. were 7.72 ± 0.87 mm. and for females they were 6.37 ± 0.59 mm. For the blood flow velocity in males the mean and s.d. were 21 ± 4.29 cm./sec. and for females they were 26 ± 4.64 cm./sec.

Whereas both diameter and blood flow volume were significantly larger in males than in females (for diameter $p[t] <0.001$; for flow volume p (Mann-Whitney) <0.01), blood flow velocity was significantly lower in males ($p[t]=0.02$).

DISCUSSION

The results of flow measurements in this series fall within the range of results of flow measurements performed by several other methods.[1,3,4] Our results for flow volume in normals are higher than in some series[2,3] and approach the levels recorded by Ganz *et al.*[4]

The advantage of the present method is that it does not necessitate an additional intervention, and can be performed during routine angiography.

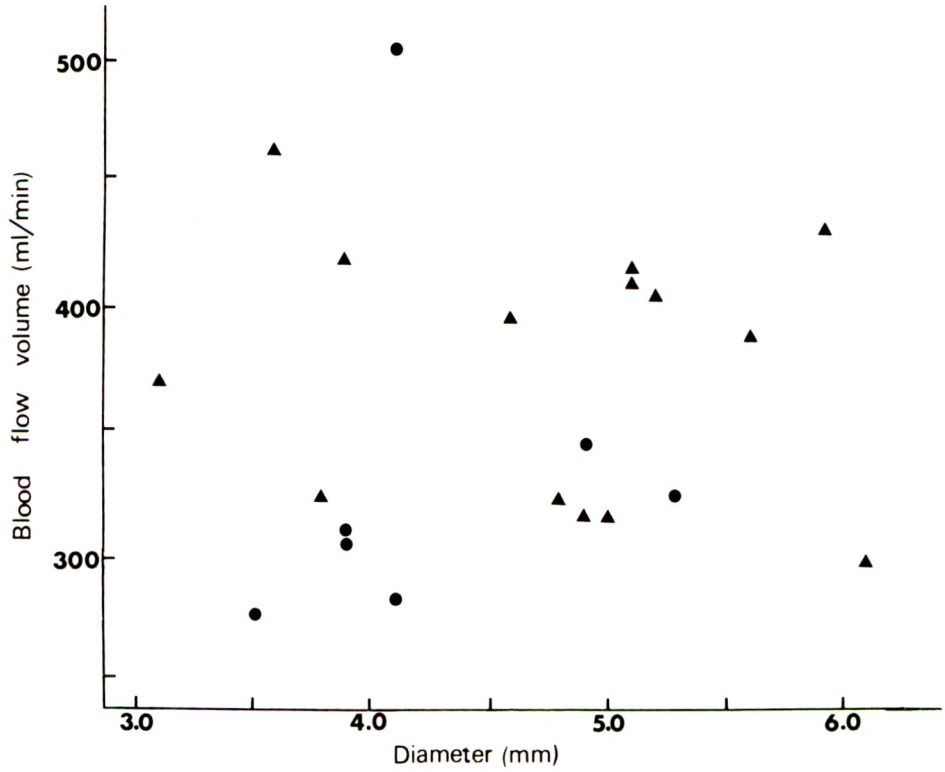

FIG. 2. Correlation of blood flow volume with diameter at the point of greatest narrowing of the external iliac artery in males (black triangles) and females (black dots), with arteriosclerosis obliterans of the lower extremities.

TABLE II

INDIVIDUAL VALUES FOR DIAMETER OF EXTERNAL ILIAC ARTERY, BLOOD FLOW VELOCITY AND BLOOD FLOW VOLUME IN NORMALS

Case No.	Sex	Age	Diameter (mm.)	Flow Velocity (cm./sec.)	Flow Volume (ml./min.)
1.	M	51	7.8	24.1	695
2.	M	54	6.9	25.9	581
3.	M	55	7.6	22.7	615
4.	M	56	6.6	28.5	585
5.	M	58	9.1	18.1	711
6.	M	60	7.8	22.3	640
7.	M	63	8.3	13.5	438
8.	M	67	8.7	17.3	620
9.	M	68	6.5	23.7	766
10.	M	70	6.7	19.6	415
11.	M	72	8.2	16.7	528
12.	M	74	8.4	19.6	654
13.	F	54	5.4	31.7	435
14.	F	55	6.1	30.0	512
15.	F	61	5.8	26.0	405
16.	F	63	6.6	23.1	479
17.	F	64	7.2	21.0	510
18.	F	66	7.1	20.0	485
19.	F	69	6.1	33.1	578
20.	F	71	6.6	23.6	484
21.	F	78	6.4	27.0	415

The absence of correlation between the degree of stenosis of the external iliac artery and the flow volume was not unexpected. The degree of narrowing of the diameter in the anteroposterior projection in these patients was not extreme and exceeded 50 per cent in only 1 patient. With this degree of narrowing, the effect of the peripheral resistance on flow appears to be more marked than the effect of the decrease in diameter. This explains relatively large flow volumes in some patients with a considerable narrowing of the vessel, as well as low flow volumes in some patients with a relatively minor narrowing. For this reason an angiographically demonstrated decrease in the diameter of a blood vessel in 1 projection cannot be considered in any way as an index of flow.

The lower flow volumes in females, both in normals and in patients, is probably related to the smaller muscle mass of the lower extremities. The higher flow velocity in females has not been stressed previously, and might possibly be a compensatory effect for the smaller diameter. Further data obtained from different vascular beds and using other methods of flow velocity measurement may contribute to an understanding of this observation.

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REFERENCES

1. Bliss, B. P. Pressure, flow and peripheral resistance measurements during surgery for femoropopliteal occlusion. *Scandinav. J. Clin. Lab. Invest.*, 1973, Suppl. 128, pp. 179–184.
2. Dedichen, H. Prognostic significance of intraoperative flow and pressure measurements in reconstructive vascular surgery. *Scandinav. J. Clin. Lab. Invest.*, 1973, Suppl. 128, pp. 189–194.
3. Dedichen, H., and Kordt, K. F. Blood flow in normal human ileofemoral arteries studied with electromagnetic technique. *Acta chir. scandinav.*, 1974, *140*, 371–376.
4. Ganz, V., Hlavova, A., Linhart, J., and Prerovsky, I. Measurement of blood flow in femoral artery in man at rest and during exercise by local thermodilution. *Circulation*, 1964, *30*, 86–93.
5. Itzchak, Y., Yerushalmi, S., and Deutsch, V. Blood flow measurements in iliac arteries by improved angiographic cinedensitometric technique. *Invest. Radiol.*, 1975, *10*, 1–9.
6. Siegel, S. Non-parametric Statistics for Behavioral Sciences. McGraw-Hill Book Company, Inc., New York, 1956, pp. 116–126.

MESENTERIC ANGIOGRAPHY OF NON-TROPICAL SPRUE*

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ABSTRACT:

Detailed angiographic findings in 2 cases of proved sprue are described. There are dilatation of the superior mesenteric artery and its small bowel branches selectively, and increased capillary blush. Early draining into dilated veins is also noted. These findings are similar to those of the superior mesenteric arteriogram performed with pharmacological vasodilators. This similarity suggests that the dilated vessels in non-tropical sprue may be due to capillary dilatation.

NON-TROPICAL sprue is one of the common malabsorption syndromes. The small bowel roentgenographic findings in sprue are well described in the literature. However, we could find only 1 reference in the English language literature which described the angiographic findings of sprue.[2] Recently we have seen 2 patients who had abdominal angiography and were proved to have non-tropical sprue. The purpose of this communication is to report the details of the superior mesenteric angiographic findings in these 2 patients.

REPORT OF CASES

CASE 1. (M.K.) This 42 year old white male was admitted with a history of a 14 lb. weight loss during the previous 2 months and with anemia. Upper gastrointestinal and small bowel studies done elsewhere disclosed a grossly abnormal small bowel pattern with the suggestion of nodularity involving the proximal small bowel. He always had had a good appetite but complained of feeling weak. The stool had changed to loose and greasy for a while; then it became normal in character and frequency (once a day) without any specific treatment for the last several weeks. Physical examination on admission showed a temperature of 102°F. and slight distention of the abdomen. Laboratory tests showed a hemoglobin of 10.7 gm., white blood cell count 6,700 with normal differential, and slight elevation of alkaline phosphatase (107 mU/cc.). Urinalysis was within normal limits. D-xylose absorption was 2.2 gm. in 5 hours (normal: greater than 5 gm. per 5 hours). Chest roentgenograms showed no abnormality. Upper gastrointestinal study showed a dilated second portion of the duodenum with the suggestion of extrinsic pressure along the superior and lateral aspects of the apex of the bulb. The small bowel was moderately to markedly dilated with several areas of intermittent intussusception and scattering of barium (Fig. 1). These findings were consistent with sprue. On duodenoscopic examination, there was a large extrinsic mass along the lateral aspect of duod-

FIG. 1. Case 1. The small bowel is dilated. Scattering of barium and several areas of intermittent intussusception are seen.

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enum. A biopsy of the duodenum showed flattened villi consistent with non-tropical sprue. However, the 14 lb. weight loss during the last 2 months and normal bowel habits persuaded the attending physician to search for a possible hidden malignancy. A lymphangiogram showed no abnormality.

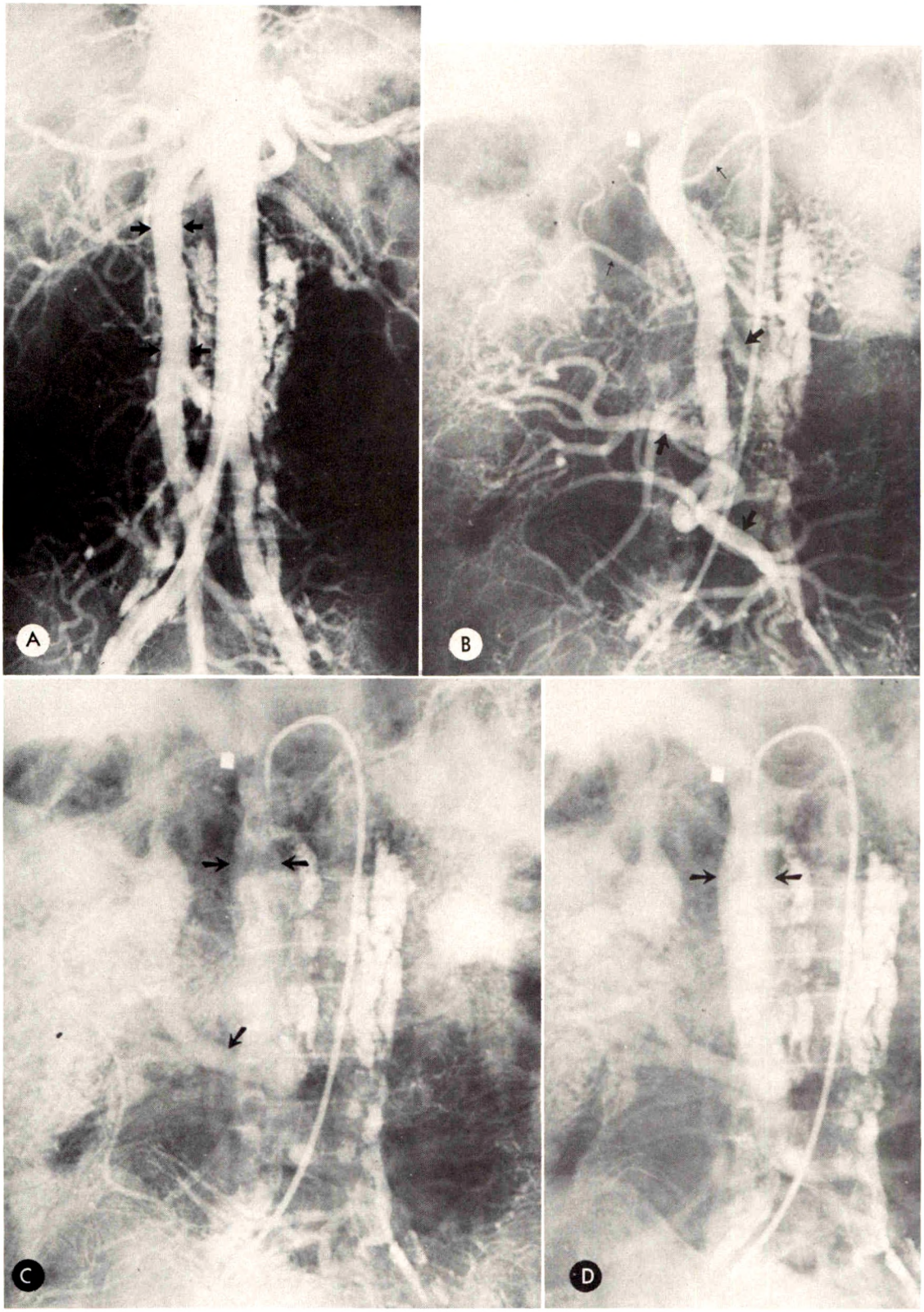
Abdominal angiography was performed, and on the mid-stream aortic injection, the superior mesenteric artery was large, measuring 1.2 cm. in diameter (abdominal aorta measured 1.7 cm.) (Fig. 2*A*). Selective catheterization of the superior mesenteric artery showed that the main superior mesenteric artery and most of the jejunal and ileal branches were markedly dilated (Fig. 2*B*). However, the right and middle colic arteries were normal in diameter. There was a diffuse but somewhat patchy increased capillary blush of the small bowel wall. No irregularity of the arteries was noted and the arterial phase was otherwise normal. At 7 seconds, the superior mesenteric vein was opacified while arterial capillaries were still seen (Fig. 2*C*). The superior mesenteric vein and its branches were well opacified and dilated (Fig. 2*D*). The patient was subjected to exploratory laparotomy. At operation, there was rather diffuse mesenteric lymphadenopathy with lymph nodes of varying sizes. Marked hypervascularity of the mesentery of the small bowel was noted. The small bowel itself was dilated, atonic and somewhat thickened. Several areas of intussusception were found. A full-thickness biopsy of jejunum as well as removal of multiple lymph nodes was performed. Histologic examination showed non-tropical sprue and reactive hyperplasia of multiple lymph nodes. The patient was discharged on gluten-free diet and has been gaining weight.

Case II. (J.G.) This 55 year old white male was admitted, with complaints of occasional diarrhea and small caliber stool, for further study of his malabsorption syndrome. He had been hospitalized elsewhere in August, 1974, for ecchymoses on the skin of his neck, face, and thigh. Non-tropical sprue was proved by small bowel biopsy. He had been on gluten-free diet, with added folic acid, iron, and Vitamin K without much help. His diarrhea was watery and yellowish but not foamy. He claimed that food did not alter his diarrhea. Physical examination was essentially within normal limits. The abdomen was soft and not distended, and no abnormal mass was felt. The bowel sounds were active. His blood cell count was normal. Serum albumin was 2.4 gm., alkaline phosphatase was 230 mU/cc., and uric acid was 8.9 mg. per cent. Stool examination for parasites and ova was negative. Chest roentgenogram and barium enema examination were within normal limits. An upper gastrointestinal and small bowel examination demonstrated marked dilatation of the small bowel with scattering of barium (Fig. 3). The small bowel findings were consistent with sprue but because of the history of lack of improvement with a gluten-free diet, other possibilities were considered. Abdominal angiography was done as part of his work-up.

The superior mesenteric artery as well as most of the small bowel branches were markedly dilated (Fig. 4*A*). The right and middle colic arteries were normal in diameter. Increased diffuse but patchy capillary blush was noted in the entire small bowel wall. The arterial phase was otherwise normal. The superior mesenteric vein as well as its branches were well opacified early at 8 seconds and were dilated (Fig. 4*B*). A review of the previous biopsy confirmed that changes were consistent with non-tropical sprue. The attending physician asked the patient's wife (who is a nurse) to bring the samples of all the food they purchased at the health food store. To the physician's surprise, the foods were gluten-rich instead of gluten-free. The patient was discharged with a gluten-free diet. Follow-up bowel study after 1 month showed marked improvement in the abnormalities. The patient is now almost asymptomatic.

DISCUSSION

The role of mesenteric angiography in the evaluation of the malabsorption syndrome is not established. There are a few reports of angiographic findings of inflammatory bowel disease, mainly Crohn's disease and ulcerative colitis. Boijsen described a widened superior mesenteric artery, poor opacification of the superior mesenteric vein, and no evidence of shunting in sprue.[2] Our observations of these 2 cases differ slightly from those of Boijsen. In the aortic injection, the superior mesenteric artery is much wider than normal compared to the other arteries. In the arterial phase of the superior mesenteric


A
B
C
D

artery injection, there is selective dilatation of jejunal and ileal branches. The right colic and middle colic branches of the superior mesenteric artery are normal in caliber. There is diffuse but somewhat patchy increased blush in the small bowel wall during the capillary phase. The entire superior mesenteric venous system and portal vein are dilated, seen early, and well opacified.

The etiology of dilatation of the mesenteric vasculature in non-tropical sprue is not certain. It probably reflects an aspect of the pathophysiology of the disease because of the selective dilatation. Because non-tropical sprue may be due to an allergic phenomenon,[8] Boijsen suggested that the dilated superior mesenteric artery probably represents vasospasm in the small bowel villi where blood is short-circuited to the veins.[2] Our observation of increased and rather diffuse capillary blush of the small bowel is difficult to explain on the basis of vasospasm and short-circuiting of blood. The dilated superior mesenteric artery and vein, increased blush of the small bowel wall, and early and good opacification of veins are seen in the superior mesenteric arteriogram with vasodilative pharmacological agents.[3,7] The superior mesenteric angiograms in our 2 cases of sprue are quite similar to those of pharmacoangiography with vasodilators. This similarity suggests that the capillaries are dilated, either due to vasodilatation secondary to the disease itself or possibly to increased demand for blood. An allergic or toxic aspect of non-tropical sprue might be responsible for the capillary dilatation.

It is not certain what would happen to the dilated vessels in non-tropical sprue when the patient becomes asymptomatic.

Fig. 3. Case II. The small bowel is markedly dilated, and scattering of barium is also seen.

It is conceivable that the vessels might become normal or less dilated when sprue is controlled long enough.

Diffuse hypervascularity of the small bowel may not be unique in sprue. Segmental dilatation of branches of the mesenteric artery has been described in chronic inflammatory bowel diseases, such as Crohn's disease, ulcerative colitis, and actinomycosis.[1,4–6] Not only were those arteries supplying the diseased segments dilated, but other abnormal findings, such as irregular, tortuous vessels or abnormal tapering were present. Abnormal vasculature is seen with tumors, but is relatively localized. Lymphosarcoma of the small bowel may be diffuse but is generally not more than minimally hypervascular and

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Fig. 2. Case I. (*A*) Mid-stream aortic injection. Arrows show markedly dilated superior mesenteric artery. (*B*) The selective superior mesenteric artery injection demonstrates dilated small bowel branches (large arrows) and normal colic branches (small arrows). (*C*) At 7 seconds, the superior mesenteric vein and its branches are opacified (arrows) as well as the portal vein, while arterial capillaries are still seen. There is increased capillary blush of the small bowel wall. (*D*) At 10 seconds, excellent opacification of the mesenteric vein and its branches is seen.

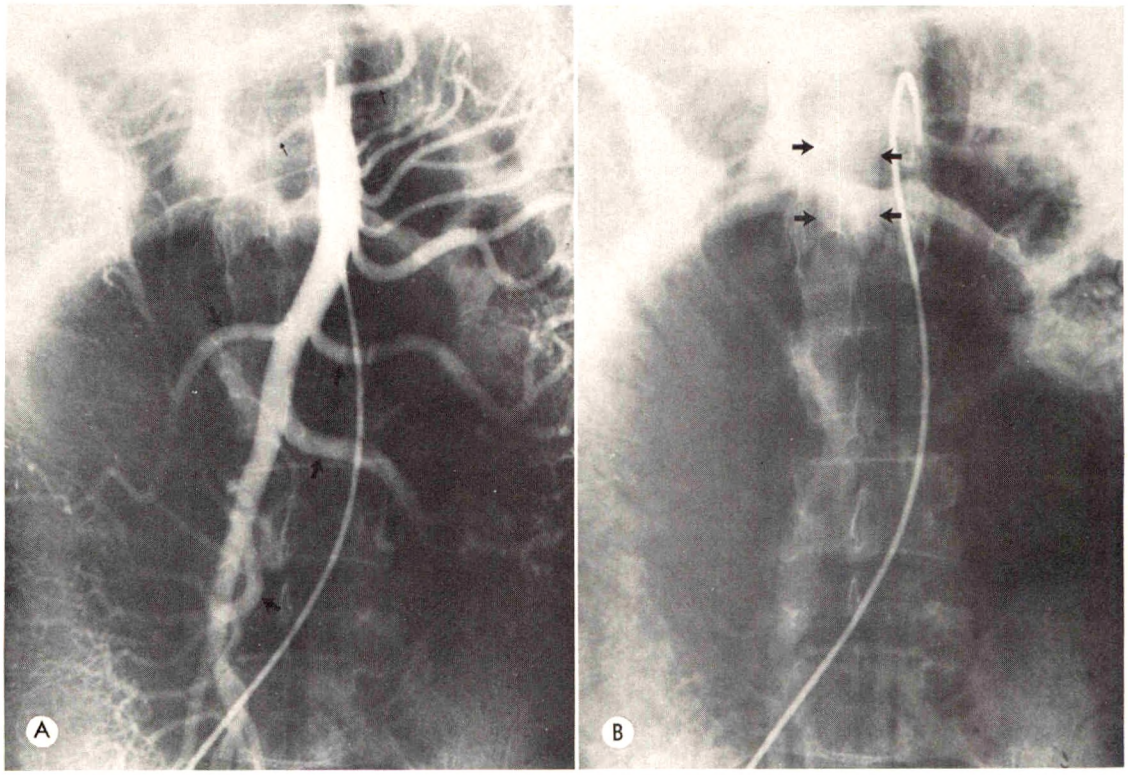

Fig. 4. Case 11. (*A*) Selective superior mesenteric artery injection demonstrates dilatation of the superior mesenteric artery and its small bowel branches (large arrows); the colic branches are normal (small arrows). (*B*) At 11 seconds, the superior mesenteric vein and its branches are well opacified and dilated. An increased capillary blush is seen in the small bowel wall, displaced by the air-filled sigmoid.

may present irregular or displaced vessels.

Mesenteric angiography thus can be helpful for evaluation of the malabsorption syndrome, especially as with our 2 patients where it was important to rule out tumor.

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REFERENCES

1. Boijsen, E., and Reuter, S. R. Mesenteric angiography in evaluation of inflammatory and neoplastic diseases of intestine. *Radiology*, 1966, *87*, 1028–1036.
2. Boijsen, E. Mesenteric angiography. In: Angiography. Volume II. Second edition. Edited by H. L. Abrams. Little, Brown & Company, Boston, 1971, pp. 1104–1105.
3. Danford, R. O., and Davidson, A. J. Use of glucagon as vasodilator in visceral angiography. *Radiology*, 1969, *93*, 173–175.
4. Lunderquist, A., and Lunderquist, A. Angiography in ulcerative colitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 18–23.
5. Lunderquist, A., Lunderquist, A., and Knutsson, H. Angiography in Crohn's disease of small bowel and colon. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 338–344.
6. Olsson, T. Angiography in actinomycosis of abdomen: report of 2 cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med , 1974, *122*, 278–280.
7. Steekal, R. J., Rosch, J., Ross, G., and Grollman, J. H. New development in pharmacoangiography (and arterial pharmacotherapy) of gastrointestinal tract. *Invest. Radiol.*, 1971, *6*, 199–211.
8. Taylor, K. B., and Truelove, S. C. Immunological reactions in gastrointestinal disease: review. *Gut*, 1962, *3*, 277–288.

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PERSISTENT HYPEREXTENSION OF THE NECK IN BREECH ("STAR-GAZING FETUS") AND IN TRANSVERSE LIE ("FLYING-FETUS"): INDICATION FOR CESAREAN SECTION

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ABSTRACT:

All pregnancies with a breech or transverse lie should be examined roentgenologically, at least after the onset of labor, and those in whom hyperextension of the neck persists should be sectioned to avoid the real danger of injury to the cervical cord incurred in vaginal delivery. The radiologist should not assume a seriously deformed fetus just from the hyperextension; most of these fetuses are otherwise normal.

THE purpose of this note is to better acquaint radiologists with the dangers of vaginal delivery of those fetuses with persistent hyperextension of the neck in breech and in transverse lie, a topic that has received only scant attention in the radiologic literature.

Properly, roentgenology is being used less and less in obstetrics with the advent of the safer modalities of ultrasound and of isotopes, but it continues indispensable for morphology of the fetus and of the pelvis, and still finds widespread use in many obstetrical problems in the smaller communities. It is especially in the latter that the radiologist might find opportunity to advise the accoucheur who might not be familiar with the problem of hyperextended neck.

The fetal pathologist recognizes many causes for the hyperextended neck: hamartoma, vascular tumor, teratoma, encephalomyelocele, parasitic fetus, ectopia cordis, arthrogryposis, and sternocleidomastoid muscle mass, among others.[7] Iniencephaly may be detected with certainty *in utero*.[4] Amniography may better define the neck mass,[3] but failure to detect such a mass is no indication that vaginal delivery is safe. Indeed, most of these cases of persistent hyperextended neck in breech and in transverse lie are not associated with a local mass.

Such a hyperextended neck is not rare; some estimates reach as high as 5 per cent of all breech presentations. Some hyperextensions are corrected with the onset of labor, and with these we are not concerned. The cause of the persistent hyperextended neck is not known, but spasm of the cervical musculature has been suggested.[6] The extended neck can persist postnatally, but usually disappears by the first or second month.

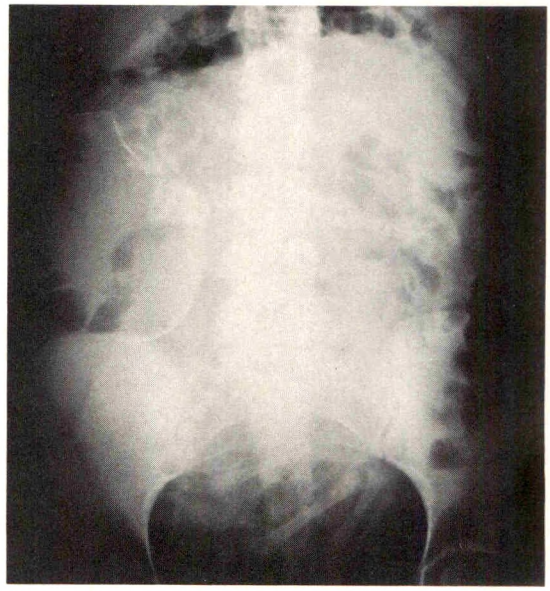

FIG. 1. Anteroposterior view shows fetus in breech with hyperextended neck giving "star gazing" appearance.

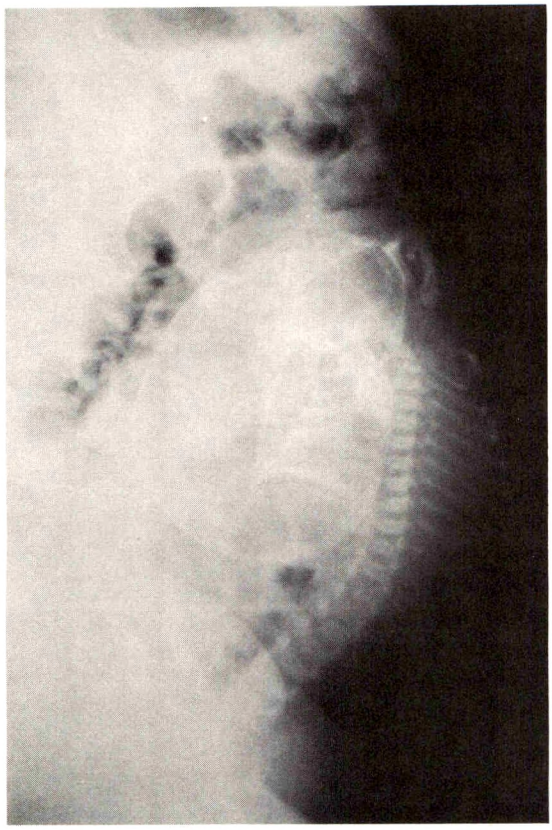

Fig. 2. Lateral view demonstrates similar findings.

There is a high incidence of injury to the cervical cord in these infants who have been delivered vaginally, probably attendant upon the extreme flexion of the head occurring in the birth canal. Of 88 cases of persistent hyperextension of the neck in breech and in transverse lie, 31 were delivered by section without a single permanent spinal cord injury; but of the 57 cases delivered vaginally, 12 (21 per cent) suffered a transection of the spinal cord.[1]

The injury is usually in the lower cervical cord, and consists of epidural and medullary hemorrhage, with lacerations and softening of the cord substance. Such injury may not be roentgenologically considered because there is commonly no bony counterpart, and the neurologic deficit may be attributed to brain injury.

There are 3 clinical presentations, and the radiologist might expect consultation in the final two:

1. Stillbirth, or sudden death soon after delivery
2. Short survival with shock, respiratory depression, and secondary changes in the lung mimicking hyaline membrane disease
3. Long-term survival with transient or permanent paralysis and spasticity.

REPORT OF A CASE

A 17 year old primipara had a normal prenatal course, noteworthy only because of a fetus in breech with hyperextended neck confirmed by roentgenography (Fig. 1; and 2). An infant with hyperextended neck was delivered by cesarean section. There were no other clinical or roentgenologic abnormalities. The neck was flexed manually without complication. The child continues to do well after 2 months of postnatal observation.

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REFERENCES

1. Abroms, I. F., Bresnan, M. J., Zuckerman, J. E., Fisher, E. G., and Strand, R. Cervical cord injuries secondary to hyperextension of head in breech presentations. *Obst. & Gynec.*, 1973, *41*, 369–378.
2. Bhagwanani, S. G., Price, H. V., Lawrence, K. M., and Ginz, B. Risks and prevention of cervical cord injuries in management of breech presentation with hyperextension of fetal head. *Am. J. Obst. & Gynec.*, 1973, *115*, 1159–1161.
3. Cash, M. B., and Kornmesser, J. G. Diagnosis of fetal neck mass by amniograpny. *Radiology*, 1968, *91*, 476–477.
4. Cimmino, C. V., and Painter, J. W. Iniencephaly. *Radiology*, 1962, *76*, 942–944.
5. Daw, E. Hyperextension of head in breech presentation. *Am. J. Obst. & Gynec.*, 1974, *119*, 564–565.
6. Gibberd, G. F. Muscular spasm in foetus: factor in production of malposition. *J. Obst. & Gynaec. Brit. Emp.*, 1935, *42*, 596–599.
7. Potter, E. L. Pathology of the Fetus and the Infant. Second edition. Year Book Publishers Inc., Chicago, 1961.

Vol. 125, No. 2

CALCIFIED INTRALUMINAL MECONIUM IN NEWBORN MALES WITH IMPERFORATE ANUS

ENTEROLITHIASIS IN THE NEWBORN

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ABSTRACT:

Five cases are reported and reference is made to 3 previous similar cases of calcified intraluminal meconium in newborn males with "imperforate anus." A rectourinary fistula was found in most of the patients; none of the cases had meconium peritonitis. The calcifications may develop in areas of prolonged stasis; the possible added role of the mixture of urine and meconium *in utero* is raised.

NORMALLY the diagnosis of meconium peritonitis is made when abdominal calcifications are detected in roentgenograms of newborns with intestinal obstruction. There has been a prenatal perforation and the patient has developed a sterile inflammatory response often associated with matted loops of intestine, presenting a problem of surgical and medical management.

Five cases are reported where multiple foci of calcified meconium have been noted on preoperative abdominal roentgenograms of newborn males with imperforate anus. The meconium in all cases was intraluminal; none of the cases had coexistent meconium peritonitis. All 5 patients were males with supralevator rectal atresia; in 4 of the 5 fistula was demonstrated between the distal colon and either the bladder or urethra; in the fifth case it may have been present but was not commented on at autopsy.

The possible action of urine on meconium to produce such calcific precipitates is discussed, as well as the role of stasis of intestinal secretions.

REPORT OF CASES

CASE 1. (Babies Hospital, N.Y., N.Y.) A full term twin male infant had marked abdominal distention at birth. Multiple anomalies were obvious (Table 1) and a nasogastric tube could not be passed into the stomach. The anal opening was absent. Transferred to Babies Hospital, the infant was found on roentgenographic studies to have esophageal atresia with distal tracheoesophageal fistula, as well as duodenal atresia (Fig. 1*A*). Osseous abnormalities included an anomalous sacrum, missing lumbar pedicles, a rudimentary left thumb, and left radio-ulnar elbow fusion. There was a single umbilical artery.

The patient was not operated upon in view of the many anomalies as well as his developing seizures and progressive deterioration. At autopsy a great variety of malformations was noted (Table 1) including those of the Vater association.[7] The calcifications were not due to meconium peritonitis. Rather, the colon was distended and contained a semiliquid suspension with gritty clumps that were calcified. The large bowel seemed to end blindly at the level of the prostate; initial dissection indeed failed to reveal a fistula. With careful sectioning of the prostate, a rectourethral fistula was found (Fig. 1, *B* and *C*). The urethra distal to

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Table I

CASE I: ANOMALIES AT NECROPSY

Gastrointestinal Tract
Esophageal atresia
Tracheoesophageal fistula
Duodenal atresia, supra-ampullary type
Gallbladder atresia, cystic duct atresia
Hypoplastic common bile duct
Malrotation, fenestrated mesentery
Liver—oval shape with absent caudate and quadrate lobe
Rectal atresia with rectoposterior urethral fistula
Genitourinary Tract
Urethral hypoplasia, severe
Kidney—excessive fetal lobulation
Respiratory
Hypoplastic lungs
Hyaline membrane disease, pulmonary hemorrhage
Choanal atresia, left
Cardiac
Patent ductus arteriosus
Atrial septal defect, Ostium II
Ventricular septal defect, high posterior
Bone
Radioulnar synarthrosis, left
Sacrum, abnormal segmentation
Thumb, missing metacarpal, left
Vertebra—lumbar, missing pedicles
Miscellaneous
Single umbilical artery
Hand, left simian crease

the fistula was markedly hypoplastic. The colon contents showed high urea nitrogen, creatinine, and uric acid levels, as well as high concentration of calcium and phosphorus, in addition to bilirubin and other meconium constituents. The colon mucosa was necrotic.

Comment. Although the fistula was difficult to find, the colon contents showed urine as a significant component. The hypoplastic urethral lumen distal to the fistula would have caused an obligatory *in utero* passage of urine into the colon. The reason for the necrosis of the colon mucosa was not apparent. The calcified concretions were felt to be meconium foci containing bilirubin as well as calcium and phosphorus precipitates.

Case II. (The Children's Hospital, St. Paul, Minn.) A full term male newborn had an obvious imperforate anus. Roentgen study showed (Fig. 2) scattered abdominal calcifications of 1–2 mm. diameter that were initially in the right lower quadrant but shifted on later roentgenograms to the left upper abdomen. There were many anomalous dorsal vertebrae and ribs. The preoperative diagnosis of meconium peritonitis was made. At the time of the transverse colostomy, there was no evidence of peritonitis. Colon irrigations led to passage of meconium masses that contained gritty clumps of calcified meconium. A voiding cystogram showed a widely patent fistula to the rectum with massive reflux to the right kidney. Intravenous pyelography failed to show a functioning left kidney. The patient subsequently developed hydrocephalus; he cannot walk, and definitive colon surgery has been postponed.

Case III. (The Children's Hospital, St. Paul, Minn.) A premature male twin with imperforate anus was observed to pass meconium in his urine. Preoperative roentgenograms showed scattered abdominal calcifications initially in the right lower quadrant and later in the left abdomen (Fig. 3). Severe respiratory distress developed requiring oxygen and assisted ventilation. A diverting colostomy was performed; there was no meconium peritonitis. Following colon irrigation, postoperative roentgenograms showed a complete disappearance of the calcifications. The infant lived several months requiring increasingly high concentrations of oxygen and larger ventilatory pressures; he died at several months of age and there was no autopsy.

Comment on Cases II *and* III. Both cases showed preoperative abdominal calcifications that puzzled the observers by shifting location from the right to the left abdomen. All of the calcifications disappeared following colostomy and cleansing enemas. The rectourinary fistula in Case II was confirmed by voiding cystography while Case III urinated meconium.

Case IV. (The Children's Hospital, Basel, Switzerland.) A full term male infant had an obvious imperforate anus at birth. Preoperative roentgenograms of the abdomen (Fig. 4, *A* and *B*) showed a markedly hypoplastic sacrum. A few small bowel loops were filled with

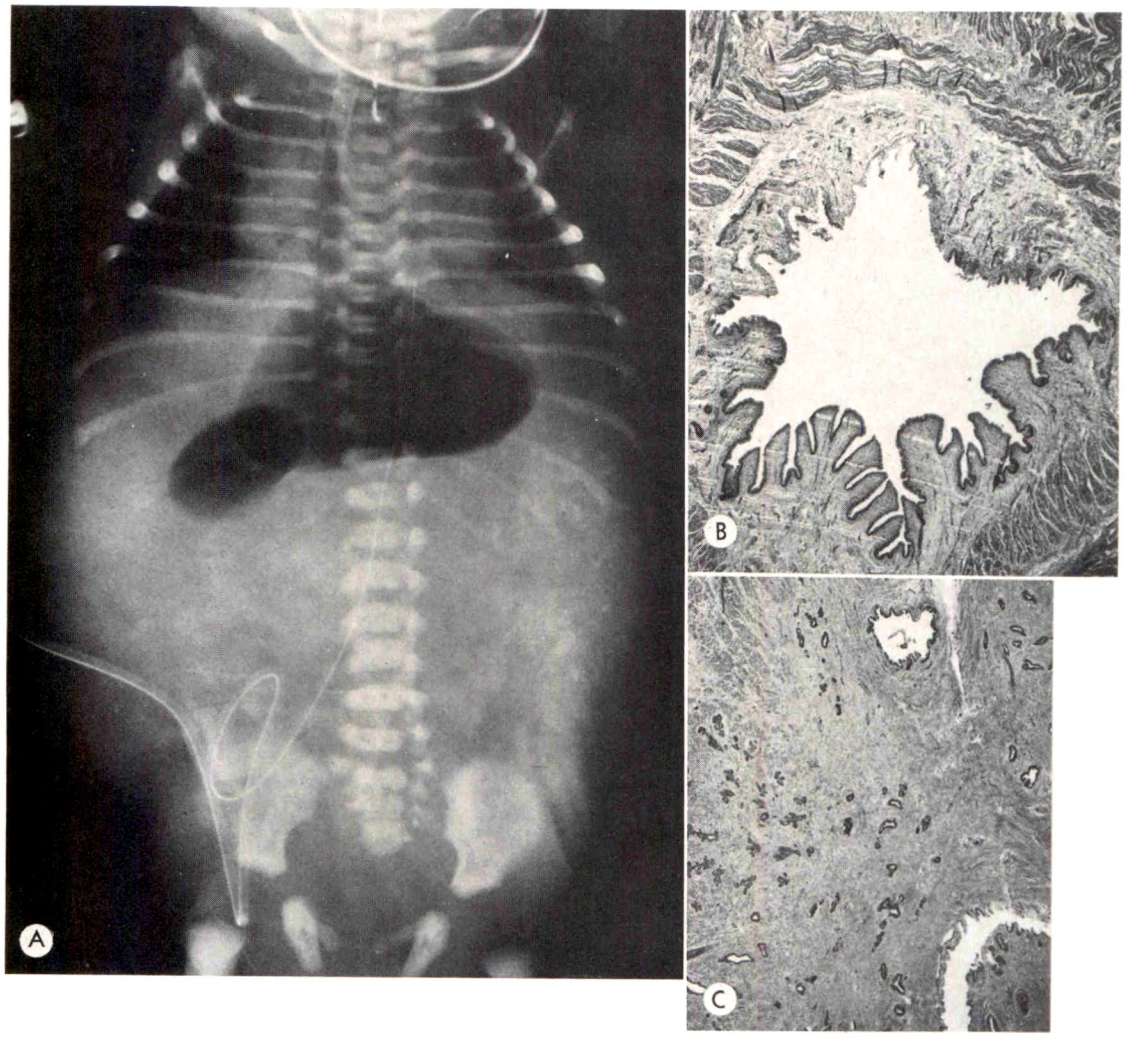

Fig. 1. Case 1. (Babies Hospital, N.Y., N.Y.) Newborn male with esophageal atresia, tracheoesophageal fistula and duodenal atresia, as well as imperforate anus. (*A*) Note numerous calcifications best seen in left flank as well as deformed sacrum and missing pedicle right side of L 1. These were part of the Vater association of anomalies. (*B*) Distal rectum in its connection to the urethra was lined in part by partially sloughed large bowel mucosa (upper half) and in part by urothelium (lower half). (HPS×99.) (*C*) Recto-urethral fistula (upper lumen) communicated with posterior urethra (lower right). (HPS×25.)

gas but the abdomen was mainly gasless with bulging flanks. There were many 1–2 mm. calcified foci. A transverse colostomy was performed and the colon cleansed of a large amount of meconium which contained gritty foci that proved to be calcified. The colon terminated at the level of the prostate; there was no meconium peritonitis. Severe cyanotic congenital heart disease was noted and definitive colon surgery was deferred. The boy died at 5 years of age of his heart disease. At autopsy there was a rectovesical fistula.

Comment. The colon was so filled with meconium and the distal small bowel with both fluid and meconium that the major portion of the abdomen appeared "gasless," although the only organic obstruction was rectal atresia.

Case v. (Los Altos Hospital, Long Beach, Calif.) A 2,400 gram male infant was noted to have imperforate anus at birth. Severe respiratory distress was the immediate problem and

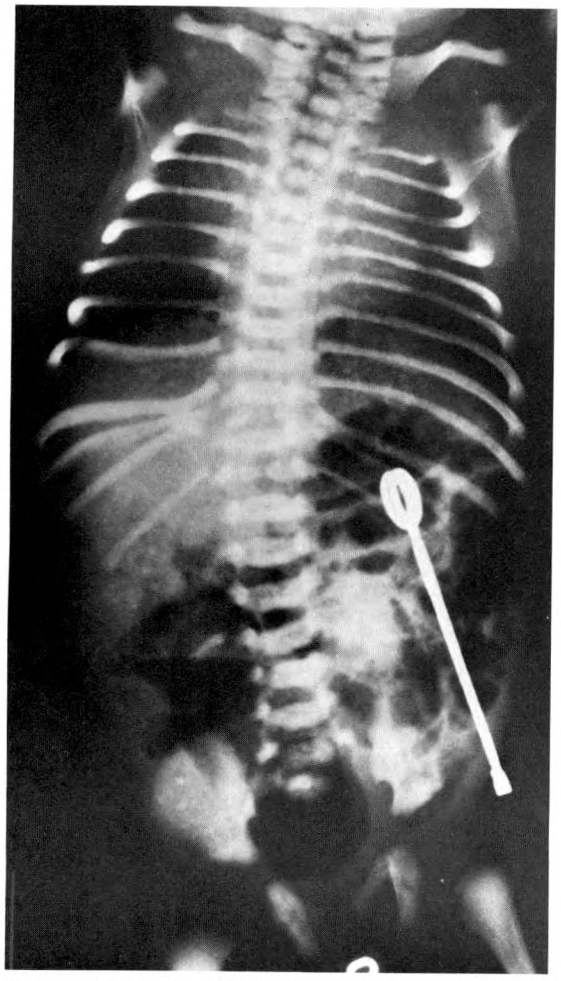

Fig. 2. Case II. (Children's Hospital, St. Paul, Minn.) Newborn male with imperforate anus. Note numerous rib and vertebral segment anomalies. Sacrum is stubby. Scattered calcifications in right side of abdomen disappeared after evacuation of meconium at the time of colostomy.

roentgenograms showed (Fig. 5) a right pneumothorax and large pneumomediastinum in addition to cardiomegaly. The abdomen showed scattered calcifications in the right flank with a markedly malformed lumbosacral spine. The pelvic bony outlet was contracted. The child was markedly acidotic and died in a few hours. Autopsy showed the kidneys and ureters to be absent. The bladder was large and thick walled, measuring 7 cm. in diameter. Its contents were not commented on. There was no note of peritonitis. The anatomic relationship of the colon to the bladder and urethra was not commented on.

Comment. Despite the tantalizingly incomplete autopsy protocol, this case is included because the infant had both abdominal calcifications and imperforate anus without meconium peritonitis. He differs from the preceding 4 cases in one crucial respect; they all had functioning kidneys and a rectourinary fistula. Case V had no kidneys, although there may well have been a fistula to the rectum: the large bladder was obviously distended by something other than urine. Unfortunately, the autopsy report failed to note its contents or the colon contents.

The other interest in the case lies in the appearance of the pelvic bony outlet, despite the large size of the bladder. In a previous communication[3] it was suggested that such an underdeveloped pelvic bony outlet bore no relation *per se* to the presence of kidneys or bladder size *per se*,[2] but rather, was probably a bony adaptation to altered muscular forces, these in turn reflecting disturbed innervation.[6]

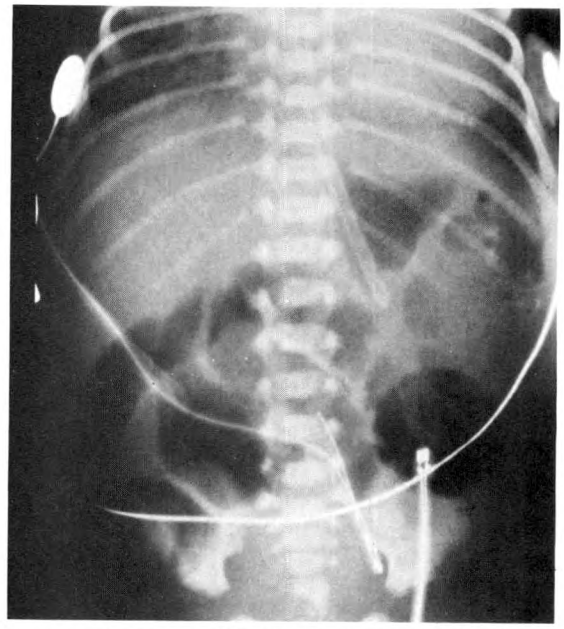

Fig. 3. Case III. (Children's Hospital, St. Paul, Minn.) Newborn male with imperforate anus. Note deformed fifth sacral segment. Scattered calcifications in left upper quadrant had moved from right lower quadrant; these disappeared after evacuation of meconium at time of colostomy.

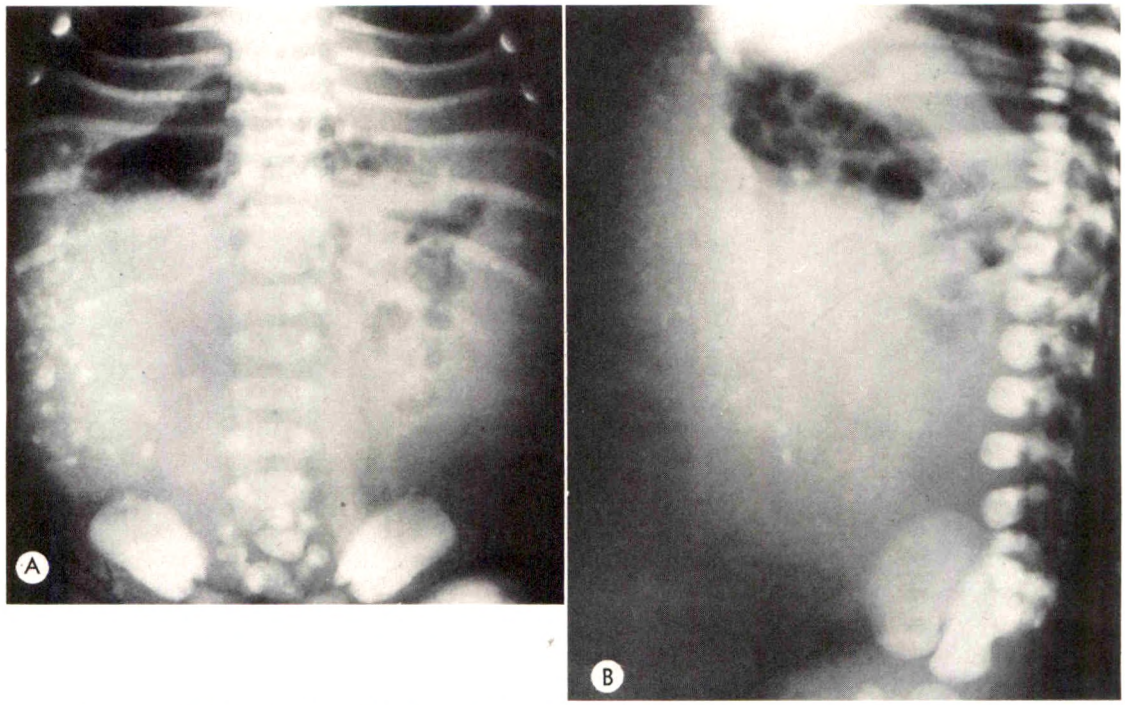

Fig. 4. Case IV. (Children's Hospital, Basel, Switzerland.) (*A* and *B*) Newborn male with imperforate anus. Note markedly defective formation of lower lumbar spine and sacrum. Most of the abdomen appears gasless due to marked distention of colon by meconium; scattered calcifications are all in lumen of colon.

REVIEW OF PRIOR REPORTED CASES

The 5 previously published cases of calcified intraluminal meconium (with coexistent meconium peritonitis) have fallen into 2 groups: those with colon obstruction[4,5] (Table II) and those with small bowel obstruction[1,8] (Table III). None of the infants or their mothers had evidence of abnormal calcium or phosphorus metabolism.

Imperforate anus with intraluminal calcified meconium (*Table II*). In 1955 Khilnani *et al.*[5] reported on a case of "enterolithiasis in the newborn." The patient was a male with imperforate anus and rectourinary fistula. At surgery there was no evidence of meconium peritonitis although this had been suggested on preoperative roentgenograms that showed many calcified densities. All of the calcifications disappeared following colostomy irrigations.

In 1962 Hillcoat[4] added 2 additional cases. One patient had his colon terminating in the region of the prostate; the second was reported as an atresia of the anus and lower rectum without fistula. It can be argued that, as in Case 1, a fistula was indeed present but missed. All the calcifications were intraluminal.

Small intestinal obstruction with calcified intraluminal meconium (*Table III*). In 1949 Camp and Roberts[1] reported an infant male with ileal stenosis and multiple calcified intraluminal meconium foci. Rickham,[8] in 1957 had an even more bizarre case with multiple meconium calcifications in dilated ileal loops. No organic stenosis was present and calcification was noted in both the distal collapsed as well as the proximal dilated segments. Neither of these cases had meconium peritonitis.

DISCUSSION

Our five cases with calcified intraluminal meconium all had imperforate anus. Thus, adding these to the previously published cases, there were 8 of 10 patients with calci-

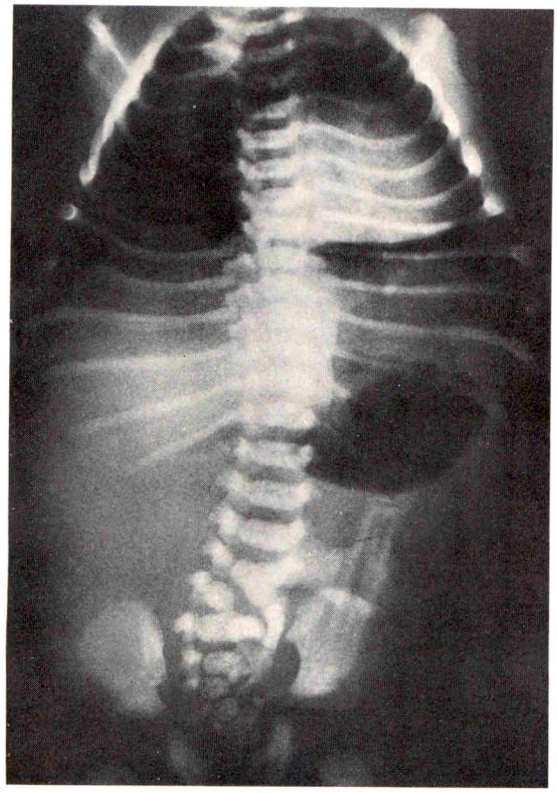

Fig. 5. Case v. (Los Altos Hospital, Long Beach, Calif.) Male with imperforate anus. Infant had renal agenesis and a huge bladder. Note deformed lumbosacral spine and scattered flank calcifications. Heart was surrounded by pneumomediastinum and patient died a respiratory death with hypoplastic lungs.

fied meconium without meconium peritonitis who had imperforate anus, and all were male. Five of the 8 cases of imperforate anus had a fistula demonstrated between the rectum and the urinary tract (bladder or urethra). This raises the possibility that the calcifications represent an interaction between urine and meconium. The problem with this explanation is that a rectourinary communication usually is present in males with the "high" form of imperforate anus; the flow of urine into the colon can be shown by voiding cystograms in such babies. Those who have actually had calcified meconium demonstrated in the colon lumen are few (5 in the current series, 3 from the prior literature).

Chemical study of meconium[9] yields no obvious answers. Meconium is a mixture of swallowed amniotic fluid, squamous cells, intestinal debris, bile salts, and succus entericus of the fetus. It is rich in protein, mucopolysaccharides, as well as calcium and phosphorus. There is no ready explanation of how or why fetal urine, with its dilute levels of urea and creatinine, should cause precipitation of calcium salts into the gritty concretions found in these patients. It should be mentioned that all fetuses have urine in their gastrointestinal tract since amniotic fluid is in a major degree voided urine in the last trimester of pregnancy. Stasis of intestinal contents has been suggested as an etiologic factor in prior reports of this finding of meconium "enterolithiasis."[5,8] An easily found example of calcification in an area of intestinal stasis would be seen in the appendix in the form of an appendicolith or fecalith. Meconium stasis was certainly present in the cases listed in Table II and the smaller group with bowel distention listed in Table III. The problem with this very reasonable

Table II

CALCIFIED INTRALUMINAL MECONIUM ASSOCIATED WITH IMPERFORATE ANUS

Author	Cases	Associated Meconium Peritonitis	Rectourinary Fistula Demonstrated
Hillcoat[4]	2	0/2	0/2
Khilnani *et al.*[5]	1	0/1	1/1
Current Cases (Berdon *et al.*)	5	0/5	4/5
Total	8	0/8	5/8

Table III

CALCIFIED INTRALUMINAL MECONIUM ASSOCIATED WITH SMALL INTESTINAL OBSTRUCTION

Author	Cases	Associated Meconium Peritonitis	Site of Obstruction
Camp and Roberts[1]	1	0/1	Ileum
Rickham[8]	1	0/1	Ileum
Total	2	0/2	2/2 Ileum

explanation is that thousands of infants with small bowel intestinal obstruction (whether from meconium ileus or ileal atresia or stenosis) have had preoperative abdominal roentgenograms with only 2 being found to have meconium enterolithiasis.[1,8] Similarly, equally huge numbers of infants with imperforate anus (with small or absent fistula) have had plain roentgenograms and only the few reported cases have shown these calcified foci.

The detection in the past of such calcifications has raised the preoperative roentgenographic diagnosis of meconium peritonitis. Meconium peritonitis results from leakage of meconium onto the serosal and parietal peritoneal surfaces of the abdominal cavity, usually presenting in the form of linear areas of calcification. Occasionally the calcification in such patients is in the wall of infarcted or ischemic bowel. It is most often associated with small intestinal atresia and has been attributed to prenatal volvulus or infarction on some other ischemic basis. It would be most unusual for peritoneal calcification to be associated with imperforate anus *per se* and most unlikely to appear in the form of numerous 1 to 2 mm. calcific densities.

To conclude, the cause of the intraluminal meconium enterolithiasis is unknown. Its finding in a male with imperforate anus (without a visible ectopic orifice) is strongly suggestive of the presence of a fistula; proof of the nature of the calcification is accomplished by a repeat abdominal roentgenogram after colostomy and evacuation of meconium masses and roentgenography of the evacuated meconium.

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REFERENCES

1. Camp, R., and Roberts, M. H. Multiple calcareous deposits in intestinal tract of newborn: report of case with stenosis of ileum. *Am. J. Dis. Child.*, 1949, *78*, 393–400.
2. Currarino, G. Association of congenitally small pelvic outlet with hypoplasia of bladder and urethra and absent kidneys. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 399–402.
3. Fellows, R. A., Berdon, W. E., and Baker, D. H. Size of pelvic bony outlet in renal agenesis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *121*, 159–165.
4. Hillcoat, B. L. Calcification of meconium within bowel of newborn. *Arch. Dis. Childhood*, 1962, *37*, 86–89.
5. Khilnani, M., Wolf, B. S., and Arnheim, E. E. Enterolithiasis in newborn. *J. Mt. Sinai Hosp.*, 1955, *22*, 58–61.
6. Magnus, R. V. Congenital rectovesical fistula and its associated anomalies. *Australian & New Zealand J. Surg.*, 1972, *42*, 197–204.
7. Quan, L., and Smith, D. W. Vater association-vertebral defects, anal atresia, tracheoesophageal fistula with esophageal atresia, radial and renal dysplasia: spectrum of associated defects. *J. Pediat.*, 1973, *82*, 104–107.
8. Rickham, P. P. Intraluminal intestinal calcification in newborn. *Arch. Dis. Childhood*, 1957, *32*, 31–34.
9. Schachter, H., and Dixon, G. J. Comparative study of proteins in normal meconium and in meconium ileus patients. *Canad. J. Biochem.*, 1965, *43*, 381–397.

PANCREATITIS AND THE BATTERED CHILD SYNDROME*

REPORT OF 2 CASES WITH SKELETAL INVOLVEMENT

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ABSTRACT:

Two cases of pancreatitis associated with the trauma of child abuse are reported. In the first case the skeletal survey was initially negative but the child later showed widespread roentgen evidence of intramedullary necrosis and periosteal new bone reaction. These lesions healed without treatment. In the second patient, the pancreatitis was accompanied by ascites and subsequently by bile peritonitis, duodenal obstruction, and a bile-filled abscess in the pancreas. This patient had skeletal fractures. The clinical problems of these 2 patients emphasize the need not only to relate pancreatitis to the battered child syndrome but also to distinguish between the bone changes due to direct trauma and those related to pancreatitis.

PANCREATITIS in children less than 5 years of age is unusual and should raise the question of trauma.[10,12] Recently, Hartley[5] reported the first case of "traumatic pancreatitis by battering." When a child with pancreatitis is hospitalized, a skeletal survey is indicated. This may reveal the osseous manifestations of the battered child syndrome (metaphyseal corner fractures, periosteal reaction, etc.). It is important to realize that the pancreatitis itself can give rise to widespread skeletal abnormalities.[6,9,13]

Two cases are presented which illustrate the clinical and roentgenographic spectrum of traumatic pancreatitis in children. In the first case, the skeletal survey was initially normal but the child went on to develop widespread painful skeletal lesions, characterized by intramedullary necrosis and periosteal new bone reaction. These were roentgenographically identical to findings described in leukemia, sickle infarction, and metastatic malignancy. With recognition of the pancreatitis,[9,13] it was possible to avoid repeat hospitalization and unnecessary diagnostic procedures. In the second case, the pancreatitis was accompanied by fractures of the long bones (although there was no admitted skeletal trauma) and aided in the diagnosis of the battered child syndrome.

REPORT OF CASES

Case 1. T.F., a 2 and 8/12 year old Black male was in good health until he was left with a baby sitter 40 hours prior to admission. When the mother returned she learned the patient had been admitted to a local hospital, allegedly because he had fallen. Subsequently, the baby sitter admitted beating the child.

At the time of admission at the referring hospital he was febrile, lethargic, vomiting bilious material, had a tender abdomen, and had multiple bruises of the head, neck, and abdominal wall. Over the next 36 hours, despite maintenance fluids, the patient became progressively oliguric and his blood urea nitrogen level rose to 80 mg. per cent. He was transferred to Babies Hospital for treatment of renal shutdown.

Physical examination on admission revealed the child to be anicteric with marked lethargy and to have a bruised, tender abdomen. His

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Fig. 1. Case 1. (*A*) Upper gastrointestinal barium study shows widened duodenal sweep with effacement of normal mucosal markings. A delay in barium transit beyond the ligament of Treitz was transient. (*B*) Roentgenograms of the feet show diffuse medullary destruction of metatarsals with periosteal new bone reaction. The phalanges are involved to a lesser extent. (*C*) Roentgenograms of the ankles show bilateral periosteal reaction surrounding the tibia and fibula; there is a large destructive lesion in the distal lateral right tibial metaphysis. The epiphyseal centers are spared. (*D*) Roentgenogram of the asymptomatic upper extremity shows similar medullary necrosis and periosteal reaction involving the ulna and metacarpals; the phalanges and humerus were involved to a lesser degree. Epiphyseal centers were spared. All lesions healed over several months without treatment or sequelae.

bowel sounds were hyperactive and there was no mass or rebound tenderness. After arrival the patient voided grossly bloody urine and continued to void over 500 cc. in the next 3 hours. The impression at the time of admission was trauma to the abdomen, possible pancreatitis, as well as central nervous system and renal trauma.

Pertinent laboratory tests included a serum amylase of 1,920 Meyers-Killian units (normal: under 35), a blood urea nitrogen of 72 mg. per cent, and a creatinine of 1.8 mg. per cent. Serum calcium was 6.8 mg. per cent and serum phosphorus was 1.4 mg. per cent. The next day urine amylase was 580 units per milligram of creatinine excreted (normal: less than 2.5 units per mg. of creatinine). Lumbar puncture, skull roentgenograms, echoencephalogram and other serum electrolytes were all normal. The sickledex was negative. Skeletal survey done on admission was within normal limits.

Three days after admission an intravenous pyelogram showed poor concentration of the contrast agent with normal kidneys. The upper gastrointestinal series done following the pyelogram revealed a widened duodenal sweep with edematous changes (Fig. 1*A*). With supportive therapy and antibiotics the patient slowly recovered.

On the seventeenth hospital day the patient

complained of pain in his feet and would not walk. Orthopedic evaluation revealed full range of motion in the feet, ankles, knees, and hips, and there was no swelling or tenderness. Neurological examination was within normal limits. By the twenty-fifth hospital day the child had begun to walk, although limping. He was discharged without roentgenography. Over the next several weeks the patient continued to limp and the mother had him seen by a private pediatrician who obtained roentgenograms of the feet (Fig. 1*B*). These showed a widespread intramedullary necrosis of the metatarsals and phalanges. There was considerable periosteal new bone reaction. Larger destructive lesions in the tibial metaphysis were also noted (Fig. 1*C*). The differential diagnosis at this time included disseminated malignancy and infection (sickle cell disease having been previously excluded). The patient's white blood cell count was normal and the child was actually spontaneously improving without therapy, about 1 week after the roentgenograms. The roentgenograms were reviewed at Babies Hospital and compared to the initial normal skeletal survey. At this time, the diagnosis of diffuse bone lesions accompanying pancreatitis was made. The upper extremities, which were clinically normal, were examined roentgenographically and they also showed widespread medullary destruction and periosteal new bone reaction (Fig. 1*D*). Only the epiphyseal centers were spared. The patient was observed without treatment over the next 5 months, and on his last follow-up examination the lesions were almost completely healed.

Comment. The diffuse skeletal abnormality seen in this patient 4 weeks after pancreatitis is striking, especially when compared to the initial negative skeletal survey. Although they could have represented widespread infection, malignant disease, or sickle dactylitis, their evolution and spontaneous regression paralleled the cases of pancreatitis described by Keating *et al.*[9] and Sperling[18] to such a degree that the investigations (bone marrow, blood cultures, bone biopsy) used by others and the attempt to modify the bone lesions with antibiotics could be avoided.

Case II. N.W. is a 19 month old white female who was in good health until 5 days prior to admission when her father placed her across his knee and "spanked" her. Soon after, she began to vomit coffee-ground material, became lethargic and oliguric. She was hospitalized and physical examination at that time revealed an agitated, bruised infant with hypoactive bowel sounds. The abdomen was not tender and there was no guarding or rebound. The initial impression was abdominal trauma, possibly pancreatitis secondary to child abuse.

Laboratory data revealed a markedly elevated serum amylase. Plain film study of the abdomen revealed no evidence of free air and only a dilated loop of jejunum. Upper gastrointestinal series, done with water-soluble contrast material showed a widened duodenal sweep and thick small bowel folds. Over the next 4 days, despite supportive therapy, her fever rose intermittently to 105° and her calcium dropped to 8 mg. per cent. She was then transferred to Babies Hospital.

Physical examination revealed a febrile, bruised infant with ascites. On an abdominal tap, 7 cc. of an orange-yellow fluid was removed. The ascitic fluid revealed an amylase of 980 Meyer-Killian units, a protein of 3,820 mg. per cent, and a calcium of 8.9 mg. per cent. An intravenous pyelogram showed the liver displaced medially by the ascites; the kidneys were normal (Fig. 2*A*). A bone survey showed long bone fractures of the right tibia and fibula, but was otherwise normal. A repeat upper gastrointestinal series using water-soluble contrast agent showed duodenal obstruction with a mass impinging on the medial aspect of the descending duodenum. This was felt to represent a duodenal hematoma (Fig. 2*B*).

The patient remained febrile and on the seventh day after injury an exploratory laparotomy revealed peritonitis secondary to acute hemorrhagic pancreatitis. Eight hundred cc. of darkly bile-stained fluid was found and there was extensive necrosis of the omentum and transverse mesocolon reflecting leakage of pancreatic enzymes. A large bile-stained cavity was found in the head of the pancreas. The entire pancreas was indurated and hemorrhagic, as were the structures of the lesser sac. Drainage tubes were inserted in Morrison's pouch and the lesser sac, as well as the placement of tubes in the stomach and gallbladder.

The postoperative course was complicated by a leak in the second portion of the duodenum, presumably in a necrotic area, as well as by severe nutritional problems. Hyperalimen-

tation was begun on the seventh hospital day. Her recovery was slow but she was discharged 3 months after admission. Repeat skeletal survey failed to show any evidence of medullary necrosis with periosteal new bone reaction.

Comment. Ascites and duodenal obstruction are unusual presentations of pancreatitis.[8] The patient was so severely ill that surgical drainage of the abscess was mandatory. Her recovery was made possible to a large degree by the use of hyperalimentation.

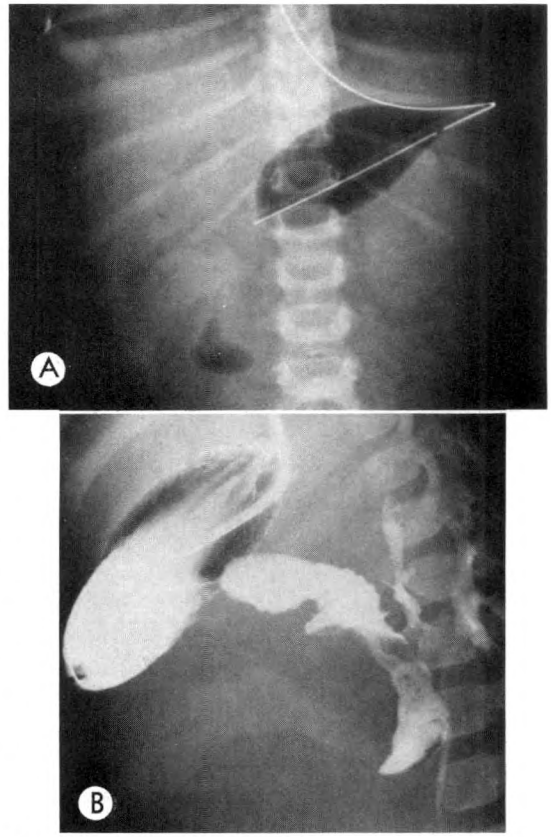


FIG. 2. Case II. (*A*) Intravenous pyelogram. Studies taken at the end of the injection show a bilateral nephrogram with faint excretion by non-obstructed kidneys in an oliguric patient. The liver, opacified by the circulating high levels of contrast material, is displaced medially by fluid. The patient proved to have peritonitis from acute pancreatitis. (*B*) Water-soluble contrast study shows duodenal obstruction. The descending duodenum shows a mass effect with proximal overhanging edges; this proved to be a bile-filled abscess in the head of the pancreas. The faint visualization of the transverse colon represents concentration of water-soluble contrast material given 4 days earlier when there was neither ascites nor duodenal obstruction.

DISCUSSION

Abdominal trauma is the most common cause of pancreatitis in the pediatric patient.[1,5,11,12] However, in the younger child who has been abused, the history of trauma may be difficult to obtain. Conversely, if a child presents with bruises and signs of abuse, traumatic pancreatitis must be considered and the clinical signs and symptoms recognized. Once trauma has been established, a search for skeletal injury must be performed (Table I).

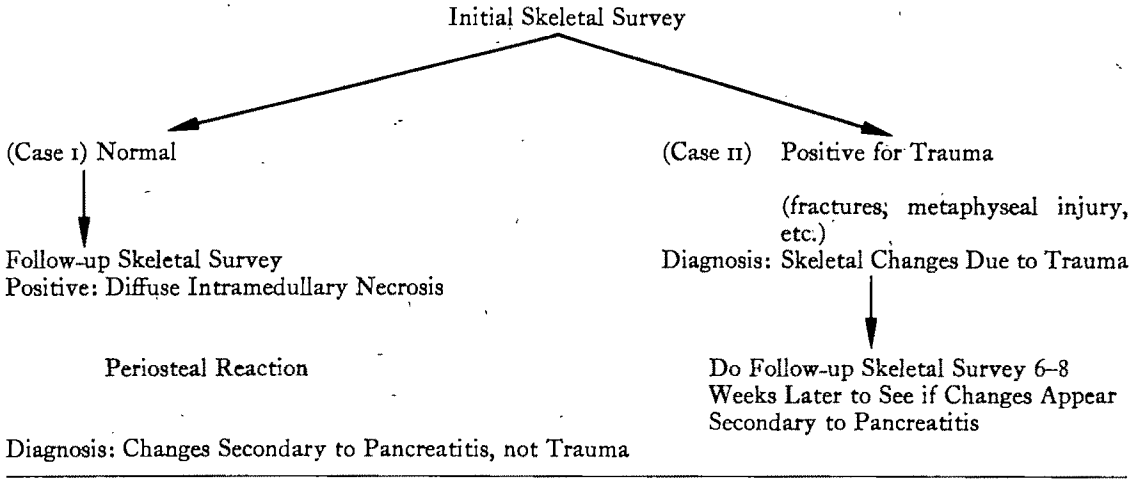
The abdominal roentgenographic signs of pancreatitis in children are well described.[14] Both of our patients demonstrated the appropriate coarsened duodenal mucosa and duodenal atony on contrast study. Case II had obstruction of the duodenum and widening of the duodenal loop. The mass obstructing the duodenum was proved to be an abscess involving the head of the pancreas.

The osteolytic lesions seen following pancreatitis are most likely the result of intramedullary fat necrosis.[6,7,13] The pancreatic enzymes, especially lipase, liberated within the pancreas during an acute inflammatory period, may reach the blood stream directly or *via* absorption from the peritoneal fluid into lymphatics and then into the circulation. One possible mechanism causing these roentgen findings is a reaction of pancreatic enzymes with the fat in the bone marrow causing hydrolysis and subsequent necrosis.[7] Although not found in our case, calcific stippling can be seen within these lesions and is caused by the combination of serum or tissue calcium with the hydrolyzed necrotic fatty marrow.[2,4]

The most striking roentgenographic feature is the involvement of virtually all tubular bones with epiphyseal sparing. The site of involvement parallels that seen in other disorders of the small blood vessels, especially diaphyseal and small bone

TABLE I

BONE LESIONS AND POST-TRAUMATIC PANCREATITIS



infarctions, as seen in sickle cell anemia. Healing is virtually complete unless the epiphyseal plate has been compromised, in which case the involved bone may be short. If the bone lesions are not appraised in view of the previous pancreatitis, fruitless roentgenographic surveys and surgical procedures for malignancy[13] can be undertaken. If hematogeneous osteomyelitis is thought of when the roentgenographic findings are noted, a long and ineffective course of intravenous antibiotics can be given without altering the course.[9]

A second type of bone lesion seen in adults with pancreatitis is aseptic necrosis, especially of the femoral heads.[1,2,7] Neither our patient nor other pediatric patients previously reported have had this kind of lesion. The pathophysiology of the aseptic necrosis in adults is felt to be bone infarction with ischemia caused by enzymatic damage to the endothelial cells of blood vessels resulting in thrombosis. This type of lesion is most often seen in adult patients with chronic pancreatitis.

In children with pancreatitis with widespread bone involvement it is not possible to distinguish between diffuse infarction and diffuse fat necrosis—indeed, both may be occurring. The sparing of the epiphysis may reflect the richness of the blood supply to the epiphysis which changes from childhood to adult life, adults having a less secure vascular anatomy. In any event, the pancreatic infarction pattern is identical to the presumed ischemic sickle pattern. Youngsters with sickle cell disease develop shaft and small tubular bone involvement while the adults with sickle cell disease develop epiphyseal involvement.

While multiple skeletal injury is well known in the battered child syndrome, it has been only recently that child abuse was described as the cause of pancreatitis.[3,10] The causal relationship between bone lesions and pancreatitis in children who have been abused is now confirmed. Diffuse periosteal reaction, lytic lesions, and the roentgenographic signs of pancreatitis should now be included among the roentgenographic findings of the battered child syndrome.

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REFERENCES

1. Blumenthal, H. T., and Probstein, J. G. Acute pancreatitis in newborn, in infancy and childhood. *Am. Surgeon*, 1961, *27*, 533–539.

2. Boswell, S. H., and Baylin, G. J. Metastatic fat necrosis and lytic bone lesions in patient with painless acute pancreatitis. *Radiology*, 1973, *106*, 85–86.
3. Caffey, J. Pediatric X-ray Diagnosis. Volume 2. Sixth edition. Yearbook Medical Publishers, Chicago, 1972, p. 1132.
4. Gerle, R. D., Walker, L. A., Achord, J. L., and Weens, H. S. Osseous changes in chronic pancreatitis. *Radiology*, 1965, *85*, 330–337.
5. Hartley, R. C. Pancreatitis under age of five years: report of three cases. *J. Pediat. Surg.*, 1967, *2*, 419–423.
6. Immelman, E. J., Bank, S., Krige, H., and Marks, I. N. Roentgenologic and clinical features of intramedullary fat necrosis in bones in acute and chronic pancreatitis. *Am. J. Med.*, 1964, *36*, 96–105.
7. Jaffe, H. L. Metabolic Degenerative and Inflammatory Disease of Bones and Joints. Chapter 22. Lea & Febiger, Philadelphia, 1972, p. 674.
8. Kalwinsky, D., Fritelli, G., and Oski, F. A. Pancreatitis presenting as unexplained ascites. *A.M.A. Am. J. Dis. Child.*, 1974, *128*, 734–736.
9. Keating, J. P., Shackelford, G. D., Shackelford, P. G., and Ternberg, J. I. Pancreatitis and osteolytic lesions. *J. Pediat.*, 1972, *81*, 350–353.
10. Kempe, C. H., Silverman, F. N., Steel, B. F., Droegemueller, W., and Silver, H. K. Battered child syndrome. *J.A.M.A.*, 1962, *181*, 17–24.
11. Leistyna, J. A., and Macaulay, J. C. Traumatic pancreatitis in childhood: report of case and review of literature. *A.M.A. Am. J. Dis. Child.*, 1964, *197*, 644–648.
12. Silverman, A., Roy, C. C., and Cozzetto, F. Pediatric Clinical Gastroenterology. C. V. Mosby Company, St. Louis, 1971, p. 470.
13. Sperling, M. A. Bone lesions in pancreatitis. *Australasian Ann. Med.*, 1968, *17*, 334–340.
14. Young, L. W., and Adams, J. T. Roentgenographic findings in localized trauma to pancreas in children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 639–648.

COMPLEMENTARY ROLE OF SONOGRAPHY AND ARTERIOGRAPHY IN MANAGEMENT OF UTERINE CHORIOCARCINOMA*

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ABSTRACT:

Uterine choriocarcinoma develops fairly frequently after passage of a hydatidiform mole and very rarely after normal pregnancy or abortion. The disease is highly curable by chemotherapy, especially if detected early. Histologic examination of uterine curettings is unreliable and the principal indicator of active primary or metastatic disease is the HCG titer.

The ability to visualize the tumor is helpful for a variety of reasons. In the past, this has been achieved primarily through arteriography. Our experience with 6 patients suggests that sonography is as sensitive a detector as arteriography and perhaps somewhat more specific. These facts, plus its convenience and repeatability, make ultrasound the method of choice for visualization of intrauterine malignant trophoblastic disease. Arteriography will continue to play an important role where the sonographic findings are equivocal and where local invasion or distant metastases are suspected.

UTERINE choriocarcinoma arises from trophoblast, the embryonic tissue which attaches the ovum to maternal endometrium. Although this tumor is relatively rare, its importance is enhanced in that it is presently the only form of cancer that can be consistently cured by chemotherapy.[1,4,10,12,18] Another important, if somewhat confusing, aspect of the disease is the fact that the diagnosis rests primarily upon a laboratory test—the urinary or serum human chorionic gonadotropin (HCG) titer—rather than upon histopathologic examination of tumor tissue. The latter has been found to be notoriously unreliable[8,9,10,12] because of difficulties in differentiating normal trophoblast, hydatidiform mole, and malignant trophoblast.

Because of this unreliability, it is helpful to have available other methods of tumor visualization. Until recently, the most reliable and widely-used method was arteriography.[2,3,5,6,9,20,22] During the past 2 years at our hospital, ultrasound has been used in addition to arteriography in the study of this disease and has proved exceedingly valuable. Our purpose in this paper is to describe the sonographic characteristics of uterine choriocarcinoma and to discuss the complementary role of the 2 modalities in management of these patients.

CLINICAL FEATURES

Approximately 50 per cent of cases of uterine choriocarcinoma develop after passage of a hydatidiform mole. Twenty-five per cent develop after normal pregnancy and 25 per cent after spontaneous or induced abortion.[5,12] The risk of development of choriocarcinoma in women who have had hydatidiform mole is from 2–8 per cent,[4,5,12] this being approximately 1,000 times greater than after normal pregnancy or abortion.[1] In addition, another 16 per cent of patients with hydatidiform mole go on to develop invasive mole, or chorioadenoma destruens.[8] The spectrum of malignant trophoblastic disease includes both choriocarcinoma and chorioadenoma

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destruens, although the latter is considered somewhat less dangerous and generally remains confined to the pelvic organs.

Elevation of the HCG titer is a highly sensitive indicator of the presence of malignant trophoblastic disease. It is not specific, however, since it also occurs with normal pregnancy, hydatidiform mole, or retained benign molar tissue following evacuation. Because the clinical index of suspicion is so high in women who have had a hydatidiform mole, they should be placed on oral contraceptives and undergo precautionary HCG assays every 2 weeks for the first 2 months after treatment, then every month for 1 year.[4,8,17] If the mole has been completely evacuated, the titer should return to normal within 60 days. If the titer instead begins to rise during this period, or remains elevated at its termination, a presumptive diagnosis of malignancy should be made. Diagnostic uterine curettage is not particularly helpful at this stage because of the difficulty in differentiating benign from malignant trophoblastic tissue. Even if no trophoblast whatsoever is recovered in the specimen, malignancy cannot be ruled out since the tumor cells may have burrowed too deeply into the myometrium to be reached by the curette.[9]

The development of choriocarcinoma after a full term or interrupted pregnancy is a more insidious process, since these women do not routinely undergo periodic HCG assay. Vaginal bleeding is generally the first symptom and this usually results in performance of a dilatation and curettage. Regardless of the histologic findings, the HCG titer should be determined in patients in this clinical setting.

Choriocarcinoma produces early and widespread metastases. Organs most frequently involved include the lungs, kidneys, liver, ovaries, brain, spleen, heart, and gastrointestinal tract.[8,11] Local invasion of the vaginal vault and parametrium is also common.

Treatment of malignant trophoblastic disease with chemotherapeutic agents has been remarkably effective, with cure rates in many comprehensive series ranging from 75–95 per cent.[1,4,10,12,17,18] The drugs of choice are methotrexate, or actinomycin D, or both in combination.[17] Lewis[17] has found that patients can be divided into high and low risk groups. In the latter group are those women with initial urinary HCG titers of less than 100,000 International Units (IU) per 24 hours, who are treated within the first 4 months after the presumed onset and whose metastatic disease is limited to the pelvic area and lungs. Those with higher initial titers, longer duration of disease, or metastatic lesions of the liver and brain are at greater risk but are still curable in 55–70 per cent of cases.[1,10,17]

ANGIOGRAPHIC FEATURES

The arteriographic characteristics of uterine choriocarcinoma have been well documented in earlier papers.[2,3,5,6,9,20,22] The uterine arteries are enlarged and very tortuous. Irregular vascular spaces and staining are seen during the capillary phase. The late phase generally shows dense opacification of the draining pelvic veins, although this is dependent to a certain extent upon the size and vascularity of the tumor. Direct shunting through arteriovenous fistulae is usually not present although this may develop after chemotherapy. Examples of arteriograms in choriocarcinoma are shown in Figure 1, *A* and *B* and Figure 2, *A–C*. The first case demonstrates diffuse involvement of the entire uterus; the second shows striking but more localized changes in a tumor that was found to be only 1.5 cm. in diameter at hysterectomy.

There is some question as to the source of the hypervascularity and staining which characterize choriocarcinoma angiographically. Contrast medium injections of resected specimens often show few or no abnormal vessels within the tumor,[3,5,22] although others contain considerable neovascularity.[2] It seems likely that the often dramatic *in vivo* arteriographic findings are due not to "tumor" vascularity and stain-

ing, but rather to an intense decidual reaction of the uterus itself. The increased vascularization of any uterus containing trophoblastic tissue constitutes a serious disadvantage in the use of arteriography, since it may cause other nonmalignant conditions to mimic choriocarcinoma. These include normal pregnancy,[3,9] missed abortion,[9] retained benign molar tissue after evacuation,[3] and some cases of hydatidiform mole before evacuation.[5]

SONOGRAPHIC FEATURES

Our initial interest in sonography in patients with this disease was stimulated by the nonspecificity of arteriography, as well as by the safety, simplicity, and repeatability of an ultrasound examination. During the past 2 years, 6 patients with suspected choriocarcinoma have been studied. A consistent sonographic pattern has been found in 5 of these women who actually had malignant trophoblastic disease. The tumors produce an amorphous pattern of intrauterine echoes which appear at low or normal sensitivity. The uterus is generally somewhat enlarged. The abnormal echo pattern correlated well with the location and size of the tumor as determined by arteriography or hysterectomy. Follow-up sonograms repeated at relatively short intervals during and after chemotherapy were highly accurate in documenting both disappearance of the lesions and later recurrence in some cases.

REPORT OF CASES

The patient whose studies are shown in Figure 1 was a 33 year old woman who developed vaginal bleeding and elevation of her urinary HCG titer (30,000 IU per 24 hours *vs.* a normal range of 100–200 IU) 5 months after passage of a hydatidiform mole. Arteriography (Fig. 1, *A* and *B*) demonstrated an extremely hypervascular lesion involving the entire uterus. Sonography (Fig. 1*C*) revealed an amorphous pattern of abnormal internal echoes throughout the organ. After 3 weeks of treatment with methotrexate and actinomycin D, her HCG titer had dropped significantly but was still somewhat elevated at 1,000 IU. Repeat sonography at this time (Fig. 1*D*) confirmed the presence of persistent tumor, as manifested by abnormal intrauterine echoes. The size of the uterus and degree of involvement both had diminished significantly. After another 3 weeks of chemotherapy, the HCG titer was normal, and sonography (Fig. 1*E*) showed an essentially normal uterus. Ten weeks later, however, her HCG titer was once again found to be elevated, and a sonogram (Fig. 1*F*) showed an increase in size of the uterus with a dense cluster of abnormal echoes on its anterior aspect. Repeat arteriography at this time (not shown) demonstrated abnormal vascularity and staining, although to a lesser extent than on the initial study. Because of this evidence of recurrent disease, hysterectomy was performed. The specimen contained a large focus of choriocarcinoma within the anterior wall.

The case shown in Figure 2 is that of a 26 year old woman who had aborted a hydatidiform mole at an outside hospital and then received multiple courses of chemotherapy in response to persistently high HCG titers. After 1 year, she was referred to the Downstate Medical Center where her initial work-up revealed a urinary HCG titer of 2,400 IU per 24 hours. Arteriography (Fig. 2, *A–C*) showed a small area of abnormal vascularity and staining on the left side of the uterus. Sonography (Fig. 2, *D* and *E*) showed uterine enlargement with a very localized cluster of abnormal internal echoes 2 cm. to the left of the midline. The patient's disease was considered to be refractory to chemotherapy, and hysterectomy was performed. Examination of the specimen demonstrated a 1.5 cm. choriocarcinoma nodule within the myometrium on the left side. This case indicates the accuracy with which both arteriography and ultrasound can detect small lesions of this type.

Figure 3 is a sonogram of a 20 year old woman who was found, on a routine chest roentgenogram, to have multiple pulmonary nodules 6 months after a spontaneous abortion. Her urinary HCG titer was 150,000 IU per 24 hours. Both arteriography and sonography suggested choriocarcinoma. The sonogram (Fig. 3*A*) showed an enlarged uterus with multiple abnormal internal echoes. The patient was given a single 5 day course of methotrexate. A HCG titer shortly thereafter was within normal limits and a follow-up sonogram (Fig. 3*B*) revealed a normal uterus. The patient has remained clinically well with normal titers during the ensuing 8 months.

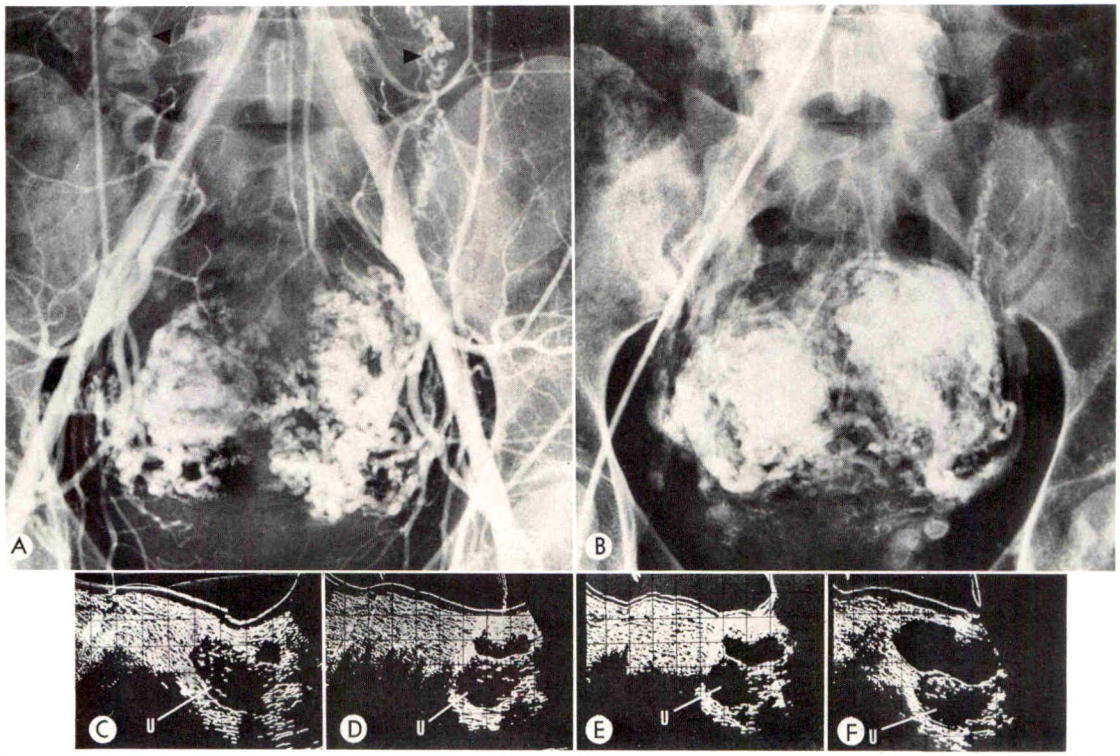

Fig. 1. (*A*) Arterial phase of a lower abdominal aortogram in a patient with uterine choriocarcinoma. The myometrial branches of both uterine arteries are markedly enlarged and tortuous. The ovarian arteries (black arrowheads) are also enlarged and contribute blood supply to the tumor. (*B*) Capillary phase. The entire uterus shows dense hypervascularity and irregular staining. (*C*) Midline longitudinal sonogram of the pelvis performed the same day as the arteriogram. On this and all subsequent sonograms, the patient's head is to the left of the photograph, the anterior abdominal wall faces upward, U = uterus, and the sonolucent structure anterior to the uterus is the filled bladder. Note the internal tumor echoes scattered throughout the enlarged uterus at this time. (*D*) After 3 weeks of chemotherapy, the uterus has decreased in size but some tumor echoes are still present, especially anteriorly. (*E*) After 3 more weeks of chemotherapy, the patient is clinically free of disease. Sonography shows the uterus at the upper limits of normal in size with no definite tumor echoes visualized. (*F*) Ten weeks later, the uterus has again enlarged and the cluster of abnormal echoes on its anterior aspect signifies recurrence of the tumor. This was confirmed by hysterectomy.

In 2 other women whose malignant trophoblastic disease was recognized by persistent HCG titer elevation after passage of a hydatidiform mole, sonography proved helpful in visualizing the tumor and following its progress during chemotherapy. The sixth patient was suspected of having choriocarcinoma on the basis of histologic examination of uterine curettings obtained after passage of a mole. However, her HCG titer was normal and sonography showed a perfectly normal uterus. On this basis, it was felt that the pathologic diagnosis was a false positive and no further treatment was given. The patient has remained free of any evidence of disease for over 1 year since then.

DISCUSSION

The value of sonography in hydatidiform mole is well established.[7,15,16] This report is the first discussion of its applicability to choriocarcinoma as well. Even though the primary determinant of activity of disease and need for treatment is the HCG titer, there is a number of reasons why visualization of the tumor is of value.

First and foremost, in a patient who develops an elevated titer after passage of a hydatidiform mole, the possibility of normal pregnancy must be ruled out before assuming the presence of malignant tropho-

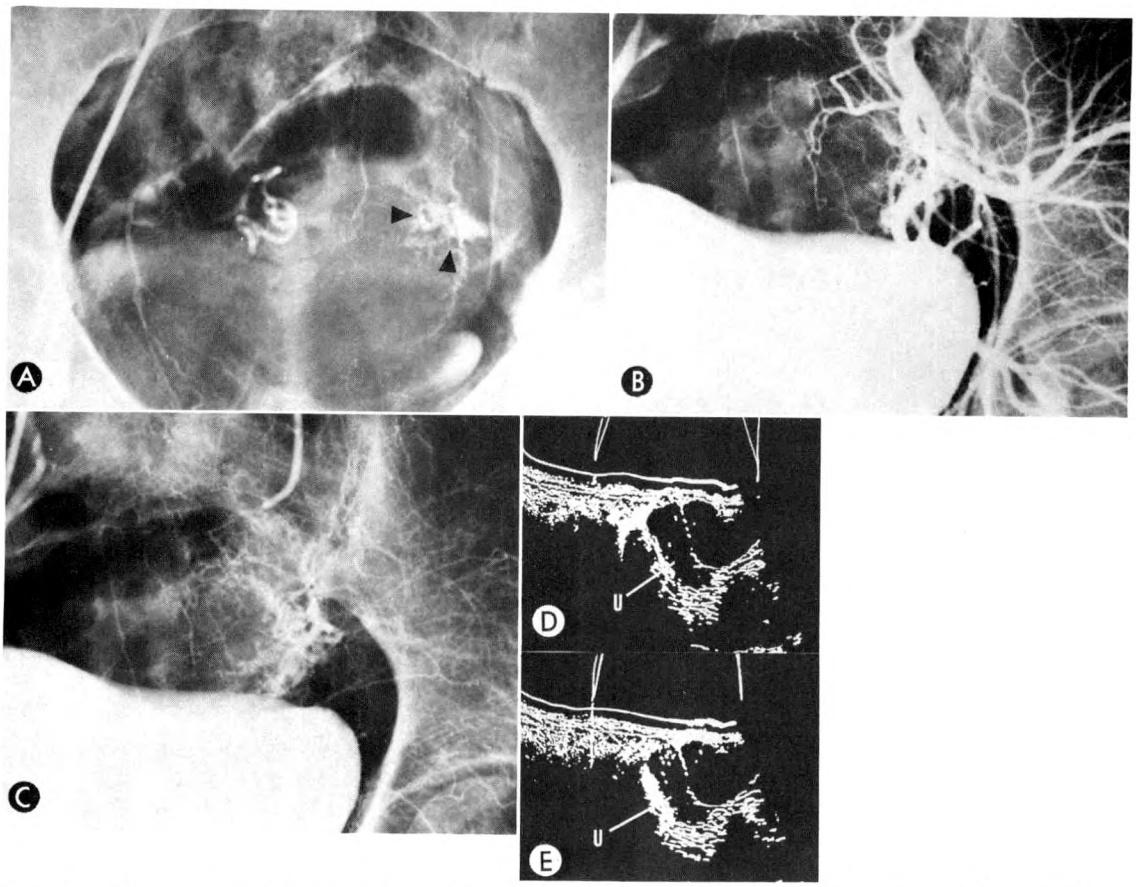

FIG. 2. (*A*) Late phase of a lower abdominal aortogram in a patient with a previous history of hydatidiform mole who had persistent elevation of her urinary HCG titer in spite of chemotherapy. A normal uterine artery is seen on the right but abnormal vascularity and staining appear to be present on the left side (black arrowheads). (*B*) Early and (*C*) late arterial phases of a selective left hypogastric arteriogram. Fine caliber neovascularity and staining are seen within a localized area of the left side of the uterus. (*D*) Midline longitudinal sonography the next day shows an enlarged uterus with several non-specific solitary echoes. (*E*) A longitudinal sonogram 2 cm. to the left of the midline, however, shows a localized cluster of tumor echoes. Hysterectomy revealed a 1.5 cm. nodule of choriocarcinoma within the myometrium on the left side, thereby confirming the arteriographic and sonographic findings.

blastic disease. This can be accomplished quickly and simply by sonography. Second, as Hertz has pointed out,[10] some choriocarcinomas which are deeply imbedded within the myometrium may be resistant to chemotherapy and require hysterectomy. Uterine involvement is not invariably present, however, as evidenced by reported cases in which the uterus was found to be normal even though metastases were relatively widespread.[10,11,13] Sonography thus offers the important advantage of being able to differentiate patients without uterine involvement from those in whom intrauterine lesions are present and who may in rare cases require hysterectomy. Third, sonography is helpful in following patients after treatment. In some instances, the titer level will hover in the borderline range or become slightly elevated after initially having returned to normal. Comparison of follow-up sonograms with the initial post-treatment sonogram may detect or rule out early intrauterine recurrence in these questionable situations. Finally, although radioimmunoassay is probably the most accurate method of detecting HCG levels, a number of other

less reliable methods is still in use at commercial laboratories.[4] Sonography is a simple additional test for the presence of intrauterine disease if there is any question as to the accuracy of the HCG assay method.

The abnormal intrauterine echo pattern seen in choriocarcinoma can be seen occasionally in other conditions. These include degenerating myomata, leiomyosarcomas, endometrial carcinomas, and certain cases of missed abortion and endometritis.[14] However, none of these conditions is associated with elevation of the HCG titer. The occurrence of this echo pattern in association with an elevated titer in a patient with a previous pregnancy of any type can therefore be considered relatively specific for the presence of intrauterine choriocarcinoma.

Because sonography is fast, safe, painless, and easily repeatable, we feel it to be the initial method of choice for attempted visualization of malignant trophoblastic disease. Arteriography can still play a valuable role, however, in cases where the sonographic findings are equivocal. Arteriography also can be very helpful in detecting spread to pelvic organs outside the uterus, such as the parametrium and ovaries. Small lesions in these areas may not be amenable to ultrasonic detection, but are sufficiently hypervascular to be visualized by arteriography.[5,6,20] Its value in detecting metastases to distant organs, such as the kidneys, liver, spleen, gastrointestinal tract or brain, remains undetermined since surprisingly few cases of this sort have been reported. Patrick *et al.*[19] performed angiography on a patient with metastatic choriocarcinoma of the kidney, and Stilp *et al.*[21] briefly described studies of 3 patients with cerebral metastases. All were apparently avascular. As discussed earlier, the marked hypervascularity and staining associated with choriocarcinoma at arteriography may be due primarily to the uterine decidual reaction, with the tumor itself often remaining relatively avascular. Since decidual reactions do not occur in other organs, it is not surprising that metastatic lesions in distant sites can also be avascular. In searching for such lesions, the angiographer must look carefully for space-occupying masses which displace arteries or destroy parenchyma. The absence of abnormal vascularity should not rule out the possibility of trophoblastic metastases, even though the primary lesion has traditionally been considered a hypervascular tumor from the arteriographic viewpoint.

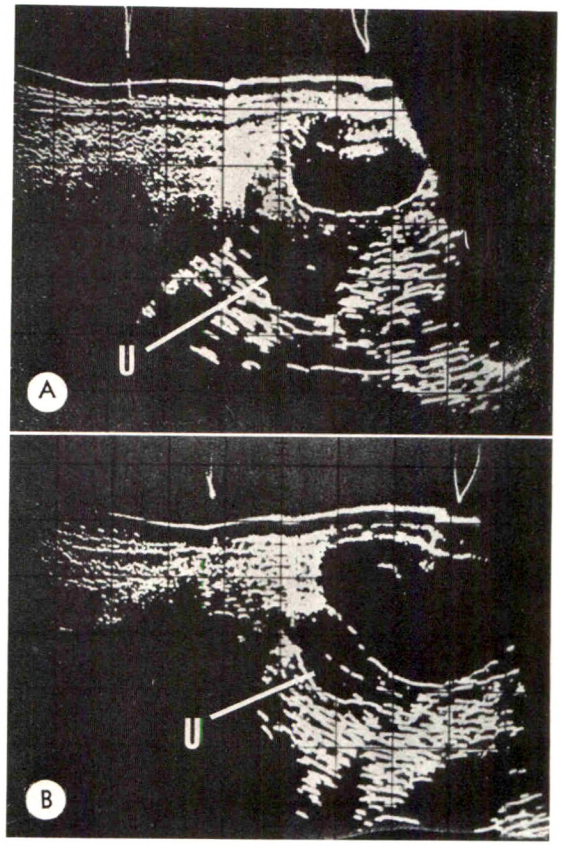

Fig. 3. (*A*) Midline longitudinal sonography in a patient with intrauterine malignant trophoblastic disease. The uterus is enlarged with tumor echoes which appear concentrated anteriorly and in the fundus. (*B*) Ten days later, after a single course of chemotherapy, the uterus appears entirely normal. The centrally-located linear echoes represent the normal endometrial cavity.

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REFERENCES

1. Bagshawe, K. D., Golding, P. R., and Orr, A. H. Choriocarcinoma after hydatidiform mole: studies related to effectiveness of follow-up practice after hydatidiform mole. *Brit. M. J.*, 1969, *3*, 733–737.
2. Borell, U., Fernström, I., Moberger, G., and Ohlson, L. The Diagnosis of Hydatidiform Mole, Malignant Hydatidiform Mole and Choriocarcinoma with Special Reference to the Diagnostic Value of Pelvic Arteriography. Charles C Thomas, Publisher, Springfield, Ill., 1966.
3. Borell, U., Fernström, I., and Westman, A. Value of pelvic arteriography in diagnosis of mole and chorionepithelioma. *Acta radiol.*, 1955, *44*, 378–383.
4. Brewer, J. I., Eckman, T. R., Dolhart, R. E., Torok, E. E., and Webster, A. Gestational trophoblastic disease: comparative study of results of therapy in patients with invasive mole and with choriocarcinoma. *Am. J. Obst. & Gynec.*, 1971, *109*, 335–340.
5. Brewis, R. A. L., and Bagshawe, K. D. Pelvic arteriography in invasive trophoblastic neoplasia. *Brit. J. Radiol.*, 1968, *41*, 481–495.
6. Cockshott, W. P., Evans, K. T., and Hendrickse, J. P. de V. Arteriography of trophoblastic tumours. *Clin. Radiol.*, 1964, *15*, 1–8.
7. Gottesfeld, K. R., Taylor, E. S., Thompson, H. E., and Holmes, J. H. Diagnosis of hydatidiform mole by ultrasound. *Obst. & Gynec.*, 1967, *30*, 163–171.
8. Hellman, L. M., and Pritchard, J. A. Williams Obstetrics. Appleton-Century-Crofts, New York, 1971.
9. Hendrickse, J. P. de V., Cockshott, W. P., Evans, K. T., and Barton, C. J. Pelvic angiography in diagnosis of malignant trophoblastic disease. *New England J. Med.*, 1964, *271*, 859–866.
10. Hertz, R. Biological aspects of gestational neoplasms derived from trophoblast. *Ann. New York Acad. Sc.*, 1971, *172*, 279–287.
11. Hou, P. C., and Pang, S. C. Chorionepithelioma: analytic study of 28 necropsied cases, with special reference to possibility of spontaneous regression. *J. Path. & Bact.*, 1956, *72*, 95–104.
12. Jacobs, E. M., and Johnson, F. D. Gestational choriocarcinoma. *Oncology*, 1972, *26*, 231–237.
13. Kissane, J. Choriocarcinoma. *Am. J. Med.*, 1970, *49*, 395–403.
14. Kobayashi, M., Hellman, L. M., and Cromb, E. Atlas of Ultrasonography in Obstetrics and Gynecology. Appleton-Century-Crofts, New York, 1972.
15. Kohorn, E. I., and Blackwell, R. J. Diagnosis of hydatidiform mole by ultrasonic B-scanning. *J. Obst. & Gynec. Brit. Emp.*, 1968, *75*, 1014–1018.
16. Leopold, G. R. Diagnostic ultrasound in detection of molar pregnancy. *Radiology*, 1971, *98*, 171–176.
17. Lewis, J. L., Jr. Chemotherapy of gestational choriocarcinoma. *Cancer*, 1972, *30*, 1517–1521.
18. Li, M. Chemotherapeutic and immunologic aspects of choriocarcinoma. *J.A.M.A.*, 1972, *222*, 1163–1164.
19. Patrick, C. E., Norton, J. H., and Dasco, M. R. Choriocarcinoma in kidney: case report. *J. Urol.*, 1967, *97*, 444–448.
20. Shimkin, P. M., Van Thiel, D. H., and Ross, G. T. Selective hypogastric arteriography in uterine choriocarcinoma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *111*, 535–540.
21. Stilp, T. J., Bucy, P. C., and Brewer, J. I. Cure of metastatic choriocarcinoma of brain *J.A.M.A.*, 1972, *221*, 276–279.
22. Takahashi, M., and Nagata, I. Angiography of trophoblastic tumors: correlation of pelvic angiography with direct fourfold magnification angiography of uterine specimen. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *112*, 779–787.
23. Wilson, G., Colodny, S., and Weidman, W Comparison of amniography and pelvic angiography in diagnosis of hydatidiform mole. *Radiology*, 1967, *87*, 1076–1079.

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ULTRASOUND IN THE MANAGEMENT OF ELECTIVE ABORTION*

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ABSTRACT:

Diagnostic ultrasound has an important place to play in aiding the obstetrician who has an abortion practice. It is most useful in accurately dating the time of conception. Based on sonographic dating, an abortion may be possible in patients with multiple pregnancy or in whom there is a mass in addition to pregnancy. The performance of a saline or urea abortion is made easier since the precise site of the uterus can be mapped out. Once an abortion has commenced, the presence or absence of retained products can be easily verified by ultrasound.

B-SCAN ultrasound is a well accepted procedure in the management of obstetric patients;[3] several hundred examinations are performed each week in many centers on pregnant women as they progress to term. This modality is also useful in the evaluation of patients scheduled to undergo elective abortion, but heretofore this use has not been described in the literature. The following discussion and case reports demonstrate the clinical applications of sonography in elective abortion and is based on over 1,000 sonograms performed to evaluate patients about to undergo elective abortion at The Johns Hopkins Hospital.

DETERMINATION OF THE DURATION OF PREGNANCY

There are well accepted tables relating the fetal biparietal diameter to gestational age.[1] The most frequent use of ultrasound in relation to elective abortion is in the calculation of the duration of pregnancy. Medical, moral, legal, and emotional factors permit suction curettage and saline injection only within well defined periods of pregnancy. In most institutions curettage is considered unsafe after 12 to 14 weeks; other methods are illegal after 26 weeks. Sonography has been helpful in deciding which technique was appropriate in patients with a borderline gestational age.

REPORT OF CASES

CASE 1. T.F., an obese 25 year old para 1001 presented with amenorrhea of 15 weeks' duration. Palpation was difficult because of the patient's obesity but suggested that the uterine size was consistent with a 12 week pregnancy. A sonogram showed a small uterus containing fetal parts but no gestational sac or fetal head. Measurement of the uterus from the sonogram showed a size consistent with an 11 week gestation according to the tables prepared by Hellman *et al.*[2] No complications followed a suction abortion which delivered a fetus whose size and weight were within the limits for age predicted previously by sonography.

Comment. It is generally accepted that suction abortion should not be performed more than 12 to 14 weeks after conception. Although by dates this pregnancy was beyond this limit, both the sonogram and palpation showed a fetus which was less mature. Since the clinician's opinion was confirmed by the sonogram, a suction abortion was performed. On many similar occasions conflict between clinical findings and the patient's dates has been resolved by sonog-

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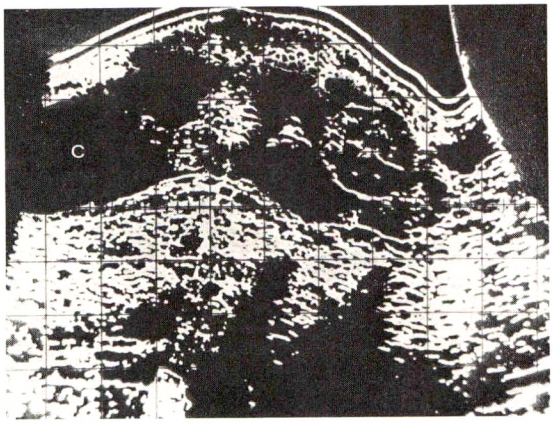

FIG. 1. Longitudinal sonogram of a patient with a single vertex presentation. At the fundus of the uterus is a large cystic lesion (C), later shown to be a benign cystic teratoma (dermoid cyst). The placenta is anterior.*

* All longitudinal scans are presented with patient's head to the left; lower marker designates the symphysis pubis, upper marker the umbilicus.

raphy even at this early stage in pregnancy.

CASE II. B.D., a 22 year old para 0 presented desiring abortion claiming that she was 20 weeks pregnant. On palpation, the uterus was felt to be of a 24 week pregnancy size. However, a sonogram showed a fetal biparietal diameter consistent with a 20 week pregnancy. A saline elective abortion was performed uneventfully and confirmed that the fetus was of a 20 week size.

Comment. It is not the general practice at The Johns Hopkins Hospital to perform abortions at 24 weeks. Had ultrasound facilities been unavailable, an abortion would not have been performed in this instance because palpation indicated a 24 week pregnancy. However, palpation has been shown to be less accurate than ultrasound in the estimation of gestational age,[4] and with the information from the sonogram that a 20 week pregnancy was present, saline elective abortion was performed. On several other occasions palpation has suggested that a pregnancy was more advanced than it was subsequently shown to be by sonography and at delivery; many of these discrepancies occur because of coincident polyhydramnios. For this reason, we now routinely check the duration of pregnancy by ultrasound in patients desiring abortion, whose pregnancies appear to be between 22 and 28 weeks in duration by palpation.

LARGE UTERUS

In most cases where the examiner feels that the pregnancy is further along than dates indicate, sonography supports the physical examination. Patients may bleed early in pregnancy or may report their last menstrual period erroneously, either intentionally or not. However, if there is a discrepancy or any question about the physical examination, sonography allows a more accurate delineation of the duration of the pregnancy and perhaps then an abortion can be performed.

CASE III. Mass plus pregnancy. M.J., a 19 year old para 0 was 16 weeks pregnant by dates but 24 weeks pregnant by examination. Sonography showed a 22 week gestation and a fundal cystic mass 12×15×7 cm. in diameter (Fig. 1). An elective abortion was performed by saline injection, with the passage of a fetus of a size consistent with a 22 week pregnancy. A unilateral dermoid cyst was removed at laparotomy 4 weeks later.

Comment. On 5 occasions we have found ovarian cysts or dermoids in association

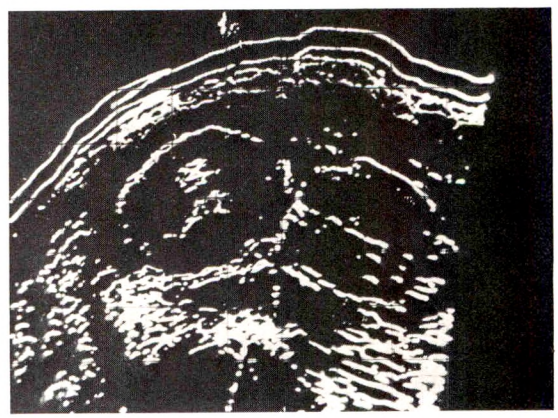

FIG. 2. Twin pregnancies in a girl who presented with a 26 week pregnancy by examination. The biparietal diameter of twins was consistent with a 22 week gestation, and they were successfully aborted.

with pregnancies which have led the examining clinician to believe the palpated uterus was so large that an abortion would not be possible. Following the ultrasonic delineation of the coincident mass, all have had uneventful elective abortions.

Case iv. Twins. U.V., a 13 year old para 0 was 22 weeks pregnant by dates but 26 weeks pregnant by examination. Sonography showed a 22 week twin gestation (Fig. 2), and the patient had an uneventful elective abortion. The size of the twins was consistent with a 22 week duration of pregnancy.

Comment. A confident diagnosis of twins can easily be made by B-scan ultrasound. Although we have seen a number of twins amongst our abortion population, only on 2 occasions has the discovery of twins influenced whether or not an abortion could be performed.

Case v. Myomata. B.R., a 34 year old para 0 was 18 weeks pregnant by dates. The uterine size was consistent with a 24 to 26 week pregnancy thought to be secondary to a combination of pregnancy and a myoma. This diagnosis was confirmed by sonography (Fig. 3), which showed a fetal biparietal diameter of 3.8 cm. (consistent with an 18 week pregnancy) as well as a fundal myoma. An elective abortion was performed showing the fetus to be 18 week size.

Comment. Myomata in association with pregnancy are generally diagnosed clinically by palpation since the uterus has a hard, knobby consistency. A diagnosis of myomata in this patient had been made clinically; however, confirmation by ultrasonography was valuable since the presence of the myomata had made dating by palpation inaccurate.

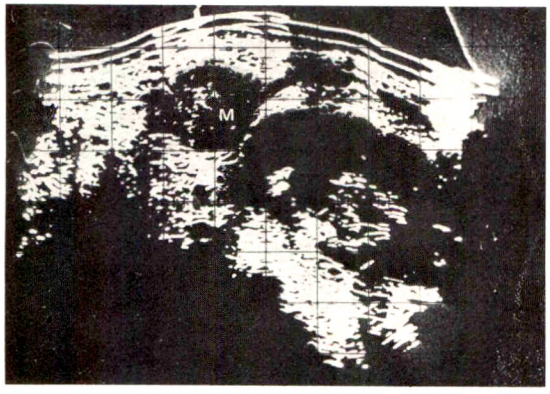

Fig. 3. Patient with an 18 week pregnancy and a myoma (M). Longitudinal sonogram.

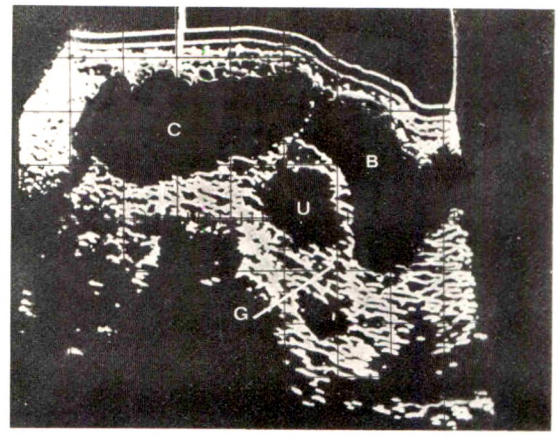

Fig. 4. Longitudinal sonogram. In addition to the uterus (U) which contains a gestational sac (G) and which is seen behind the bladder (B), there is a large cystic lesion (C) into which a saline injection was inadvertently placed.

Midtrimester Abortion

Given the decision to proceed with an elective abortion, certain mechanical or technical problems may interfere with smooth performance of the procedure and increase the chance for morbidity, particularly in the extremely obese patient. In such patients the uterus is hard to feel and delineate and there may be difficulty in obtaining amniotic fluid. Abdominal wall thickness can be determined by ultrasound and the optimal needle site mapped out for the obstetrician. On occasions, the uterus is markedly deviated to the right or left, causing problems with needle placement. The correct site for needle insertion can be suggested by ultrasound. Placental position may interfere with the amniocentesis and produce "bloody taps" if the placenta is anteriorly located. Sonography can localize the placenta and allow a clear exchange of fluid and saline. Similarly, pelvic or parauterine masses may preclude smooth entery of the amniocentesis needle into the

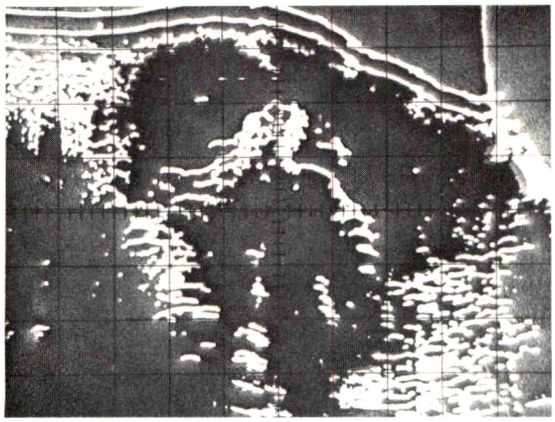

Fig. 5. Longitudinal section of an abdominal pregnancy. The uterine outline is poorly defined, the head is too close to the abdominal wall, and the placenta was never visualized.

uterus either by displacing the uterus or mimicking it in shape and consistency, thus lending themselves to inadvertent puncture with subsequent injection of the hypertonic solution.

Case vi. Injection into extrauterine mass. F.R., a 21 year old para 1001 was 10 weeks pregnant by dates and 18 weeks pregnant by examination. A pregnancy test was positive. A saline injection was done in the normal manner with 200 cc. of clear fluid exchanged for 200 cc. of normal saline. After 48 hours there were no contractions and no response to intravenous pitocin. A sonogram showed a 13×13×7 cm. cystic mass above the uterus into which it is assumed that the saline infusion had been injected (Fig. 4). Uterine measurements were consistent with an 18 week pregnancy. Treatment was by suction curettage and at exploratory laparotomy an intact dermoid cyst was removed.

Comment. Introduction of the saline into the dermoid cyst had no clinical sequelae apart from the clinical embarrassment. This case emphasizes the difficulty the clinician may have in separating a soft mass such as an ovarian cyst from the uterus.

Case vii. Abdominal pregnancy. A.H., a 19 year old para 0 was 19 weeks pregnant by dates and examination. Sonography was interpreted as a normal 20 week intrauterine pregnancy. Two saline injections and intravenous pitocin failed to induce an abortion. At laparotomy a 19 week extrauterine pregnancy was found. A review of the sonogram showed the original error in interpretation.

Comment. In retrospect, it is apparent that the sonogram appearances were abnormal (Fig. 5). The fetal head was located abnormally close to the abdominal wall, there was absence of the usual uterine outline, and it was impossible to make out the placenta. These appearances seem diagnostic of abdominal pregnancy, and we believe that the diagnosis of abdominal pregnancy will be made by ultrasound in the future.

Retained Products

Case viii. B.L., a 21 year old para 0 had a suction abortion performed at 12 weeks. Vaginal bleeding occurred 48 hours following the abortion. A sonogram was therefore performed which showed retained placenta in addition to blood clot (Fig. 6). The patient subsequently had a repeat dilatation and curettage with total removal of the retained placenta.

Comment. After most suction and saline abortions some retained products probably remain in the form of blood clots. Before a diagnosis of retained products is made ultrasonically, a structure such as a hand,

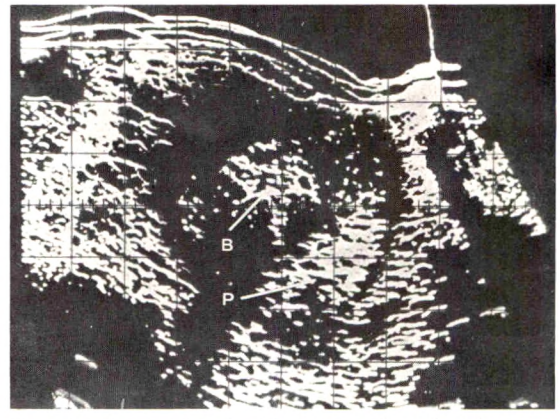

Fig. 6. This patient had had a suction abortion performed 48 hours previously. The longitudinal sonogram shows retained placenta (P) and blood clot (B) within the uterus.

head or placenta should be distinguished from the debris within the uterus. If there is any question of whether retained products are present or not, sequential sonograms over the days following the abortion should be performed since blood clots will disappear while retained products will persist.

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REFERENCES

1. Campbell, S., and Newman, G. B. Growth of fetal biparietal diameter during normal pregnancy. *J. Obst. & Gynaec. Brit. Emp.*, 1971, *78*, 513–519.
2. Hellman, L., Kobayashi, M., Fillisti, L., and Lavenhar, M. Fetal growth and development before 20 weeks gestation. *Am. J. Obst. & Gynec.*, 1967, *103*, 789–798.
3. Kobayashi, M., Hellman, L., and Cromb, E. Atlas of Ultrasonography in Obstetrics and Gynecology. Appleton-Century-Crofts, New York, 1972.
4. Underhill, R. A., Beazley, J. M., and Campbell, S. Comparison of ultrasound, cephalometry, radiology and liquor studies in patients with unknown confinement dates. *Brit. M. J.*, 1971, *3*, 736–738.

RESIDUAL URINE DETERMINATION BY ULTRASONIC SCANNING*

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ABSTRACT:

In 14 patients with an indwelling catheter, 48 different determinations of bladder volume were made using 5 different ultrasonic methods. The simplest procedure, the largest sagittal diameter of the bladder in the median plane, gave only a rough estimate of the bladder volume. The product of bladder depth, height, and width, as determined from transverse and sagittal scans, showed the best correlation to the true bladder volume ($r=0.90$). Computerized reconstruction of multiple serial cross sections proved less accurate than the much simpler depth, height, width product. None of the methods for quantitation of small volumes was good, but qualitative determination of small volumes was accurate.

ALTHOUGH large amounts of residual urine may be detected and roughly quantified by palpation and percussion, the ultimate quantitative measurement of residual urine can only be accomplished by urethral catheterization. Catheterization, however, bears the risk of infection, especially in cases of retention, and may cause traumatic edema and thereby increase a urethral obstruction. Therefore, various methods for an indirect determination of residual urine have been used. A post-voiding bladder film as part of intravenous pyelography will give an estimate of the residual urine.[2] The phenolsulphonphthalein (PSP)[7] and radioisotope[3] techniques depend on normal kidney function but give reasonable estimates of residual urine.

Since the acoustic impedances of fluid and solid tissue are very different, ultrasonic scanning should be well suited to demonstration of the urinary bladder. Holmes[5] and others[1,8] have used simple ultrasonic parameters to estimate bladder volume. The distance between the anterior and posterior walls of the bladder as measured by A-mode (one dimensional) showed a poor correlation to the true bladder volume. A closer correlation was reached when the bladder volume was estimated from two dimensional scanning pictures of the bladder.

It has been shown that serial transverse sections reconstructed by a computer give a good approximation of liver and spleen volumes[6] and might also prove valuable in determining residual urine volume.

We have analyzed this possibility in 14 patients.

MATERIAL AND METHOD

Fourteen patients were studied: 4 had prostatic hypertrophy; 5 had cystoceles; and 5 had neurogenic bladders. All had indwelling catheters. The bladder was filled with sterile saline and emptied in fractions, allowing up to 4 different volume determinations in each patient. Corrections were made for urine production during the examination and for volume of the balloon catheter. A total of 48 different bladder volume determinations was made, 16 in each of the 3 patient categories. The equipment used was a modified Hewlett Packard 7214 A ultrasound apparatus, an Ekoline B-scanner, and a Tektronix 603 storage oscilloscope. A 2.25 MHz transducer, focussed at 10 cm., was used with oil as a coupling medium.

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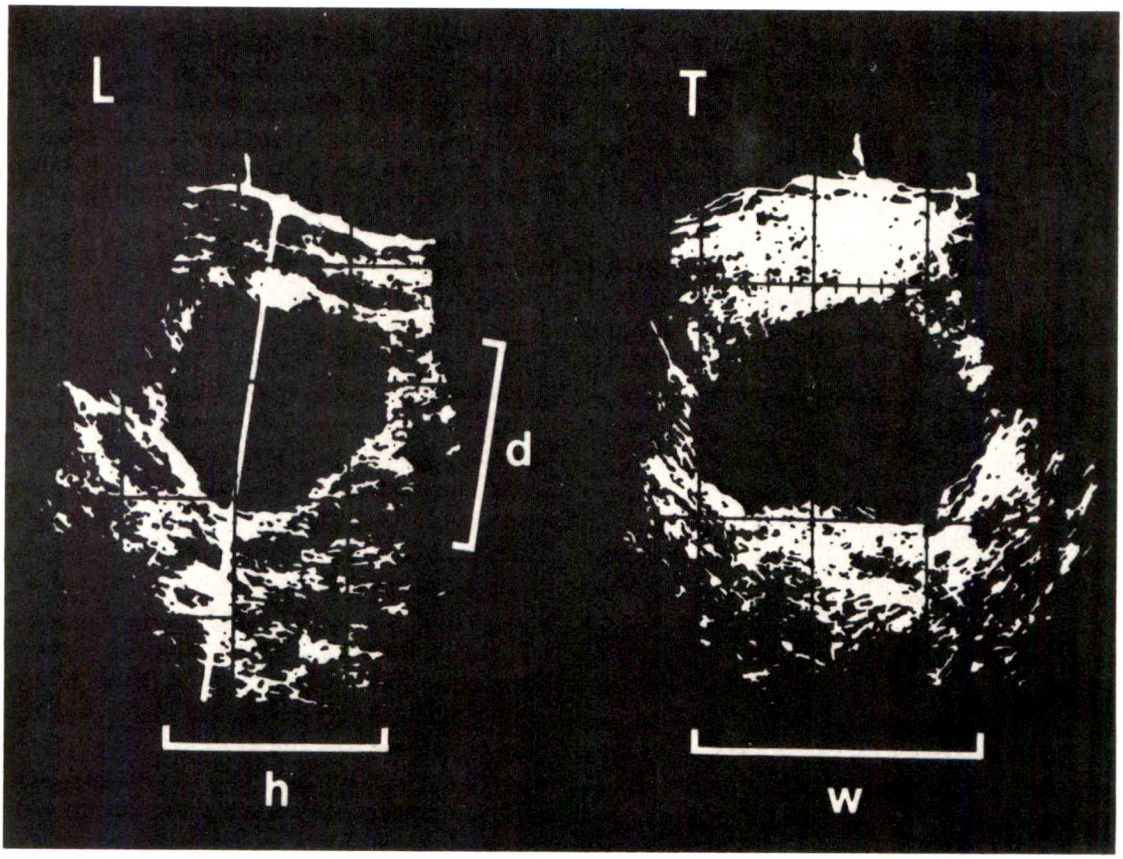

FIG. 1. On the left, a median sagittal section through the bladder. The short line off the skin marks the symphysis. The line traversing the bladder represents the largest bladder dimension obtainable in this plane with the transducer located 1 cm. above the symphysis. The bladder depth and height are indicated by d and h, respectively. On the right, a transverse section of the bladder through the line marked on the sagittal section. The short line off the skin marks the midline. The bladder width is indicated by w.

The following ultrasonic recordings were made: (1) the largest sagittal diameter of the bladder obtainable with the transducer located in the midline 1 cm. above the symphysis; (2) a sagittal scan in the midline; (3) a transverse scan 1 cm. above the pubis; and (4) serial sagittal scans spaced at 1.5 or 1.0 cm. intervals across the total width of the bladder. All scans were photographed on Polaroid prints.

The true bladder volume was plotted against the following ultrasonic parameters: (1) the sagittal diameter; (2) the area of the midline sagittal section of the bladder; (3) the sum of the areas of the sagittal and transverse sections; (4) the product of depth, height, and width as estimated from the sagittal and transverse pictures (Fig. 1); and (5) bladder volume calculated from the serial sagittal sections by means of a pencil follower and a computer using a program for spleen volume determination.[6]

RESULTS

Scattergrams were drawn of the various bladder volume estimates against true volume. These showed that the product of depth, width, and height gives the closest estimate of the true volume, followed by the sum of the sagittal and transverse areas, the midline sagittal area, and the simple sagittal diameter. The same order was found for the correlation coefficients when linear regression analysis was performed for the true values as well as for

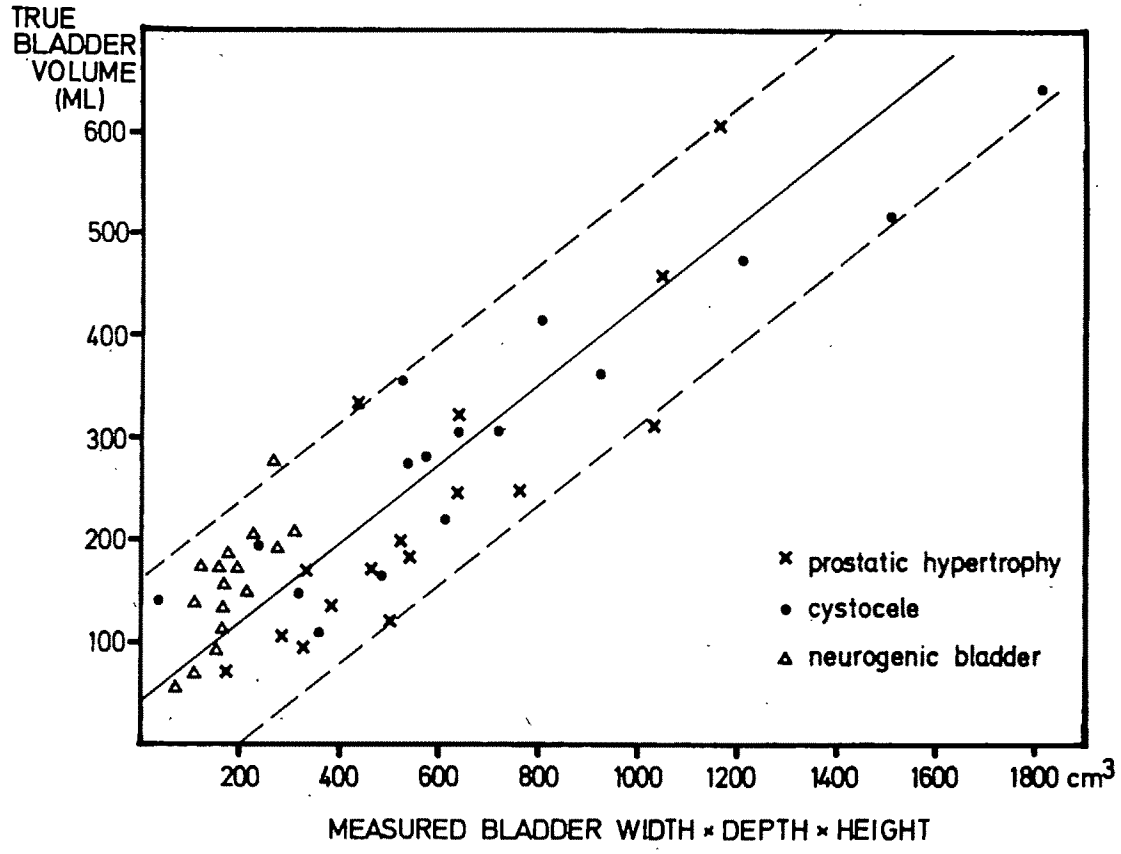

FIG. 2. Scattergram showing the relation between bladder volume estimated from bladder depth, height, and width and the true bladder volume. Also shown are the linear regression line and the 95 per cent confidence intervals.

the logarithms of the values. The volume computed from serial sagittal sections did not correlate so well as the product of depth, height, and width. Since this method required considerable extra effort, it was dropped from further consideration.

Figure 2 shows the scattergram and regression analysis for the product of depth, height, and width. The linear regression equation is: $V = [\text{Depth} \times \text{Width} \times \text{Height}] \times 1.39 + 40 \pm 117$ ml. (95 per cent confidence interval); the correlation coefficient is 0.90, $p < 0.001$ (Fig. 1).

Figure 3 shows the scattergram for the simplest method, the single sagittal diameter. Regression analysis of these data yielded poor correlation.

DISCUSSION

Since the filled bladder is not spherical, it is not surprising that the simple sagittal diameter of the bladder gives only a rough estimate of the bladder volume. This measurement is included because it may be performed with simple ultrasound apparatus (*e.g.*, an echoencephalograph or an echocardiograph). If the procedure is expanded to include more sagittal diameters of the bladder with the transducer pointing proximally and distally, as suggested by West,[8] a somewhat more precise estimate should be obtainable.

Among the volume estimates obtained from scanning pictures, the product of bladder depth, height, and width is superior to the estimates based on planimetry. This is somewhat surprising since planimetry should compensate better for individual variation in bladder shape. This may be caused by improper delineation of

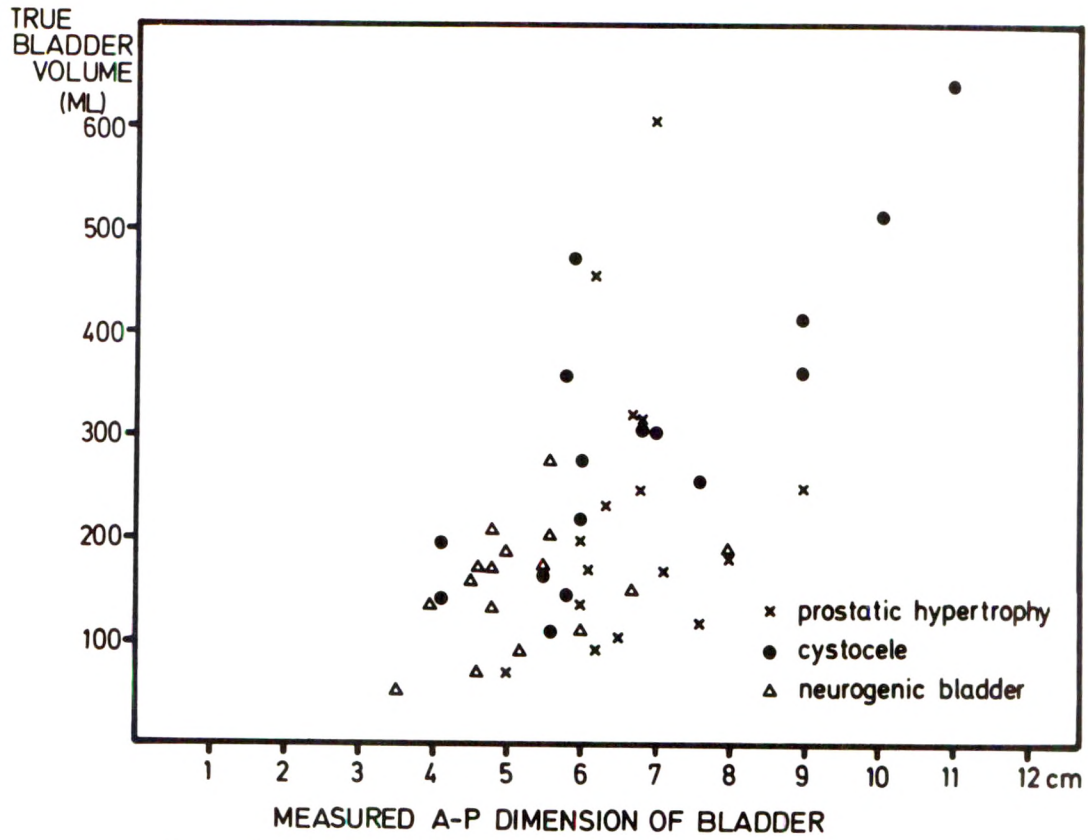

Fig. 3. Scattergram showing the relation between the sagittal diameter of the bladder and the true bladder volume.

parts of the bladder contour while scanning.

It is also interesting that the elaborate technique of volume computation from serial parallel sections is not suited for bladder volume determination. The reason appears to be that sections near the lateral borders of the bladder were difficult to record accurately. This was partly because of the poor orientation of the reflecting surfaces (*i.e.*, far from perpendicular to the sound beam) and partly because of air-filled intestine between the bladder and abdominal wall.

The 3 patient groups of prostatic hypertrophy, cystocele, and neurogenic bladder were chosen to cover different bladder configurations. Unfortunately, the bladder capacity of the patients with neurogenic bladders was below 300 ml., precluding the evaluation of these patients over a wide range of bladder shapes.

It appears from this, as from other studies, that ultrasound is rather poor in quantifying small bladder volumes.[5,8] However, it is possible to determine qualitatively whether residual urine is present.

The scanning pictures upon which these calculations are based rely upon specular reflections. Therefore it is essential that the sound beam strikes the bladder wall at a nearly normal angle. Since this is possible only for limited portions of the bladder, a reduced picture quality results, which probably explains a great deal of the inaccuracy. Advanced gray-scale techniques rely on scattered reflections and may therefore give a better delineation of the bladder and a more precise estimate of its volume.

The amount of energy introduced into the organism at diagnostic ultrasound levels is far below the amount introduced

at therapeutic levels and no harmful effects have been reported.[4]

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REFERENCES

1. Alfthan, O., and Mattson, T. Ultrasonic method of measuring residual urine. *Ann. chir. Synes. Fenn.*, 1969, *58*, 300–301.
2. Bear, E. Visualization of amount of residual urine. *J.A.M.A.*, 1936, *107*, 1886–1888.
3. Claunch, B. C., Barnes, W. T., and O'Hara, V. Determination of volume of residual urine using diodrast I-131. *J. Urol.*, 1961, *85*, 83–86.
4. Hellman, L. M., Duffus, G. M., Donald, I., Brown, T. G., and MacVicker, J. Safety of diagnostic ultrasound in obstetrics. *Lancet*, 1970, *1*, 1133–1134.
5. Holmes, J. H. Ultrasonic studies of bladder. *J. Urol.*, 1967, *97*, 684–691.
6. Rasmussen, S. N., Kristensen, J. K., Holm, H. H., and Kardel, T. Spleen volume determination by ultrasonic scanning. *Scand. J. Haemat.*, 1973, *10*, 298–301.
7. Smith, D. R. Estimation of amount of residual urine by means of phenolsulfonphthalein test. *J. Urol.*, 1960, *83*, 188–191.
8. West, K. A. Sonocystography: method for measuring residual urine. *Scandinav. J. Urol. & Nephrol.*, 1967, *1*, 68–72.

ROENTGENOLOGIC EVALUATION OF A NEW PROSTHETIC SPHINCTER FOR THE TREATMENT OF URINARY INCONTINENCE*

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ABSTRACT:

A new prosthetic device for the treatment of urinary incontinence, caused by a variety of etiologies, has been used 8 times at the University of Michigan Medical Center.

Since the entire device is filled with contrast medium, the radiologist plays a major role in its evaluation.

A brief description of the implantable urinary sphincter and its normal and abnormal roentgenographic appearances are presented and discussed.

Roentgenologic findings which could lead to early clinical detection of mechanical failure are illustrated.

RECENT advances[3] in the treatment of urinary incontinence have led to the development of an implantable prosthetic urinary sphincter§ which enables patients to achieve external voluntary control of bladder function. Early results of others as well as our own experience are encouraging from the standpoints of restoration of urinary continence, and applicability of this device for treating urinary incontinence due to a variety of etiologies.[1,2]

Roentgenologic evaluation of this device is possible because the components of the artificial sphincter are filled with radiopaque contrast medium. Thus, mechanical complications and device malfunction can be recognized roentgenographically.

The purposes of this paper are to describe this device briefly, depict its normal roentgenographic appearance, and illustrate roentgenographic signs which can lead to early clinical detection of mechanical failure. To our knowledge, there are no previous reports on this subject in the radiologic literature.

DESCRIPTION OF DEVICE

The prosthetic sphincter is made up of 4 components (Fig. 1): a reservoir, an inflatable occlusive cuff, which completely encircles the urethra or vesical neck, and two pumps (one, a cuff inflating mechanism and the other, a cuff deflating mechanism). The entire system is fluid-filled with contrast medium (Fig. 2) and comprises a closed system which does not communicate with the urinary tract. Catheter-like connecting tubes, which pass through the inguinal canals, serve as conduits for fluid transfer between the components of the device. The tubing is joined together by angled or straight stainless steel connectors (Fig. 1).

Each pump, consisting of a bulb and 2 valves, permits unidirectional fluid flow to and from the cuff (Fig. 2). By compressing the inflate bulb, fluid flows into the cuff, occluding the urethra or vesical neck, depending on cuff position.‖ When a predetermined pressure within the cuff is reached, no additional fluid can pass into

§ Silicone rubber composition; manufactured by G.U. Prosthetics Division, American Medical Systems, Inc., Golden Valley, Minnesota.

‖ The cuff may be implanted around the vesical neck or proximal bulbous urethra in the male, or around the vesical neck in the female.

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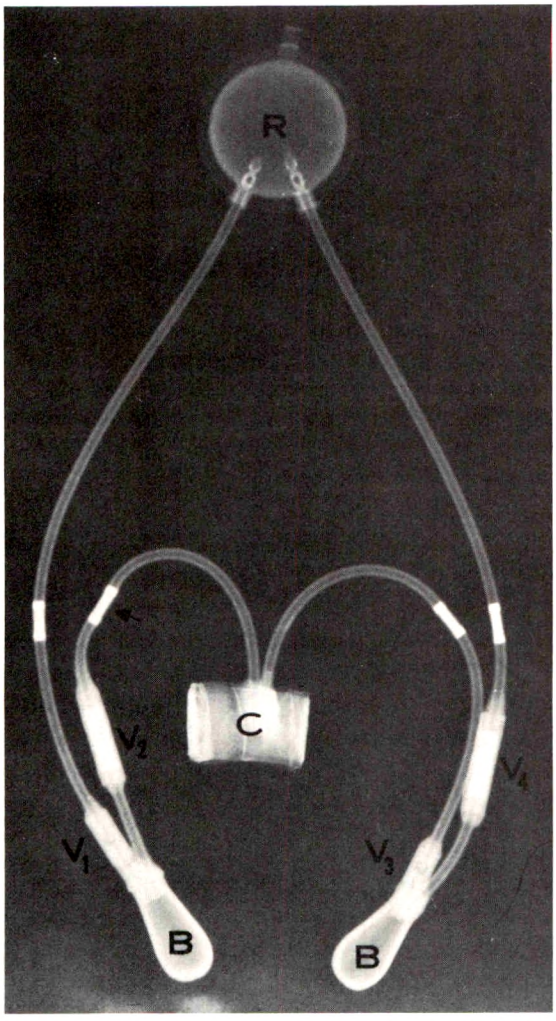

FIG. 1. Plain roentgenogram of the implantable prosthetic urinary sphincter without contrast material.

The 4 major components of the system (reservoir (R), urethral cuff (C) and two bulbs (B)) are joined together by connecting tubes. The connecting tubes are linked to each other by straight stainless steel connectors (arrow). The 4 valves insure unidirectional fluid flow (V_1, V_2, V_3, V_4).

it. Compression of the deflate bulb withdraws fluid from the cuff (thereby deflating the cuff and releasing urethral constriction) back into the reservoir. The bulbs (pumping mechanism) are implanted subcutaneously either in the scrotum or the labia. For standardization purposes, the inflate and deflate bulbs are implanted on the right and left sides, respectively. The reservoir is attached to the posterior rectus muscle sheath. The surgical procedure for implantation of this device has been described by Scott.[1]

MATERIAL AND METHOD

At the University of Michigan Medical Center, urinary incontinence has been treated by one of us (ACD) utilizing this implantable prosthetic urinary sphincter in 8 patients (6 males and 2 females), from 12 to 78 years of age. Etiologies of incontinence treated with this device include postsurgical transurethral prostate resection (4 patients), trauma (2 patients) and neurologic causes (2 patients).

A plain roentgenogram of the pelvis to include the entire device was obtained within 1 week of surgical implantation on all 8 patients. Follow-up roentgenograms were

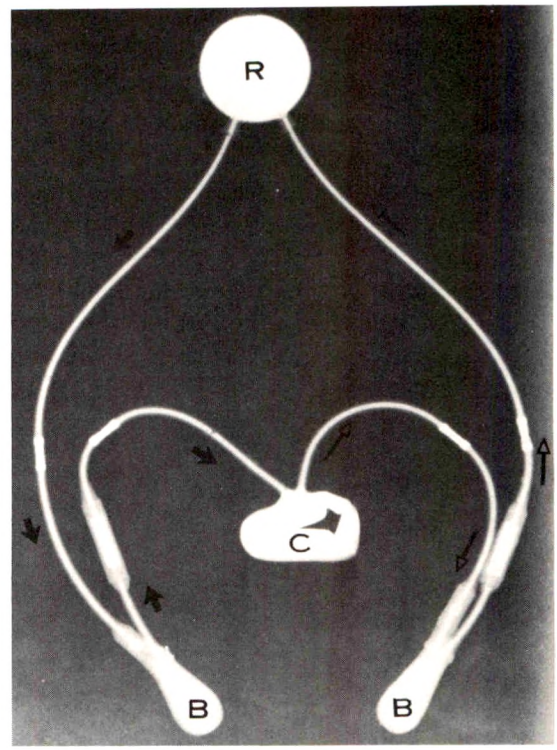

FIG. 2. Plain roentgenogram of the prosthetic sphincter with contrast material.

The entire system is filled with contrast medium, and the route of unidirectional fluid flow to (short solid arrows) and from (long open arrows) the cuff is shown. Compression of the inflate bulb has resulted in the flow of contrast into the urethral cuff. R = reservoir. C = cuff. B = bulb.

obtained only when problems were encountered clinically. All of the roentgenograms were analyzed using the following criteria:

1. Location and position of the reservoir, bulbs, and cuff
2. Appearance of the cuff (inflated or deflated) and bulbs (full or compressed((Fig. 3)
3. Location and course of connecting tubes (gently curved, angulated or kinked)
4. Presence of air within the system
5. Evidence of contrast material extravasation outside the system.

RESULTS

I. NORMAL ROENTGENOGRAPHIC APPEARANCE

The contrast filled reservoir has a round configuration and projects over the sacrum near the midline (Fig. 3). It is seen just above the urinary bladder if the latter contains contrast material opacified urine (Fig. 4). Indentations in the contour of the reservoir may be seen normally (Fig. 5). The occlusive cuff has a variable shape and is located near the midline beneath or projecting over the pubic symphysis, depending on the site of surgical implantation (Fig. 4; and 6). The relationship of the cuff to the soft tissues of the penis indicates its anatomic location either around the vesical neck or proximal bulbous urethra (Fig. 4). The 2 bulbs are identified within the scrotum or labia and should be at approximately the same level, in relation to each other (Fig. 5).

The connecting tubes should emerge from and enter the cuff symmetrically and describe a gently curved course to and from

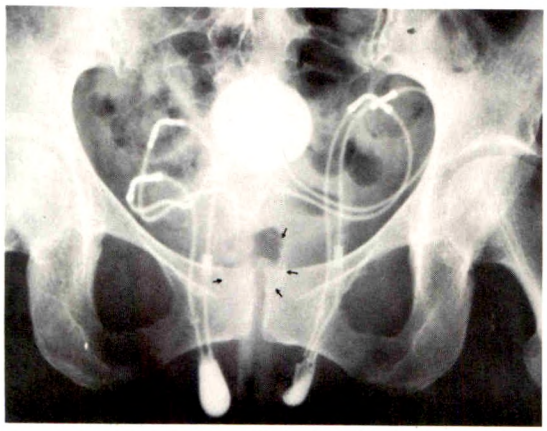

Fig. 3. Deflated cuff (nonocclusive configuration) around the vesical neck.

Minimal residual contrast is faintly seen in the cuff (small arrows) following compression of the deflate bulb (large arrows). The cuff projects over the pubic symphysis, indicating its anatomic location around the vesical neck. The perfectly round configuration of the reservoir is normal. The slightly flattened (compressed) configuration of the deflate bulb within the left labium may be seen normally when the cuff is empty. Note the presence of air within the connecting tubes and deflate bulb.

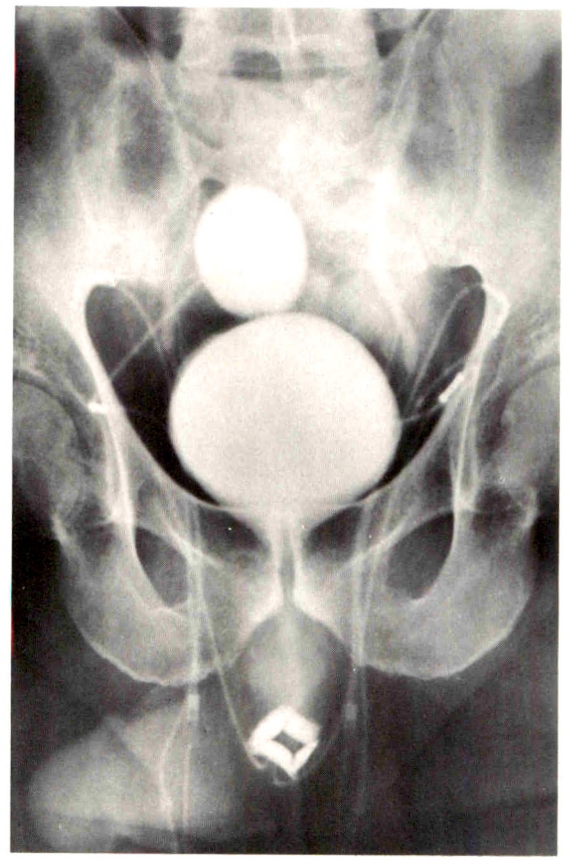

Fig. 4. Normal relationship of the sphincter to the urinary tract during excretory urography.

The round reservoir is seen just above the urinary bladder and between the ureters. The relationship of the contrast filled cuff to the soft tissues of the penis, and its position beneath pubic symphysis, indicates its location around the proximal bulbous urethra.

Fig. 5. Normal roentgenographic appearance of sphincter with urethral cuff inflated.

The entire sphincter is filled with contrast material. The bulbs have an ovoid configuration and they are at approximately the same level within the scrotum. Note the symmetry of the connecting tubes entering and leaving the inflated urethral cuff. The triangular lucency in the center of the cuff demarcates the position of the bulbous urethra. The indentation in the contour of the reservoir superiorly is normal. A healed right inferior pubic ramus fracture is present.

the reservoir without acute angulation (Fig. 5). The tubing should be continuous without defects or points of disconnection. The connecting tubes are joined together by angled or straight roentgenographically opaque connectors.

The entire system should be outlined by contrast material and no air should be present within the system. No contrast material should be seen outside the system (Fig. 5; and 8*B*).

II. ABNORMAL ROENTGENOGRAPHIC APPEARANCE

A. *Plain Films*

1. Asymmetric Bulb Position

In our only pediatric case, the position of the deflate bulb in the left labium was higher than the inflate bulb in the right labium (Fig. 6).

Comment. Although this made it more difficult for the patient to grasp and compress the deflate bulb than the inflate bulb, the device functioned properly.

2. Twisted Urethral Cuff

A markedly tortuous and abnormal course of the connecting tube exiting from the urethral cuff was seen in another case. Slight deviation of urethral cuff position, off the midline, was also present (Fig. 7).

Comment. At operation, torquing of the urethral cuff with pressure erosion into the urethra was confirmed.

3. Kinked Connecting Tubes

In a third case, abrupt angulations in the connecting tubes entering into and exiting from the inflate bulb were obvious roentgenographically (Fig. 8*A*).

Comment. The kinks were considered the cause of the patient's inability to inflate the occlusive cuff, and resulted in recurrence of urinary incontinence. Surgical correction of the kink rendered the patient continent (Fig. 8*B*).

4. Presence of Air within the System

The presence of air in the contrast-filled system was seen in three cases. Well de-

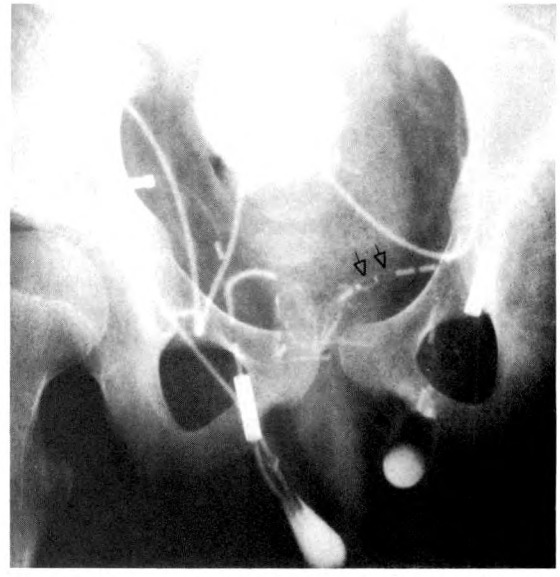

Fig. 6. Asymmetric bulb position and air within the system.

The deflate bulb in the left labium is higher than the inflate bulb in the right labium. Lucent filling defects in the contrast-filled connecting tubes (open arrows) and deflate bulb (solid arrow) represent air within the system.

fined filling defects in the contrast-filled connecting tubes are diagnostic on the plain roentgenogram (Fig. 6).

Comment. Decubitus or upright views are mandatory to detect air in the reservoir, which may be the only site of air within the entire system (Fig. 9).

B. *Retrograde Urethrogram*

1. Urethral Extravasation

Extravasation of contrast material from the urethral lumen was seen surrounding the urethral cuff in 1 case (Fig. 10).

Comment. At operation, a periurethral abscess which eroded into the urethra was found. No signs of abscess within the soft tissues were seen on the plain roentgenogram.

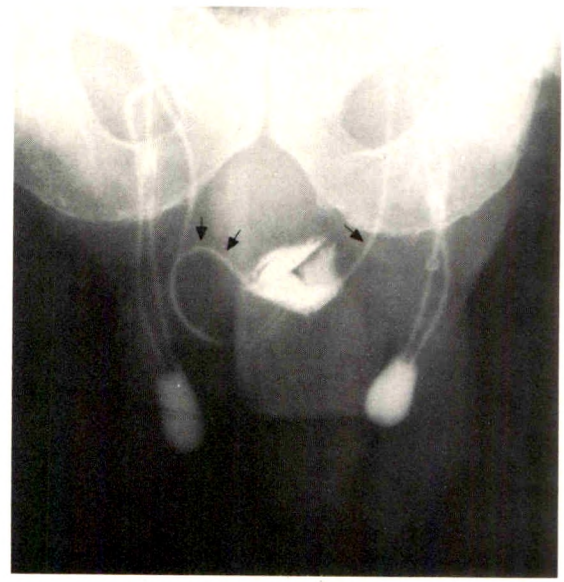

Fig. 7. Twisted urethral cuff.

The position of the urethral cuff is to the left or midline. The markedly tortuous and redundant course of the connecting tube leaving the urethral cuff is abnormal (arrows).

DISCUSSION

The radiologist plays a major role in evaluating the mechanical and functional integrity of the implantable prosthetic urinary sphincter. Increasing use of this device makes familiarity with the normal and abnormal postoperative roentgenographic appearance essential.

Several types of roentgenologic examinations may be used to evaluate the artificial sphincter. Plain roentgenograms are sufficient for identification of the position and location of the various components which

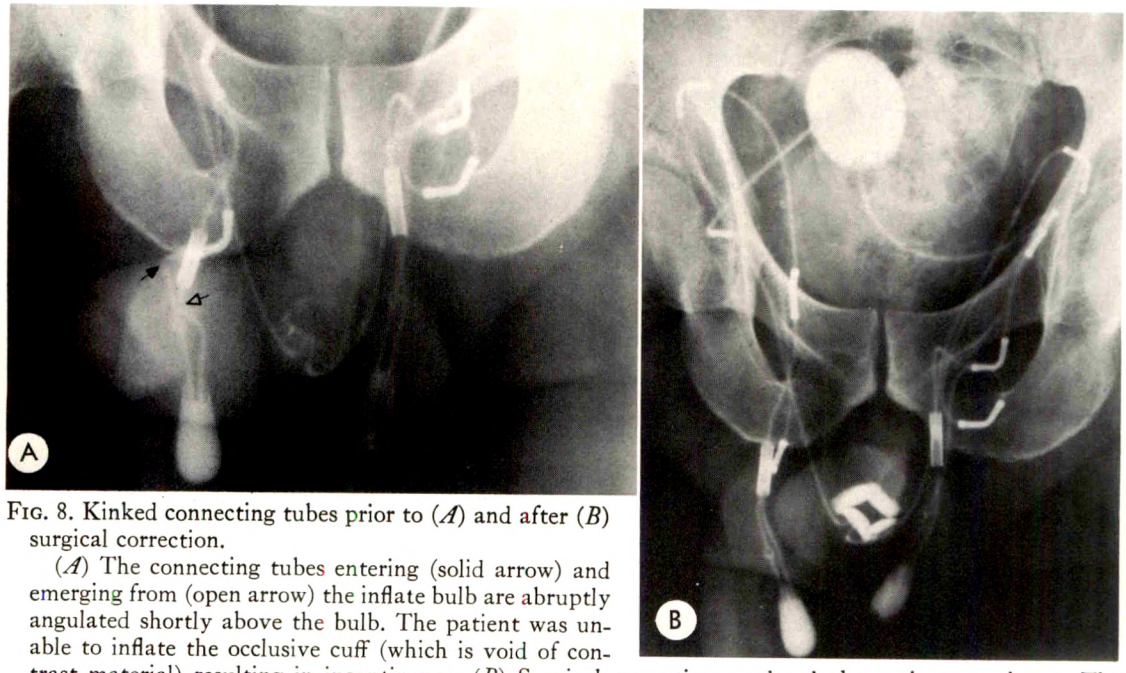

Fig. 8. Kinked connecting tubes prior to (*A*) and after (*B*) surgical correction.

(*A*) The connecting tubes entering (solid arrow) and emerging from (open arrow) the inflate bulb are abruptly angulated shortly above the bulb. The patient was unable to inflate the occlusive cuff (which is void of contrast material) resulting in incontinence. (*B*) Surgical correction rendered the patient continent. The kinks are no longer present. Note that one angled connector has been replaced by a straight one.

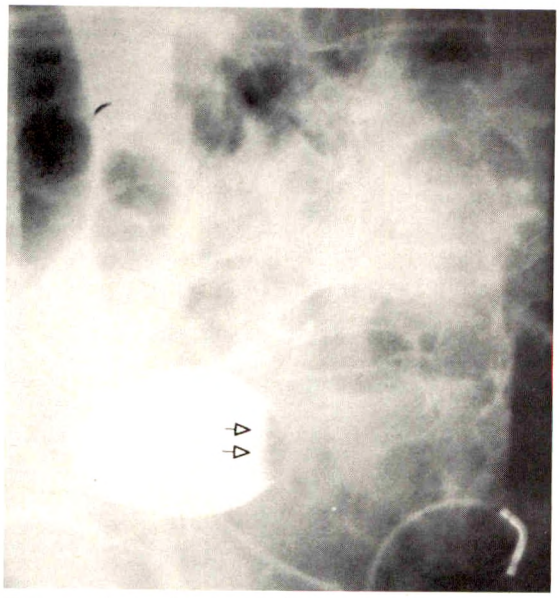

FIG. 9. Air in the reservoir. Right decubitus view.
A cone down view of the reservoir shows an air-fluid level (arrows) within it indicating the presence of air. Upright views may be used as an alternative to decubitus views to demonstrate air in the reservoir.

comprise the device. Identification of most mechanical complications accounting for device malfunction can also be established from plain roentgenograms. Sphincter function may be monitored roentgenographically by fluoroscopy or fluorocystography. The latter is useful for roentgenographic documentation of bladder leakage (incontinence) with straining or coughing in the upright position, and may be performed after excretory urography.[1] Fluoroscopy allows direct visualization of change in cuff configuration, as the patient squeezes the inflation or deflation bulb.

The significance and type of complications which we encountered were variable. The presence of air in the system was the most frequent immediate postoperative roentgenographic finding and is almost unavoidable despite careful surgical technique. Although its significance is not known, we did not consider mechanical failure in any of our cases to be the result of air within the system. The presence of air at a strategic point could theoretically produce an "air block" within the system and impede fluid transfer.

The complications of twisted urethral cuff, periurethral abscess and kinked connecting tubes were more serious and required surgical intervention. All 3 were demonstrable roentgenographically. Not all complications, however, can be detected roentgenologically. Valve failure cannot be perceived roentgenographically and is, therefore, a clinical diagnosis by exclusion. Two patients had valve failure in our series. In such cases, the radiologist's role is to exclude other causes for device malfunction.

Other potential complications which lend themselves to roentgenographic detection include reservoir or bulb leakage, connecting tube disconnections and tube breakage.

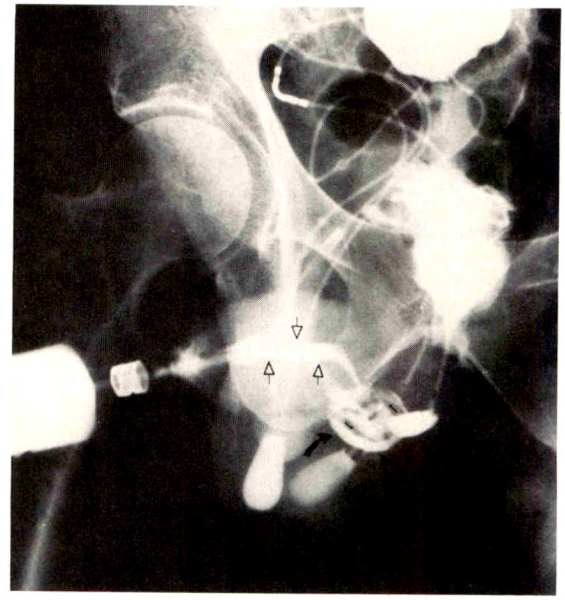

FIG. 10. Urethral extravasation. Retrograde urethrogram.
Extravasation of contrast material from the urethral lumen is present. The outer circular collection of contrast material represents leakage around the urethral cuff (curved arrows). Since there is no contrast within the cuff itself, it appears radiolucent (tiny arrows). The inner circular collection of contrast material represents leakage along the inner aspect of the urethral cuff, and the lucency within it represents a nonopacified segment of the urethra. Some contrast material has refluxed into the prostatic fossa and bladder, and some is seen in the anterior urethra (open arrows).

We have not encountered any of these.

In summary, most complications which occur can be diagnosed or confirmed roentgenographically. The radiologist's role is therefore crucial in evaluating patients treated with this device, since early recognition of mechanical complications may prevent irreversible damage.

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REFERENCES

1. Scott, F. B., Bradley, W. E., and Timm, G. W. Treatment of urinary incontinence by implantable prosthetic sphincter. *Urology*, 1973, *1*, 252–259.
2. Scott, F. B., Bradley, W. E., and Timm, G. W. Treatment of urinary incontinence by implantable prosthetic urinary sphincter. *J. Urol.*, 1974, *112*, 75–80.
3. Timm, G. W., Bradley, W. E., and Scott, F. B. Experimental evaluation of implantable externally controllable urinary sphincter. *Invest. Urol.*, 1974, *11*, 326–330.

XEROGRAPHIC EVALUATION OF RADICAL VULVECTOMY SPECIMENS*

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ABSTRACT:

Xerography of the radical vulvectomy specimen provides a permanent and visible record of the major lymph node distribution within the specimen. Such documentation is valuable to both pathologist and surgeon alike when making decisions regarding the necessity of further therapy.

The combined effort represented by this "team" approach results in a high degree of accuracy not available with other techniques. Only if this kind of lymph node assessment is applied to each and every case will it be possible to evaluate accurately the logic and efficacy of the new therapeutic concepts regarding invasive carcinoma of the vulva.

THE therapeutic approach to invasive carcinoma of the vulva has recently undergone modification in light of the data presented by Green *et al.*,[1] Lundwall,[2] Rutledge *et al.*[3] and others. The classic treatment, radical vulvectomy and bilateral groin dissection with simultaneous pelvic lymphadenectomy for all cases regardless of stage, has given way to the more logical limitation of the procedure at the inguinal ligament, particularly in Stage I and II lesions. This change in operative philosophy stems from the fact that when the inguinal and femoral lymph nodes are negative, so also are the deep pelvic lymph nodes, making their routine surgical removal unnecessary. Deep pelvic lymph node dissection increases the postoperative morbidity and adds time to an already lengthy procedure.

Way,[5] who pioneered the combined operation, has since modified his views stating that in the cases with positive deep pelvic lymph nodes and negative groin lymph nodes, errors have been made in the histologic assessment of the groin lymph node status.[4] Such errors are probably common, and occur either because the pathologist fails to locate every lymph node present in the specimen or because he sections each lymph node in a suboptimal manner.

If we are henceforth to follow the concept of treating cases of Stage I and II invasive carcinoma of the vulva with primary radical vulvectomy and groin dissection, reserving pelvic lymphadenectomy for those cases where evidence of lymph node metastasis is found in the excised groin lymph nodes, then we must make every effort to decrease the false negativity rate of lymph node status assessment.

To achieve complete evaluation of the lymph nodes within the radical vulvectomy specimen, the pathologist must be able to first identify all the lymph nodes within the specimen, and second, must sample the lymph nodes in an optimal manner.

As suggested by Way,[5] we have employed xerography in evaluation of the radical vulvectomy specimen in an effort to increase the accuracy of lymph node identification.

METHOD

The tissue is brought directly from the operating room in the fresh state (Fig. 1) and multiple xerograms are performed of the entire vulvectomy specimen. Lymph

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nodes are identified by the radiologist and marked on the xerogram in the presence of the pathologist. Care must be taken to maintain the orientation of the specimen. Following this, the pathologist may choose to dissect the lymph nodes immediately or first to fix the specimen in formalin, as it was oriented on the xerogram, and dissect the lymph nodes from the fixed specimen. Using the xerogram as a guide, the lymph nodes are labelled as to their location as they are dissected from the surgical specimen. This initial xerogram will localize areas of lymph node aggregations within the vulvectomy specimen and can delineate isolated discrete lymph nodes (Fig. 2). As such, it provides the pathologist with a "road map" of the surgical specimen, pinpointing the location of the lymph nodes present. After completing his dissection, a second xerogram may indicate any lymph nodes inadvertently left behind.

Once a lymph node is identified and removed from the specimen, there remains the difficulty of identifying metastatic tumor within the lymph node.

The accuracy of histologic identification of a lymph node metastasis is dependent upon the size of the lymph node, size of the metastasis, and method of lymph node sectioning. Of these variables, only the method of lymph node sectioning can be controlled. Therefore, it is imperative that lymph nodes suspected of containing malignancy be properly sectioned. Figure 3 demonstrates the per cent probability of identifying a 2 millimeter lesion, (D_L), in variable sized lymph nodes using two different methods of sectioning.[6] If the lymph node is bisected (lower curve) and both cut surfaces sectioned, the per cent probability of finding a 2 millimeter lesion in a 10 millimeter lymph node is 35 per cent. Sectioning the same lymph node in quarters (upper curve) and sectioning alternate cut surfaces, the per cent probability of identifying the lesion increases to 80 per cent. Sectioning the lymph node into five 2 millimeter slices would further increase the probability of success. It is apparent that proper lymph node sectioning will improve our ability to identify lesions within lymph nodes.

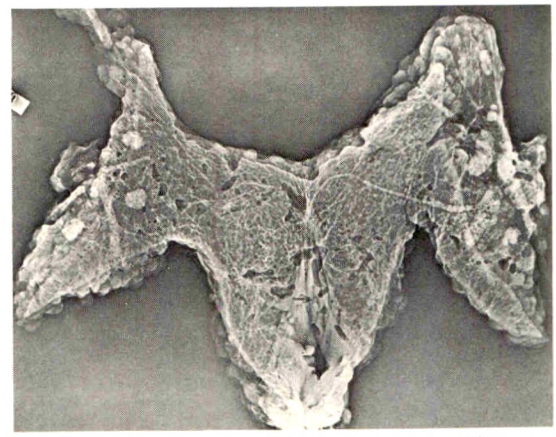

Fig. 2. Xerogram (negative mode) of similar specimen.

Fig. 1. Radical vulvectomy specimen in fresh state.

DISCUSSION

The xerogram provides an excellent record of the distribution of the lymph nodes within the specimen and specific lymph node locations can be documented. After the histological evaluation, positive lymph nodes can be marked on the xerogram thus achieving an accurate, visible, and permanent record of the orientation of positive lymph nodes within the surgical specimen.

Formalin fixation, prior to xerography, has the advantage of maintaining tissue orientation better than in the fresh speci-

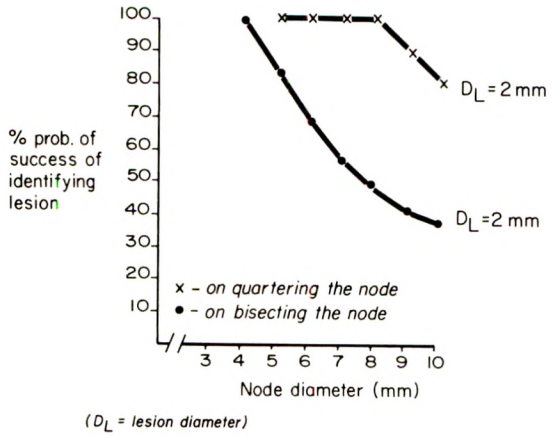

Fig. 3. Per cent probability of identifying a 2 millimeter lesion (D_L) in variable sized lymph nodes using two different methods of sectioning.

men. We have found that xerography is satisfactory on the formalin fixed vulvectomy specimen. Tissue fixed in Zenker's solution, however, does not lend itself well to xerography. Multiple small metallic densities are scattered over the specimen probably as a result of mercurial deposition from the fixative.

Very small lymph nodes, and those superimposed on one another, are difficult to identify with xerography. Occasionally, hematomas, vessels, and adipose tissue, particularly at the edges of the specimen, may be falsely interpreted as lymph nodes.

Xerography offers greater latitude than conventional film and, due to its "edge enhancement" effect, the borders of lymph nodes are more clearly seen.[7]

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REFERENCES

1. Green, T. H., Jr., Ulfelder, H., and Meigs, J. V. Epidermoid carcinoma of vulva. *Am. J. Obst. & Gynec.*, 1958, *75*, 848–864.
2. Lundwall, F. Therapeutic approach to carcinoma of vulva. *Proc. Internat. Soc. Study Vulvar Dis.*, 1973, *1*, 22–23.
3. Rutledge, F., Smith, J. P., and Franklin, E. W. Carcinoma of vulva. *Am. J. Obst. & Gynec.*, 1970, *106*, 1117–1130.
4. Way, S. Anatomic pathology of vulvar cancer. In: Aspects and Treatment of Vulvar Cancer. First International Symposium, Madrid, 1971. S. Karger AG, 1972, pp. 16–20.
5. Way, S. Personal communication.
6. Wilkinson, E. J., and Haase, L. Probability in lymph node sectioning. *Cancer*, 1974, *33*, 1269–1274.
7. Wolfe, J. N. Xeroradiography of the Breast. Charles C Thomas, Publisher, Springfield, Ill., 1973.

Vol. 125, No. 2

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Published by: Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Illinois 62717.

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Editor Emeritus: T. Leucutia, 401 Professional Building, Detroit, Mich. 48201; *Acting Editor:* E. Frederick Lang, Detroit, Mich.

Representative on the Board of Chancellors and Council of the ACR: W. B. Seaman.

Seventy-sixth Annual Meeting: Marriott Hotel, Atlanta, Ga., Sept. 30–Oct. 3, 1975.

AMERICAN RADIUM SOCIETY

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Liaison Representative on the National Council on Radiation Protection and Measurements: Lawrence Davis, Philadelphia, Pa.

Representative on the Board of Chancellors of the American College of Radiology: Luther W. Brady, Philadelphia, Pa.

Representative to the Council of the American College of Radiology: Juan A. del Regato, Tampa, Fla.

The Fifty-eighth Annual Meeting: Bayshore Inn, Vancouver, British Columbia, Canada, May 9–14, 1976.

EDITORIAL

OPERATIVE CHOLANGIOGRAPHY: THE OBLIGATION OF THE RADIOLOGIST TO THE SURGEON AND HIS PATIENT

THE technique of nonoperative removal of retained biliary tract stones has become an accepted and useful procedure.[4] Those publishing their techniques and results document the unfortunate frequency of retained biliary tract stones following cholecystectomy.[3,5,9] There is possibly an admission here of neglect and a default in the obligation to the patient undergoing cholecystectomy by not providing reasonable opportunity that common duct stones, if present, will be detected and removed at the time of the initial surgery. Residual common duct stones following cholecystectomy should be a rare, not a common occurrence. Operative cholangiography is a simple roentgenographic procedure, which should be incorporated as a routine study in all cholecystectomies, with or without suspicion of common duct stones. Many surgeons remain skeptical of its value. This skepticism can be enhanced by a disinterested radiology service unwilling to spend the time to provide precise technical assistance and demonstrate how helpful this procedure can be. The cholangiogram adds no additional risk to the patient and does not lengthen the surgical procedure significantly. Over-all morbidity and cost of patient care are reduced. Too often, surgeons who have requested operative cholangiography have discarded its use after inadequate technique and poor quality films have yielded unsatisfactory results. Twenty years of routine use of operative cholangiography in over 3,000 procedures have provided a valid basis for the following comments.

Time and again surgical intuition has been proved fallacious in the detection of stones in the common duct, either before or following choledochotomy. A surgical resident once observed that the few minutes spent in x-raying the duct are more convincing than the longer time it takes to debate whether or not choledochotomy should be performed. Even when stones in an obviously dilated common duct are strongly suspected or even palpable, an initial cholangiogram through cannulating the cystic duct provides the surgeon with an exact concept of the problem he faces: Are stones present, how many, are they impacted, is there an unusual configuration of the biliary system, are there anomalies, is there partial or complete obstruction, does the common duct enter the duodenum *via* a duodenal diverticulum? The completion cholangiogram following choledochotomy and stone extraction will indicate the presence of residual stones, obviating a second surgical procedure or added morbidity and expense of percutaneous removal at some subsequent time.

Approximately 6 per cent of patients having routine cystic duct cholangiograms show common duct stones without clinical suspicion, and, according to ordinary criteria, these would have escaped detection without a cholangiogram.[6]

In many instances, a strong clinical suspicion compels the surgeon to explore the common duct. Any surgical interference with the common duct and the sphincter of Oddi increases the morbidity, adds approximately one hospital day to the patient's stay, and provides a possible source

of biliary stricture. Therefore, of equal importance, operative cholangiography can exclude unnecessary common duct explorations by revealing normal ducts and the absence of stones despite clinical evidence to the contrary. When all age groups are included, common duct stones probably occur in about 10 per cent of patients with cholelithiasis. Clearly then, it is imperative that no effort be spared to render the surgical management of cholelithiasis definitive.

Operative cholangiography can be simple and achieved successfully only after repeated use of a well coordinated technique and a sincerely concerned team effort by the surgeon, the anesthesiologist and the radiologist. A brief period of time is spent performing the procedure. The time awaiting its interpretation by the radiologist is utilized by the surgeon for removal of the gallbladder and reperitonealization of the gallbladder bed. Equipment need not be complicated nor expensive. Currently available mobile x-ray generators provide superior quality films when competently utilized. Explosion-proof equipment which is cumbersome need not be used where non-explosive anesthesia is employed. Image amplification and remote monitoring are not requirements. There are many technical hints which the inexperienced or sometime cholangiographer should review and are available in other publications.[1,2,7,8,10,11]

The day of the uninformed patient is rapidly disappearing. If he is faced with a second surgical procedure which can be more hazardous than his initial operation, he will justifiably ask questions. With increasing attention to professional standard review and internal audit, sloppy technique by the surgical—radiologic team will be subject to more criticism. The surgeon who manifests displeasure and poor experience with operative cholangiography has not had the good fortune to have an interested radiology department or has been unwilling himself to devote efforts toward improving his surgical results.

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REFERENCES

1. Baker, J. W., and Walker, J. H. Cholestectomy and operative cholangiography. *A.M.A. Arch. Surg.*, 1959, *79*, 853.
2. Baker, J. W., Walker, J. H., and Lawrence, G. H. Operative cholangiography: Current Surg. Management II, 1960.
3. Bean, W. J., Smith, S. L., and Calonje, M. A. Percutaneous removal of residual biliary tract stones. *Radiology*, 1974, *113*, 1–9.
4. Burhenne, H. J. Nonoperative retained biliary tract stone extraction: new roentgenologic technique. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 388–399.
5. Glenn, F. Postcholecystectomy choledocholithiasis. *Surg., Gynec. & Obst.*, 1972, *134*, 249–252.
6. Jolly, P. C., Baker, J. W., Schmidt, H. M., Walker, J. H., and Holm, J. C. Operative cholangiography: case for its routine use. *Ann. Surg.*, 1968, *168*, 551.
7. Jolly, P. C., Baker, J. W., Walker, J. H., and Schmidt, H. M. Operative cholangiography. *Northwest Med.*, 1969, 639–643.
8. Jolly, P. C., and Walker, J. H. Operative cholangiography in biliary tract surgery. *Hosp. Practice*, 1972, *7*, 92.
9. Larson, R. E., Hodgson, J. R., and Priestley, J. T. Early and long term results of 500 consecutive explorations of common duct. *Surg., Gynec. & Obst.*, 1966, *122*, 744–750.
10. Lawrence, G. H., and Walker, J. H. Operative cholangiography—reappraisal. *Bull Mason Clinic*, 1961, *15*, 2.
11. Walker, J. H., and Baker, J. W. Technical hints towards improved operative cholangiography. *Bull. Mason Clinic*, 1968, *22*, 125–131.

THE NEW SPECIAL NAMES OF SI UNITS IN THE FIELD OF IONIZING RADIATIONS

At the meeting of the International Commission on Radiation Units and Measurements (ICRU) in July, 1974, the extensive worldwide response to the previous correspondence on SI units[1,2] was discussed is considerable detail. A statement on SI units was then prepared and sent to the International Committee of Weights and Measurements, CIPM, for consideration. In this statement the ICRU recommended the adoption of special names for the SI unit the joule per kilogram, J/kg, when applied to absorbed dose and related quantities, and for the SI unit the reciprocal second, s^{-1}, when applied to the quantity activity as a measure of the rate of spontaneous nuclear transformation of radioactive nuclides. The ICRU statement was also discussed at the IAEA symposium on Advances in Biomedical Dosimetry, March 10–14, 1975.[3] The CIPM treated the ICRU proposals with expedience and drafted two resolutions for consideration by the 15th General Conference of Weights and Measures, CGPM, at its meeting in May-June, 1975. At this meeting, which celebrated the 100 year anniversary of the Meter Convention, the CGPM adopted the following resolutions:

Resolution H 1. The Fifteenth Conférence Générale des Poids et Mesures: owing to the urgency, expressed by the International Commission on Radiation Units and Measurements (ICRU), to extend the use of the International System of Units to research work and applications in radiology; owing to the necessity of making the use of units as simple as possible for non-specialists; taking into account the seriousness of the risk of errors in therapy; *Adopts* the following special name of SI unit for activity: the becquerel, symbol Bq, equal to the second to the power minus one.

Resolution H 2. The Fifteenth Conférence Générale des Poids et Mesures: owing to the urgency, expressed by the International Commission on Radiation Units and Measurements (ICRU), to extend the use of the International System of Units to research work and applications in radiology; owing to the necessity of making the use of units as simple as possible for non-specialists; taking into account the seriousness of the risk of errors in therapy; *Adopts* the following special name of SI unit for ionizing radiations: the gray,* symbol Gy, equal to the joule per kilogram.

Thus the SI unit the joule per kilogram when used for ionizing radiation (absorbed dose, kerma, specific energy imparted, etc.) is given the name *gray* with the symbol *Gy*; 1 Gy = 100 rad = 1 J/kg. And the SI unit the reciprocal second (one per second) for activity is given the name *becquerel*, symbol *Bq*. 1 Bq = $1 s^{-1} \approx 2.703 \times 10^{-11}$ Ci.

Antoine Henri Becquerel (1852–1908) discovered radioactivity in 1896 ("rayons de Becquerel") and was given the Nobel Prize in physics in 1903 together with Marie and Pierre Curie.

Louis Harold Gray (1905–1965) made one of the most fundamental contributions to radiation dosimetry, the principle now known as the Bragg-Gray Principle.

The ICRU did not recommend to the International Committee of Weights and Measures that a special name be given to the SI unit of exposure to replace the currently used röntgen.

The ICRU now recommends that the special units the rad, the röntgen and the curie be gradually abandoned over a period not less than about 10 years and replaced by the SI units the gray (Gy), the coulomb per kilogram (C/kg) and the becquerel (Bq), respectively. It is desirable that preparatory steps be taken as soon as possible

* The gray is the SI unit of absorbed dose. The gray can also be used with other physical quantities expressed in joules per kilogram provided these quantities belong to the field of ionizing radiations.

by various national and international bodies in order to facilitate a smooth, safe and efficient transition.

In its report "Supplement to ICRU Report 19, Dose Equivalent"[4] the ICRU published its decision that the physical dimension of the quantity *dose equivalent* should be the same as for absorbed dose. From the radiation protection point of view the availability of a proper set of quantities and units in this field is of paramount importance. The formulation (for selection) of a suitable unit for dose equivalent is therefore a special problem which will be thoroughly discussed by ICRU.

On behalf of the International Commission on Radiation Units and Measurements:

H. O. Wyckoff, *Chairman*
A. Allisy, *Vice Chairman*
K. Lidén, *Scientific Secretary*

K. Lidén
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REFERENCES

1. Lidén, K. SI units in radiology and radiation measurement. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 932–933.
2. Lidén, K. Special radiation units and/or SI units? Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *120*, 704.
3. Lidén, K. SI units in biomedical dosimetry. In: Advances in Biomedical Dosimetry. IAEA, Vienna, 1975 (in press).
4. International Commission on Radiation Units and Measurements: Dose Equivalent. Suppl. to Report 19, ICRU, Washington, D.C., 1973.

NEWS ITEMS

POSTGRADUATE COURSE "THE RADIOLOGY OF TRAUMA"

The Department of Radiology, Cornell University Medical College, presents a Postgraduate course on "The Radiology of Trauma" at the Cerromar Beach Hotel, Dorado Beach, Puerto Rico, January 19 through 22, 1976.

Cornell University Medical College and the New York Hospital-Cornell Medical Faculty include: John A. Evans, M.D., Harold A. Baltaxe, M.D., Michael D. F. Deck, M.D., Robert H. Freiberger, M.D., Jeremy J. Kaye, M.D., Morton A. Meyers, M.D., D. Gordon Potts, M.D., and Joseph P. Whalen, M.D.; *Guest Faculty:* David H. Baker, M.D., Robert Fraser, M.D., Robert McLelland, M.D., Guy Potter, M.D., and Elliot L. Sagall, M.D.

The course is approved for 18 hours of ACR Category 1 accreditation.

For further information, please contact: John A. Evans, M.D., Department of Radiology, Cornell University Medical College, 1300 York Avenue, New York, New York 10021.

POSTGRADUATE COURSE ON GASTROINTESTINAL TRACT

The University of Utah and George Washington University will offer the thirteenth Radiology Seminars course, "The Gastrointestinal Tract," at the Las Vegas Hilton Hotel, Las Vegas, Nevada, April 1 through 4, 1976.

The format, introduced by Radiology Seminars, permits participants close contact with each faculty member. Nine 1½ to 2 hour seminars will be given. Teaching and case discussion will center on original x-ray films. The unique format requires that seminar groups be restricted to 10–12 participants; registration will be limited to 100 persons. The Course is approved for 20 hours of Category 1 credit.

For further information, please contact: P. Ruben Koehler, M.D., Department of Radiology, University of Utah Medical Center, Salt Lake City, Utah 84132.

DECISION MAKING IN DIAGNOSTIC MEDICINE

A meeting, sponsored by the Society of Nuclear Medicine, to be held on January 25, 1976, will focus on the theoretical, economic, technical, and clinical aspects of medical decision making with particular emphasis on the utilization of nuclear medicine and radiographic procedures. This full day meeting will be held in Atlanta, Georgia.

For further information, please contact: The Society of Nuclear Medicine, 475 Park Avenue South, New York, New York 10016.

COURSE IN ULTRASOUND

An exciting postgraduate course in Sonic Medicine has been organized to cover a three month period of in-residence training and will cover all aspects of ultrasonic technology as used today. It is designed to provide background, techniques, experience, and knowledge so that the individual will be able to set up both an ultrasonic laboratory and training program. The entire course of three months is broken down into segments which may be divided among individuals with different interests.

The material will cover acoustics, instrumentation, scanning, and applications to obstetrics, gynecology, ophthalmology, cardiology (adult and pediatric), the abdomen, the urinary tract, and the nervous system.

For the benefit of the students, enrollment for each three month course will be limited. The second course will begin January 5, 1976, and the third on April 12, 1976.

For further information, please write to: Division of Continuing Education, Bowman Gray School of Medicine, 300 South

Hawthorne Road, Winston-Salem, North Carolina 27103.

SYMPOSIUM ON RADIOPHARMACEUTICAL DOSIMETRY

A symposium will be held between April 26 and 29, 1976, in Oak Ridge, Tennessee. It will cover the most up-to-date concepts of dosimetry as it relates to the use of radiopharmaceuticals. Invited and submitted papers will focus on the various facets of the problem in an attempt to resolve some of the continuing questions on dosimetry.

It is sponsored by the Oak Ridge Associated Universities with co-sponsorship of the Energy Research and Development Administration, the National Cancer Institute, the Bureau of Radiological Health, and the Health Physics Society. For further information, please contact: Roger J. Cloutier, Chairman, Special Training Division, Oak Ridge Associated Universities, P. O. Box 117, Oak Ridge, Tennessee 37830.

MIDWINTER RADIOLOGICAL CONFERENCE

The 28th annual Midwinter Radiological Conference of the Los Angeles Radiological Society will be held in Los Angeles on January 30 and 31 and February 1, 1976. Parallel sessions will be devoted to diagnostic radiology and radiation therapy. Workshops on the first day will require reservations. Commercial exhibits will be presented.

There will be available a post-convention seminar in Hawaii, with diagnostic sessions each morning for a week. Further optional extensions are available. The sessions are approved for continuing education credit.

For further information, please write to: J. Stanley Lance, M.D., Chairman, Midwinter Conference Committee, Los Angeles Radiological Society, Los Angeles, California.

CANCER PREVENTION AND DETECTION

The third International Symposium on Detection and Prevention of Cancer will be held in New York City, April 26 to May 1, 1976. Conferences, panels, symposia, courses, and workshops are planned for the review of recent advances in immunology, tumor susceptibility, identification of high risk groups, and clinical and laboratory manifestations of cancer.

The meeting is co-sponsored by the Mount Sinai School of Medicine of The City University of New York, The American Cancer Society, and The International Agency for Research on Cancer of the World Health Organization.

The program committee will receive papers until November 15, 1975. Category 1 credit is offered for this meeting.

For additional information and registration forms, please write to: H. E. Nieburgs, M.D., Mount Sinai School of Medicine of the City University of New York, Fifth Avenue and 100th Street, New York, New York 10029.

DIAGNOSTIC RADIOLOGY OF THE URINARY TRACT

The Louisiana State University will sponsor the Aspen Seminar on Diagnostic Radiology of the urinary tract, in Aspen, Colorado, February 22 through February 26, 1976. Sessions will be held in the early morning, late afternoon and evening.

This will be an interdisciplinary continuing education experience in diagnostic uroradiology, surgical urology, radiation therapy, and uropathology, covering neoplastic, inflammatory, and traumatic conditions.

Category 1 credit is offered.

For further information, please contact: Erich K. Lang, M.D., Program Director, Professor and Head, Department of Radiology, Louisiana State University School of Medicine in Shreveport, P.O. Box 3932, Shreveport, Louisiana 71130.

BOOK REVIEW

Clark's Positioning in Radiology. Ninth edition. In 2 Volumes. By Kathleen C. Clark, M.B.E., Formerly Principal of the Ilford Department of Radiography and Medical Photography at Tavistock House, London. Revised and Edited by James McInnes, F.S.E., Society of Radiographers and Royal Photographic Society, London. Part I: Routine Radiography, pp. 480, with 1,700 illustrations, price, $29.95. Part II: Specialized Radiography, pp. 335, with 1,250 illustrations, price, $32.50. Distributed by Year Book Medical Publishers, 35 East Wacker Drive, Chicago, Ill. 60501.

This is the ninth edition of this remarkable book, and the first to appear in 2 volumes. The seventh edition was reviewed in this Journal in December, 1957. It is a beautiful, exhaustive, well-organized book. It would be difficult to imagine any problem in adult radiography which is not covered by information here.

The presentation is divided generally into anatomic regions, and the wealth of material about each region is subdivided further by indication of the basic positions for routine examination. For each position shown there is a clear, beautifully reproduced photograph of the patient during examination, and immediately adjacent on the page, the resulting roentgenogram. The quality of the reproduction of the roentgenograms is excellent, and the illustrations provide a suitable paragon for the technologist. Similar treatment is given to problem patients: those in casts or other immobilization; those unable to assume positions necessary for standard projections; and those with deformities. In many instances roentgenograms of disease processes are presented to enable the technologist to make necessary compensations. There are even examples purposely included to show an improper examination and its correction. Each section includes detailed discussion of the pertinent anatomy.

Throughout the accompanying text are admonitions and means to minimize patient irradiation. Standard exposure technique for the average patient is included with each position, and instructions for modification are given.

Division of the work into two volumes makes this large amount of information more readily accessible. In addition to extensive coverage of the axial and appendicular skeleton in 11 sections, the first, basic volume includes examination of the chest, abdomen, and skull. Specialized radiography in the second volume includes examination of the salivary glands, temporal bones, neuroradiography, angiography, tomography, cineradiography, and many others. As in earlier editions, there are supplements covering contrast media and personnel protection.

The book describes pediatric radiography less extensively, but adequately. The language, contrast media, and films are British, but the illustrations and information are universal.

For the old fashioned radiologist who feels that he cannot or should not complain about the technologists' efforts unless the radiologist can do it better himself, this book is a *sine qua non*. It is even more necessary in the department of the radiologist who does not feel that way.

E. Frederick Lang, M.D.

BOOKS RECEIVED

Arthrography of the Shoulder: the Diagnosis and Management of the Lesions Visualized. By Julius S. Neviaser, M.D., Clinical Professor of Orthopaedic Surgery, George Washington University Medical School; Formerly, Consultant-Lecturer at the U. S. Naval Hospital, Bethesda, Md., and the Walter Reed Army Hospital, Washington, D. C.; Past Chairman of the Departments of Orthopaedic Surgery at the Washington Hospital Center, Washington, D. C., and the Prince George's General Hospital, Cheverly, Md. Cloth. Pp. 272, with many illustrations. Price, $22.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1975.

Textbook of Nuclear Medicine Technology. Second edition. By Paul J. Early, B.S., President, Nuclear Medicine Associates, Inc., Cleveland, Ohio; Muhammad Abdel Razzak, M.B.B.Ch., D.M., M.D. (Cairo), F.A.C.P. (U.S.A.), Professor of Medicine, Medical Unit and Division of Nuclear Medicine, Faculty of Medicine, Cairo University, Cairo, A.R.E.; Nuclear Medicine Consultant, Medical Services, Egyptian Armed Forces; and D. Bruce Sodee, M.D., F.A.C.P., Associate Professor of Radiology (Nuclear Medicine), George Washington University, Washington, D. C.; Director, Nuclear Medicine Institute, Cleveland, Ohio; Director, Nuclear Medicine Department, Hillcrest Hospital, Cleveland, Ohio.

Cloth. Pp. 496, with 412 illustrations. Price, $18.95. C. V. Mosby Company, Publishers, 3301 Washington Boulevard, St. Louis, Mo. 63103, 1975.

Diagnostic Uses of Ultrasound. By Barry B. Goldberg, M.D., Head, Section of Diagnostic Ultrasound, Episcopal Hospital; Professor of Radiology, Temple University Health Sciences Center, Philadelphia, Pa.; Morris N. Kotler, M.D., Head, Non-Invasive Cardiac Laboratory, Hahnemann Hospital; Associate Professor of Medicine, Hahnemann Medical College, Philadelphia, Pa.; Marvin C. Ziskin, M.D., M.S., Bm.E., Associate Professor of Radiology and Medical Physics, Temple University Health Sciences Center, Philadelphia, Pa.; and Robert D. Waxman, B.A., Consultant in Ultrasound, Episcopal Hospital, Philadelphia, Pa. Cloth. Pp. 466, with many illustrations. Price $30.00. Grune & Stratton, Inc., 111 Fifth Avenue, New York, N. Y. 10003, 1975.

Radionuclide Studies of the Spleen. By Richard P. Spencer, M.D., Ph.D., Professor and Chairman, Department of Nuclear Medicine, University of Connecticut School of Medicine, Farmington, Conn.; and Howard A. Pearson, M.D., Professor and Chairman, Department of Pediatrics, Yale University School of Medicine, New Haven, Conn. Cloth. Pp. 209, with some figures. Price, $34.95. CRC Press, Inc., 18901 Cranwood Parkway, Cleveland, Ohio 44128, 1975

Synopsis of Gynecologic Oncology. By Philip J. Disaia, M.D., C. Paul Morrow, M.D., and Duane E. Townsend, M.D., Los Angeles County/University of Southern California Medical Center; Women's Hospital, Department of Obstetrics and Gynecology, Section of Gynecologic Oncology, Los Angeles, Calif. Cloth. Pp. 344, with many figures. Price, $19.50. John Wiley & Sons, Inc., Publishers, 605 Third Avenue, New York, N. Y. 10016, 1975.

Photographic Atlas of Fetal Anatomy. By Wesley W. Parke, Ph.D., Professor of Anatomy, Southern Illinois University School of Medicine. Cloth. Pp. 435, with many illustrations. Price, $34.50. University Park Press, Chamber of Commerce Building, Baltimore, Md. 21202, 1975.

Tomography and Cross Sections of the Ear. By Galdino E. Valvassori, M.D., and Richard A. Buckingham, M.D. Cloth. Pp. 200, with 510 illustrations. Price, $65.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1975.

Tracer Methods in Hormone Research. By E. Gurpide, Ph.D., Professor of Biochemistry and Obstetrics and Gynecology, Mount Sinai School of Medicine, City University of New York, New York, N. Y. Cloth. Pp. 188, with 35 figures. Price, $31.00. Volume 8 of Monographs on Endocrinology. Springer-Verlag New York, Inc., 175 Fifth Avenue, New York, N. Y. 10010, 1975.

Radiology in Primary Care. Edited by Glenn V. Dalrymple, M.D., Professor and Chairman, Department of Radiology; Professor of Biometry, Physiology, and Biophysics, University of Arkansas Medical Center; Chief, Radiology Service, Veterans Administration Hospital; Professor of Medical Physics, University of Arkansas at Little Rock, Little Rock, Ark.; and John E. Slayden, M.D., Assistant Professor of Radiology (Nuclear Medicine), University of Arkansas Medical Center, Little Rock, Ark. Paper. Pp. 330, with 717 illustrations. Price, $22.50. C. V. Mosby Company, 3301 Washington Boulevard, St. Louis, Mo. 63103, 1975.

Tecnica e Metodologia Radiologiche: Basi Fisiche ed Applicazioni Diagnostiche. By Rosario Corseri, Libero Docente in Radiologia. Cloth. Pp. 1,085, with many figures. Price, L. 30.000. Cedam, Via Jappelli, 5—35100 Padova, Italy, 1975.

Carcinogenesis and Radiation Risk: a Biomathematical Reconnaissance. By W. V. Mayneord, C.B.E., D.Sc., F.R.S., Professor Emeritus of Physics as applied to Medicine, Institute of Cancer Research (University of London); Consultant to the Central Electricity Generating Board; and R. H. Clarke, Ph.D., Central Electricity Generating Board, Research Department, Berkeley Nuclear Laboratories, Berkeley, Gloucestershire, England. Paper. Pp. 112. British Journal of Radiology, Supplement No. 12. Published by the British Institute of Radiology, London, 1975.

Nuclear Medicine. Edited by Henry N. Wagner, Jr., M.D., Professor of Medicine, Radiology, and Environmental Health and Director of Nuclear Medicine and Radiation Health, The Johns Hopkins Medical Institution, Baltimore, Md.; with the collaboration of Irvin Block, Senior Editor, Hospital Practice. Cloth. Pp. 254, with many illustrations. Price, $18.95. HP Publishing Co., Inc., 485 Madison Avenue, New York, N. Y. 10022, 1975.

Chemotherapy of Urogenital Tumors. By Gerald P. Murphy, M.D., D.Sc., and Arnold Mittelman, M.D., Roswell Park Memorial Institute, Buffalo, N. Y. Foreword by Willet F. Whitmore, Jr., M.D., Chief, Urologic Service, Memorial Hospital for Cancer and Allied Diseases, New York, N. Y. Cloth. Pp. 268, with some figures. Price, $24.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1975.

A Study Guide in Nuclear Medicine: a Modern, Up-to-Date Presentation. Compiled and Edited by Fuad Ashkar, M.D., Assistant Professor of Radiology and Medicine, Division of Nuclear Medicine, University of Miami School of Medicine, Miami, Fla.; August Miale, Jr., M.D., Associate Professor of Radiology; Chief, Nuclear Medicine Service I, University of Miami School of Medicine,

Miami, Fla.; and William Smoak, M.D., Associate Professor of Radiology; Chief, Nuclear Medicine Service II, University of Miami School of Medicine, Miami, Fla. Cloth. Pp. 473, with some figures. Price, $22.75, (Paper, $17.50). Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1975.

CORONARY ARTERIOGRAPHY. By Goffredo G. Gensini, M.D., Clinical Professor of Medicine, State University of New York Upstate Medical Center; Director, The Monsignor Toomey Cardiovascular Laboratory and Research Department, St. Joseph's Hospital Health Center, Syracuse, N. Y. Cloth. Pp. 488, with many figures. Price, $48.00. Futura Publishing Company, 295 Main Street, Mount Kisco, N. Y. 10549, 1975.

TEXTBOOK OF ANATOMY AND PHYSIOLOGY IN RADIOLOGIC TECHNOLOGY. Second edition. By Charles A. Jacobi, B.Sc. FNCRT (A.R.C.R.T.), R.T. (A.R.R.T.), M.T. (A.S.C.P.), Professor and Chairman, Department of Radiologic Technology, University of Nevada, Las Vegas, Nev.; member, Radiological Safety Board, University of Nevada System; member, Preprofessional Advisement Committee, University of Nevada, Las Vegas; member, Education Qualifications and Standards Committee, A.R.C.R.T.; previously Assistant, Department of Medical-Dental Associates; and member of Radioisotopes Committee, Oregon Technical Institute, Klamath Falls, Oregon. Cloth. Pp. 437, with 361 illustrations. Price, $14.00. C. V. Mosby Company, 3301 Washington Boulevard, St. Louis, Mo. 63103, 1975.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. James Franklin Martin, 300 S. Hawthorne Rd., Winston-Salem, N. C. 27103. Annual Meeting: Marriott Hotel, Atlanta, Ga., Sept. 30–Oct. 3, 1975.

American Radium Society
Secretary, Richard H. Jesse, Dept. of Surgery, M. D. Anderson Hosp. and Tumor Institute, Houston, Tex. 77025. Annual Meeting: Bayshore Inn, Vancouver, B. C., Canada, May 9–14, 1976.

Radiological Societyof North America
Secretary, Dr. Theodore A. Tristan, Harrisburg, Pennsylvania. Annual Meeting: McCormick Place on the Lake, Chicago, Ill., Nov. 30–Dec. 5, 1975.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting.

Section on Radiology, American Medical Association
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.
Oral examinations will be held: Statler Hilton, Dallas, Texas, Dec. 8–13, 1975; Marriott Motor Hotel, Chicago, Ill., June 14–19, 1976; Sheraton Biltmore, Atlanta, Ga., Dec. 6–11, 1976.
Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.
Deadline for filing applications for any examination in 1976 is September 30, 1975.

American Board of Nuclear Medicine, Inc. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.
Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017.

American Association of Physicists in Medicine
Secretary, Dr. Robert G. Waggener, Dept. Radiol., Univ. Tex. Health Sciences Center, San Antonio, Tex. 78284 Annual Meeting:

American Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wis., 54601. Annual Meeting: Hyatt Regency Hotel in the Embarcadero Center, San Francisco, Calif., Oct. 8–12, 1975.

American Nuclear Society
Program Chairman, Werner K. Kern, IRD, Space Nuclear Systems, F 309, U. S. Atomic Energy Commission, Washington, D.C. 20545.

American Institute of Ultrasound in Medicine
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

American Society of Neuroradiology
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

Fourteenth International Congress of Radiology
Rio de Janeiro, Brazil, Oct. 23–29, 1977.
President Elect: Prof. Dr. Nicola C. Caminha; *General Secretary:* Dr. Durval Couto. Av. Churchill, 97-50 and-LC-39 20,000 Rio de Janeiro, Brazil.

International Society of Radiology
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

Twelfth Inter-American Congress of Radiology
President, Dr. Publio Vargas Pazzos, Casilla 5227, Guayaquil, Ecuador. Meeting in Quito, Ecuador, 1979.

Inter-American College of Radiology
President, Manuel Viamonte, Jr., M.D., Mt. Sinai Medical Center, 4300 Alton Rd., Miami Beach, Fla. 33140.
Secretary, Gaston Morillo, M.D., Jackson Memorial Hosp., 1700 N.W. 10th Ave., Miami, Fla. 33152.
Counselor for the United States, J. A. del Regato, M.D., Univ. of So. Fla. College of Medicine, 13000 No. 30th St., Tampa, Fla. 33612.

Third Congress of the European Association of Radiology
Secretary-General, P.O. Box 14, 79 Lauriston Place, Edinburgh BHI IDB, Scotland.

Second Asian and Oceanian Congress of Radiology
Secretary, Dr. Buenaventura U. V. Angtuaco, Congress Secretariat Headquarters, 2161 Taft Ave., Manila, Philippines. Meeting: Manila, Philippines, Nov. 10–14, 1975.

Alabama Chapter of ACR
Secretary-Treasurer, Dr. Lawrence E. Fetterman, 1720 Springhill Ave., Suite 201, Mobile, Ala. 36604. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society, Chapter ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

American Osteopathic College of Radiology
Annual Scientific Meeting: Americana Hotel, Bar Harbor, Fla. Program Administrator: Lawrence J. Jordan, D.O., Suite 21, Ponce American Bldg., 4601 Ponce de Leon Blvd., Coral Gables, Fla. 33146.

American Thermographic Society
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Irwin M. Freundlich, 1501 N. Campbell Ave., Dept. Radiol., Tucson, 85724. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-Tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. Richard Seibold, Jr., Wadley Hosp., Texarkana, Ark.—Tex. 75501. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Arch W. Templeton, Univ. of Kansas Med. Center, 39th and Rainbow Blvd., Kansas City, Kansas 66103. Annual Meeting.

Atlanta Radiological Society
Secretary-Treasurer, Dr. Marshall J. Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on fourth Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Bavarian-American Radiologic Society
Secretary, LTC. Marco J. DiBiase, M.D., Radiology Service, Heidelberg, 130 Station Hosp., APO N. Y. 09102. Meets quarterly.

Blockley Radiological Society
Secretary-Treasurer, Dr. William H. Green, 616 East 24th St., Chester, Pa. 19013.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Ralph Brancaccio, 7901 4th Ave., Brooklyn, N. Y. 11209. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. George J. Alker, Jr., E. J. Meyer Mem. Hosp., Buffalo, N. Y. 14215. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. Duane J. Gillum, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Alfred Brettner, Auburn Mem. Hosp., Auburn, N. Y. 13021. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Daniel E. Lewis, 309 East State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, March, and May at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer Dr. E. Jerome Schoolar, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harold J. Lasky, 55 E. Washington St., Suite 1735, Chicago, Ill. 60602. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Charles M. Greenwald, 7007 Powers Blvd., X-Ray Dept., Parma, Ohio 44129. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. John Pettigrew, Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Robert R. Burns, 1400 Stemmons Ave., Dallas, Tex. 75208. Meets the 3rd Monday of every month at 6:30 P.M., at the Airport Hotel, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Ekkehard S. Schubert, Wilmington Med. Center, P. O. Box 1951, Wilmington, Del. 19899.

East Bay Radiological Society
Secretary-Treasurer, Dr. Robert L. Stein, 43 Dormidera Ave., Piedmont, Calif. 94611. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary. Dr. Paul J. Popovich, 1350 S. Hickory St., Melbourne, Fla. 32901. Meets twice annually, in the spring with the Annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Herbert Johnson, Davis Blvd., Tampa, Fla. 33606. Meets on 4th Thursday of the months January, March, May, October, and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Jay I. Rozen, Suite 216, 6400 Prospect, Kansas City, Mo. 64132. Meets 5 times a year.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary, Dr. David C. Hillman, P. O. Box 610544, North Miami, Fla. 33161. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Sidney B. W. Wong, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Robert D. Dooley, Hinsdale Med. Center, Hinsdale, Ill. 60521. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John A. Knote, Home Hosp., Lafayette, Ind. 47902.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Dale L. Roberson, 1948 First Ave., NE, Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific session is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph In-

firmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Harry L. Stein, North Shore Univ. Hosp., 300 Community Dr., Manhasset, N. Y. 11030.

Los Angeles Radiological Society
Secretary, Dr. Arthur F. Schanche, 333 North Prairie Ave., Inglewood, Calif. 90301. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference. Century Plaza Hotel, Los Angeles, Calif.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. J. Gardiner Bourque, 3155 Stagg Dr., Suite 230, Doctors Bldg., Beaumont, Texas 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Peter E. Giustra, Dept. Radiology, Penobscot Bay Medical Center, Rockland, Maine 04841. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Joseph T. Ferrucci, Jr., Massachusetts Gen. Hosp., Boston, Mass. 02114.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Thomas P. Coburn, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38163. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Francis P. Shea, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin E. Goldberg, Box 292, Mayo Memorial Health Sciences Center, Minneapolis, Minn. 55455.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Robert Bodmer, Suite 622 Doctors Bdg., Omaha, Neb. 68131. Meets the third Wednesday of the month, Oct. through March, at the Clarkson Hosp., Omaha, Neb. and the fourth Friday of April at the Lincoln Country Club, Lincoln, Neb.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

N.E. Society for Radiation Oncology
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

New England Roentgen Ray Society
Secretary, Dr. Melvin E. Clouse, 185 Pilgrim Rd., Boston, Mass. 02115. Meets the third Friday of Oct. and Nov., and third Thurs. of each month. Jan. through May, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass. at 3:00 P.M. Annual Meeting: May 20, 1976.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. W. M. Jordan, 1100 Central Ave. S.E., Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference.

New York Roentgen Society
Secretary-Treasurer, Dr. Thomas C. Beneventano, 111 East 210th St., Bronx, N. Y. 10467. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., April 22–24, 1976. A.M.A. Cat. I credit. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 6 Secor Dr., Port Wash., N. Y. 11050.

North American Society of Cardiac Radiology
Secretary-Treasurer, Dr. Erik Carlson, Univ. Calif., San Francisco, Calif. 94143.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, P. O. Box 6001, Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

NORTHEASTERN OKLAHOMA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Donald R. Morton, Dept. of Radiology, St. Clare's Hosp., Schenectady, N. Y. 12304. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. John D. Earle, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. Patrick J. Grinsell, 1207 Fairchild Ct., Woodland, Calif. 95695. Meets on the fourth Monday, Sept. through May at the Mansion Inn, Sacramento, Calif.

NORTHWESTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

NORTHWESTERN OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Richard W. Siders, 421 Michigan, Toledo, Ohio 43624. Meets 3rd Wednesday from September to May.

OHIO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. James Farmer, Cleveland, Ohio.

OKLAHOMA STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

ORANGE COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. William E. Danko, 2601 E. Chapman, Orange, Calif. 92667. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

OREGON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ray F. Friedman, 3324 S. W. 44th St., Portland, Ore. 97221. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Kenneth D. Moores, 1118 9th Ave., Seattle, Wash. 98101. Annual Meeting: Benson Hotel, Portland, Ore., May 7–9, 1976.

PENNSYLVANIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Joseph A. Marasco, Jr., St. Francis General Hosp., Pittsburgh, Pa. 19141.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. Marvin E. Haskin, 230 N. Broad St., Philadelphia, Pa. 19102. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. Klaus M. Bron, Presbyterian-Univ. Hosp., Pittsburgh 15213. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

RADIATION RESEARCH SOCIETY
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting.

RADIOLOGICAL SOCIETY OF CONNECTICUT, INC., CHAPTER OF ACR
Secretary, Dr. Gerald L. Baker, 85 Jefferson St., Hartford, Conn. 06106. Meets in Oct., Jan., March, and June.

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

RADIOLOGICAL SOCIETY OF NEW JERSEY, CHAPTER OF ACR
Secretary, Dr. Fred M. Palace, 11 Dale Dr., Morristown, N.J. 07960. Monthly meetings in East Orange and Trenton, N.J. Semi-Annual Meeting in Nov.

RADIOLOGICAL SOCIETY OF RHODE ISLAND, CHAPTER OF ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA
Secretary-Treasurer, Dr. A. Franklin Turner, LAC-USC Medical Center, Room 3418, 1200 North State St., Los Angeles, Calif. 90033. Meets three times a year, usually October, February and May.

RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

REDWOOD EMPIRE RADIOLOGICAL SOCIETY
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

RICHMOND COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Frederick S. Vines, 11830 Rothbury Dr., Richmond, Va. 23235. Meets third Thursday of each month at the Aberdeen Barn.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, Rochester General Hosp., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual Meeting:

SAN ANTONIO-CIVILIAN MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

SAN DIEGO RADIOLOGICAL SOCIETY
President, Dr. Donald J. Fleischli, 7849 Fay Ave., La Jolla, Calif. 92037. Meets the first Wednesday of each month Sept. through June at the University Club.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Robert L. Koch, 3838 California St., Suite 106, San Francisco, Calif. 94118.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION OF RADIOLOGY, NATIONAL MEDICAL ASSOCIATION
Secretary, Dr. Ruby Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broadway, Baltimore, Md. 21205. Annual meeting: Marriott Hotel, Atlanta, Ga., Sept. 28–29, 1975.

Society of Gastro-intestinal Radiologists
Secretary-Treasurer, Dr. John R. Amberg, University Hospital, 225 W. Dickinson St., San Diego, Calif. 92103.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual Meeting.

South Bay Radiological Society
Secretary, Dr. Robert Mindalzun, Valley Radiologists, Inc., 100 O'Connor Dr., San Jose, Calif. 95128. Meets the 2nd Monday of each month at the Santa Clara County Medical Society Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society Chapter of ACR
Secretary-Treasurer, Dr. Richard T. Love, Santa Barbara Med. Clin., 215 Pesetas Lane, Santa Barbara, Calif. 93110. Meets fourth Tuesday of alternate months, Sept., Nov., Jan., March and May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Association
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. J. W. Maxwell, P.O. Box 2144 Mobile Ala. 36601. Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 30–Feb. 1, 1976.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Jerry W. Grise, Methodist Hosp., 1265 Union Ave., Memphis, Tenn. 38104. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bdg., San Antonio, Tex. 78205.

The Fleischner Society
Secretary, Dr. E. Robert Heitzman, State University of New York Upstate Medical Center, Syracuse, N. Y. 13210. Annual Meeting:

Tri-State Radiological Society
Secretary, Dr. Thomas Harmon, St. Mary's Hosp., Evansville, Ind. 47750. Meets bimonthly on the third Wednesday at area Hospitals.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Patrick D. Lester, Dept. Radiol., University of Utah Medical Center, Salt Lake City, Utah 84112. Meets quarterly at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 3500 Kensington Ave., Suite 2-A, Richmond, Va. 23221.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Donald J. Hesch, 3216 N.E. 45th Place, Seattle, Wash. 98105.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Leonard Cutler, 16 Guion Place, New Rochelle, N. Y. 10802. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. June Unger, Wood VA Hosp., Wood, Wis. 53193. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wis. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Rafael Umaña-Umaña, Apartado 8-5340, San José, Costa Rica.

Asociacón de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9², Calle A 0–05, Zona 1, Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St. Catherine St. West, Montreal 107, Que., Canada. Annual Meeting:

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada.

Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

FACULTY OF RADIOLOGISTS
Honorary Secretary, P. D. Thomson, 28 Portland Pl., London, W1N 4DE.

MONTREAL RADIOLOGICAL STUDY CLUB
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

SOCIÉTÉ CANADIENNE-FRANÇAISE DE RADIOLOGIE
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

THE HOSPITAL PHYSICISTS' ASSOCIATION
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

TORONTO RADIOLOGICAL SOCIETY
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

THE ROYAL AUSTRALASIAN COLLEGE OF RADIOLOGISTS
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

SOUTH AMERICA

COLÉGIO BRASILEIRO DE RADIOLOGIA
Secretary-General, Dr. José Marcos Pires de Oliveira, Caixa Postal 5984, São Paulo, Brazil.

FEDERACIÓN ARGENTINA DE SOCIEDADES DE RADIOLOGÍA
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

SOCIEDAD ARGENTINA DE RADIOLOGÍA
Secretary-General, Dr. Alba de la Torre, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

SOCIEDAD BOLIVIANA DE RADIOLOGÍA
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

SOCIEDADE BRASILEIRA DE RADIOLOGIA
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

SOCIEDADE BRASILEIRA DE RADIOTERAPIA
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

SOCIEDAD CHILENA DE RADIOLOGÍA
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

SOCIEDAD COLOMBIANA DE RADIOLOGÍA
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

SOCIEDAD ECUATORIANA DE RADIOLOGÍA
Secretary, Dr. Luis Blum, Casilla 3712, Guayaquil, Ecuador.

SOCIEDAD PARAGUAYA DE RADIOLOGÍA
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

SOCIEDAD PERUANA DE RADIOLOGÍA
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

SOCIEDAD DE RADIOLOGÍA DEL ATLANTICO
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

SOCIEDAD DE RADIOLOGÍA DEL NORDESTE ARGENTINO
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

SOCIEDAD DE RADIOLOGÍA DE LA PLATA
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

SOCIEDAD DE RADIOLOGÍA, CANCEROLOGÍA Y FÍSICA MÉDICA DEL URUGUAY
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

SOCIEDADE DE RADIOLOGÍA DE PERNAMBUCO
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

SOCIEDAD DE ROENTGENOLOGÍA Y MEDICINA NUCLEAR DE LA PROVINCIA DE CÓRDOBA
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

SOCIEDAD DE RADIOLOGÍA, RADIOTERAPÉUTICA Y MEDICINA NUCLEAR DE ROSARIO
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

SOCIEDAD SALTEÑA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

SOCIEDAD VENEZOLANA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

CONTINENTAL EUROPE

ÖSTERREICHISCHE RÖNTGEN-GESELLSCHAFT
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

SOCIÉTÉ ROYALE BELGE DE RADIOLOGIE
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

SOCIÉTÉ EUROPÉENNE DE RADIOLOGIE PÉDIATRIQUE
Secretary, Dr. J. Corbaton, Clinica Infantil "La Paz," Av. Generalissimo, 117 Madrid 34, Spain. Annual Meeting:
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 730, Paris Cadet 15, France.

SOCIÉTÉ FRANÇAISE DE RADIOLOGIE MÉDICALE, MÉDECINE NUCLÉAIRE ET ÉLECTROLOGIE, and its branches: SOCIÉTÉ DU SUD-OUEST, DU LITTORAL MÉDITERRANÉEN, DU CENTRE ET LYONNAIS, DU NORD, DE L'OUEST, DE L'EST, DU MASSIF CENTRAL, D'OUTRE-MER ET D'ÉTRANGER.
Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

SOCIÉTÉ FRANÇAISE DE NEURORADIOLOGIE
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting:

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK. 8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Norsk Forening for Medicinsk Radiologi
General Secretary, Gunnar Stake, Rikshospitalet, Oslo, Norway.

Scandinavian Radiological Society
Secretary-General, Professor C. F. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägn 5 C., Finland. Annual Meeting.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary-General, Dr. Pilar Gallar Barberá, Villanueva, 11, Madrid 1, España. Meets every other Saturday evening of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa i-Portugal.

Svensk Förening för Medicinsk Radiologi
Secretary, Dr. Hanz Ringertz, Röntgendiagnostika Avdelningen, Karolinska Sjukhuset 104 01 Stockholm 60, Sweden.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Bengal Radiological Association
Honorary Secretary, Dr. B. Chatterji, 262 Rash Behari Ave., Calcutta 700019, India. Meets 1st Fri. and 3rd Wed. of every month.

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwa', 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Majid Rooholamini, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
Secretary, Dr. M. A. Lerner, Hesharon Hosp., Petah-Tigva, Israel.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, C. A. Beetlestone, M.B., B.S., D.M.R.D., Dept. Radiol., University College Hosp., Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. A. Visser, P. O. Box 8850, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

ABSTRACTS OF RADIOLOGIC LITERATURE

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ROENTGEN DIAGNOSIS

Neck and Chest

Brock, Lord. Long survival after operation for cancer of the lung. *Brit. J. Surg.*, Jan., 1975, *62*, 1–5. (From: Guy's Hospital and the Brompton Hospital, and Department of Surgical Sciences, Royal College of Surgeons, London, England.)

The author shares his 35 year experience with 293 cases of cancer of the lung operated by lobectomy, simple pneumonectomy, and radical pneumonectomy. In his series, the over-all survival rate was 32 percent, with a mortality of 13 percent. In 86 cases where lobectomy was performed 34 patients (40 percent) lived five years or more. With simple pneumonectomy, 21 patients (24 percent) survived longer than five years. With radical pneumonectomy, 39 patients (33 percent) lived five years or longer. The five year survival rate following radical pneumonectomy of 39 percent approaches that of lobectomy and suggests that radical pneumonectomy does improve the chance of five year survival. Radical pneumonectomy in the author's series is an operation which the author described in 1955 and involves resection of a wider group of lymph nodes than are involved by the growth and which, otherwise, would not be removed. The survival rate is diminished when the lymph nodes are involved, but the author cites nine examples of patients who have survived longer than five years with positive nodes. In this author's experience, and in other published series, regardless of which type of operation is performed, there is a one in three chance of a five year survival with surgery.

The author cites many examples of extremely long term survival (longer than 20 years) which emphasizes the fact that cancer of the lung can be cured surgically. The author suggests that biological factors may play an important role in long term survival. In the author's experience, pleural infection postoperatively may enhance the long term survival rate. The author feels that preoperative irradiation is extremely useful and increases the chance for survival. He routinely uses preoperative irradiation in patients 35 years of age or younger and often advises preoperative irradiation in the older groups. He does not feel that postoperative irradiation, in his experience, has been beneficial. The author gives nine examples of patients who developed a new primary growth many years following resection of a lung neoplasm. Several of these examples were resected with good long term survival.—*Daniel P. Link, M.D.*

Higgins, George A., Shields, Thomas W., and Keehn, Robert J. The solitary pulmonary nodule: ten-year follow-up of Veterans Administration-Armed Forces cooperative study. *A.M.A. Arch. Surg.*, May, 1975, *110*, 570–575. (Address: George A. Higgins, M.D., VA Hospital, 50 Irving Street, N.W., Washington, D. C. 20422.)

A cooperative study by the Veterans Administration and Armed Forces was carried out with observation of over 1,100 patients with asymptomatic solitary pulmonary nodules. Three hundred and ninety-two of these nodules were found to be primary bronchogenic carcinoma. Long term survival of these patients with carcinoma seems to depend on size of the lesion, age at operation, and interval between the last normal and the first abnormal roentgenogram. The histologic cell type and the extent of the surgical resection did not seem to influence long term survival. There were 370 patients with carcinoma in whom resection was possible and only 67 were living and free of disease ten years following the operation.

Numerous tables and graphs accompany this excellent article. Especially interesting are the graphs comparing the survival rate in patients whose lesions were either smaller than or greater than 3.5 cm in diameter. A similar graph showing the much better survival rate in those patients under age 55 as compared to older patients, is also of interest.

This study covered 15 years and involved 65 VA hospitals and 13 military hospitals.—*Richard E. Kinzer, M.D.*

Macquet, V., Lafitte, P., Rogeaux, Y., and Pinchon, R. Influence des conditions météorologiques dans la survenue du pneumothorax spontané. (Influence of meteorological conditions on the occurrence of spontaneous pneumothorax.) *Lille méd.*, Nov., 1974, *19*, 901–906. (From: Service de Pneumologie, Centre Hospitalier, Tourcoing, France.)

The authors try to correlate meteorological statistics within a ten mile radius around Lesquin (a suburb of Lille, situated in a mining district) from June, 1967 to June, 1972, and the incidence of spontaneous pneumothorax in the region in those five years, namely, 180 cases.

Ninety-nine of these patients had pre-existing pulmonary conditions such as silicosis, emphysema, and arrested tuberculosis. This is taken into account in their statistical analysis. They observe that the greatest incidence of spontaneous pneumothorax occurs during the months of November, December, and January. The next most common occurrence is from June through August. These seasonal variations were evaluated in the light of changes of temperature, atmospheric pressure, relative humidity and climatic upheavals. They conclude that relative humidity rise (November through January) is the significant factor in causing spontaneous pneumothorax. Temperature changes are not of importance. Variations

in atmospheric pressure gains great significance only if the changes (rise or fall) are sharp (brutal is the word used by the authors).

Reviewing the literature, they note that the incidence of coronary thrombosis and susceptibility (due to lowered resistance) to infections follow almost the same seasonal pattern.—*Jerair N. Sarian, M.D.*

Abdomen

Wong, John, and Loewenthal, John. Heterotopic bone formation in abdominal scars. *Surg., Gynec. & Obst.*, June, 1975, *140*, 893–895. (From: Royal Prince Alfred Hospital, Sydney, Australia.)

Twelve patients with heterotopic ossification in abdominal scars were studied. Operative excision was required in seven: two for local symptoms; two for suspected local malignant recurrences; and three for further intra-abdominal procedures. Two of the operated cases and the five non-operated cases were confirmed roentgenographically.

Characteristically there appears a rock-hard mass in the upper end of a midline abdominal incision within one year postoperatively. The excised specimen usually shows osseous tissue which may contain active bone marrow. The bones measured from 3.0 to 15.0 cm in length in this series. Theories of origin include: (1) local seeding of periosteal or perichondral cells into the wound when either the xiphoid or the pubis borders the incision; or (2) the presence of multipotent embryonic connective tissue cells which undergo metaplasia. Once diagnosed roentgenographically, the only indication for excision would be local discomfort.—*Arch H. Hall, M.D.*

Laufer, Igor, Mullens, J. Edward, and Hamilton, John. The diagnostic accuracy of barium studies of the stomach and duodenum: correlation with endoscopy. *Radiology*, June, 1975, *115*, 569–573. (Address: Igor Laufer, M.D., Department of Radiology, McMaster University Medical Centre, Hamilton, Ont., Canada L8S 4J9.)

The authors compare the roentgenologic and endoscopic findings in a series of consecutive patients examined by fiberoptic endoscopy and compare the diagnostic accuracy of the standard barium meal with the accuracy of the double contrast technique. There were 240 patients available for this study. Most of the patients had had a barium study done within two weeks prior to the endoscopic examination and, because of this, the endoscopic findings were accepted as the correct diagnosis. In this study the authors were concerned with the detection of lesions rather than the pathologic diagnosis.

Roentgenologic errors were classified as either: (a) false negative lesions discovered endoscopically but not detected on the preceding roentgen-ray study; or (b) false positive lesions diagnosed roentgenologically in which adequate endoscopic examination of the area in question revealed no corresponding abnormality. One hundred and seventy-five of the patients had had a standard barium study and there were 39 roentgenological errors (22 percent). Approximately one-half of these errors were false negatives and one-half were false positives. Sixty-five patients were examined by double contrast technique and in this group there were four roentgenologic errors (six percent). Fluoroscopy was used primarily for determining the correct volume of barium, adequacy of mucosal coating and positioning rather than for definitive diagnosis, and the spot films obtained either by residents or technologists were then interpreted by the radiologists or a group of radiologists independently of the fluoroscopic observations.

The most common causes of error were failure to detect an abnormality on the roentgenogram and prominent mucosal folds which could either hide lesions or could simulate an ulcer crater. It is felt by the authors that the double contrast technique for the roentgenologic study of the stomach and duodenum offers a significant improvement in the diagnostic accuracy.—*Donald N. Dysart, M.D.*

Sankaran, S., and Walt, Alexander J. The natural and unnatural history of pancreatic pseudocysts. *Brit. J. Surg.*, Jan., 1975, *62*, 37–44. (Address: A. J. Walt, M.D., Department of Surgery, Wayne State University School of Medicine, Detroit, Mich. 48201.)

The authors present their experience with 131 pseudocysts of the pancreas in 112 patients. In their review, they analyze the natural history of the pancreatic pseudocysts, look at the different modes of therapy, and assess the significant complications.

Seventy-five percent of patients were in the fourth and fifth decades, but the patients ranged in age from 16 to 79 years. Eighty-six patients were men and 26 patients were women. Ninety percent of the pseudocysts were preceded by pancreatitis and ten percent followed trauma. Alcoholism was the predominant cause of pancreatitis. Many of the post-traumatic pseudocysts were produced by blunt or direct penetrating blows to the pancreas. Most of the patients showed characteristic symptoms and signs including upper abdominal pain and weight loss. A mass was palpable in only half of the patients. A number of the patients had chronic ascites and some patients had transient hypertension. Laboratory findings included elevated amylase and leukocytosis. Many of the patients were jaundiced. Some pseudocysts in the region of the head of the pancreas were associated with a raised serum alkaline-phosphatase, but a normal serum bilirubin. Roentgenographic studies demonstrated a mass in the region of

the pancreas in only 75 percent of the cases. In three patients, the mass was detected only on an intravenous pyelogram. Twenty-five percent of the patients had a unilateral or bilateral pleural effusion with or without an atelectasis or pneumonia in the lower lung fields. Five patients had an elevated left diaphragm as the only abnormality in the chest roentgenograms. Abdominal roentgenograms showed pancreatic calcification in 13 patients and each of these patients had raised serum-amylase levels.

Forty of the pseudocyst patients showed spontaneous regression of the pseudocysts or were associated with one or more complications, including rupture, hemorrhage or ascites. Ten patients showed spontaneous regression. Three pseudocysts drained into the duodenum, duodenum and common bowel duct, and colon, respectively. Ruptured pseudocysts were found in 19 patients. There was an acute rupture into a variety of adjacent organs and spaces. The authors found massive hemorrhage associated with the pseudocysts as an unpredictable, serious complication, which has a high mortality. In fact, bleeding was the prime cause of mortality in this series. Pancreatic ascites was seen in 18 patients. Pancreatic ascites was diagnosed by elevated serum-amylase, elevated ascitic fluid amylase, and elevated ascitic protein. Seventy-nine patients in this series showed no preoperative complications.

In the authors' experience spontaneous regression of pseudocysts does occur in approximately eight percent of patients, but because of the unpredictability and the severity of the potential complications, surgical therapy is strongly suggested. The timing of the surgical procedure, in this disease, is still unclear since the immature phase of the pseudocyst is associated with the most delicate type of lining. The authors prefer to operate on mature pseudocysts; consequently patients with fresh pseudocysts remain in the hospital for two to four weeks before an operation.

The authors mention recent reports of a high degree of multiplicity in pseudocyst pancreas and suggest careful roentgenological work-up with pancreatography to exclude small additional lesions. Their most satisfactory surgical results were obtained by cystoduodenostomy. For lesions of the body and the tail of the pancreas, anastomosis to the stomach or jejunum has been performed.—*Daniel P. Link, M.D.*

Brewster, David C., Retana, Alvaro, Waltman, Arthur C., and Darling, R. Clement. Angiography in the management of aneurysms of the abdominal aorta: its value and safety. *New England J. Med.*, April, 1975, *292*, 822–825. (Address: R. Clement Darling, M.D., 3 Hawthorne Place, Boston, Mass. 02114.)

One hundred and ninety patients with the clinical diagnosis of abdominal aortic aneurysm underwent preoperative angiography. Each patient had a pulsatile abdominal mass that was considered to be an aortic aneurysm. The patients were usually examined with retrograde femoral angiography, unless there were decreased femoral pulses, and the axillary approach was utilized. Ninety-three percent of the patients were examined with the retrograde femoral approach with mid-stream catheter aortography.

Of the 190 patients with the clinical diagnosis of aneurysm of the abdominal aorta, eleven percent did not have an aneurysm and the pulsatile mass represented either a tortuous aorta or other intra-abdominal masses. In four patients, the pulsatile mass was a pancreatic lesion.

Forty-one percent of the patients had associated aneurysmal disease of the iliac vessels and 15 percent had aneurysms of other pelvic vessels. Other vascular abnormalities included: stenotic lesions of the renal or superior mesenteric arteries, or both; multiple renal arteries; and vascular lesions in the lower extremities and in the aortocranial vessels.

Although major complications did not occur in the series presented, possible complications include aneurysm perforation, dislodgement of mural thrombus, iatrogenic dissection of a diseased vessel, etc.

Eighty-three percent of patients in the series showed curvilinear calcification on the lateral lumbar spine roentgenograms and this is one of the most reliable routine roentgenographic diagnostic findings. When these calcifications were present, there was a definite aortic aneurysm in 95 percent of the presented patients. Ultrasonography has also been used in diagnosing abdominal aortic aneurysms.

Thus, aortic angiography is most valuable in delineating the anatomic extent of the aneurysmal disease prior to operation rather than aiding in diagnosis. Actually, the decision as to whether the aneurysm in any particular patient is operable, may depend on the angiographic findings. Also, angiography is helpful in evaluating the extent of surgery necessary, including possibly renal artery grafting if the stenotic lesions of the renal arteries were determined to be hemodynamically important.

The definite indications for preoperative angiography in patients with clinically suspected aneurysm of the abdominal aorta include the noncalcified aneurysm (indicating a possible nonaneurysmal mass with transmitted pulsations, mycotic aneurysm, etc.), an aneurysm extending to the xyphoid process on physical examination or a widened thoracic aorta, the presence of hypertension suggesting the possibility of renal vascular disease, symptoms suggesting intestinal ischemia, and patients with coexisting aortoiliac or femoropopliteal occlusive disease or aneurysm.—*Ronald J. Ross, M.D.*

Lamarque, J.-L., Senac, J.-P., Bruel, J.-.M.,

de Sars, P., and Boulet, P. L'hépatographie à l'huile iodée émulsionnée par voie artérielle. (Hepatography by intra-arterially injected iodized oil emulsion.) *Presse méd.*, Jan., 1975, *4*, 263–267. (Address: Pr. J.-L. Lamarque, Service Central de radiodiagnostic clinique, Saint-Eloi, F 34059 Montpellier Cedex, France.)

Based upon 250 cases, hepatography obtained by intra-arterial injection of 150 cc of fat-soluble emulsion of an iodized compound (A.G. 52-315) following regular selective arteriography (celiac trunk or superior mesenteric) gave excellent hepatographic images in 10 percent of cases. In 49 percent the images were good and in 34 percent fair. Only in 7 percent did hepatography fail and this was because of severe liver damage. In none of the cases injected was there any serious complications, in particular, no pulmonary embolus ensued. Tomographic studies of the liver were made from 2 to 5 hours after injection, and demonstrated parenchymal lesions as small as 2 mm. The study significantly contributes to topographic pathological anatomy as well as physiology of the liver.—*Jirair N. Sarian, M.D.*

Bücheler, E., Thelen, M., Schirmer, G., Schulz, D., Frommhold, H., Siedeck, M., and Käufer, C. Katheterembolisation der Milzarterien zum Stopp der akuten Varizenblutung. (Catheter embolization of the splenic artery as treatment for acute bleeding from varices.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, March, 1975, *122*, 224–229. (Address: Prof. Dr. E. Bücheler, Radiologische Klinik, Abteilung Röntgendiagnostik, 2 Hamburg 20, Martinistrasse 52, Germany.)

Embolization of the splenic artery with fibrin foam, in order to stop acute bleeding from varices in the esophagus and stomach, was carried out in two patients. This procedure was performed immediately after demonstration of the varices by means of catheterization and selective injection into the splenic artery and superior mesenteric artery. The fibrin foam was injected slowly to prevent escape of the material into other branches of the celiac axis, and after the procedure an angiogram was made for control.

By this method it is possible to achieve temporary cessation of bleeding from the varices. The procedure is indicated in desperate situations, as when intubation has been unsuccessful in control or has to be discontinued, when endoscopic treatment is contraindicated, or when the liver function or other medical considerations prevent emergency construction of a shunt.

Permanent results cannot be expected and recurrence of bleeding will occur. However the use of splenic embolization produces an interval without bleeding during which liver function can be improved so that a decompressing shunt operation becomes possible for definitive management.—*Modified Published English Abstract.*

Hermanutz, K. D., Bücheler, E., and Klais, E. Möglichkeiten und Grenzen der Kavographie in der Tumordiagnostik. (The scope and limitations of cavography in tumor diagnosis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, March, 1975, *122*, 230–237. (Address: Dr. K. D. Hermanutz, Radiologische Universitätsklinik, 53 Bonn, Venusberg, Germany.)

A study was carried out to determine the accuracy of contrast studies of the inferior vena cava in localizing retroperitoneal masses, to determine their extent, to determine whether or not it is possible to classify the type of tumor present, and to determine whether or not the presence of thrombi can be regarded as a sign of invasion of the vena cava by a malignant tumor. Of a total of 500 examinations, 144 were selected and in these 144 diagnosis had been confirmed surgically, histologically, at autopsy, or by the clinical course.

Several types of abnormality of the roentgen appearance were considered and the value of each was examined statistically. The types of abnormality are: ill-defined impression on the vena cava with distortion of the contour of the vessel; a well-defined sharp impression; the presence and type of collateral vessels; the location and degree of displacement of the vena cava; occlusion of the vessel; the presence of filling defects; and obstruction of retroperitoneal veins.

Most of these abnormalities were not valuable for differentiating between a benign and malignant tumor. One sign was found to be fairly reliable in this differentiation. The ill-defined impression on the border of the vena cava with breaks in the continuity of the vascular margin indicated a malignant process, while the other abnormalities were nonspecific.—*Modified Published English Abstract.*

Didolkar, M. S., Malhotra, Y., Holyoke, E. D., and Elias, E. G. Cystadenoma of the pancreas. *Surg., Gynec. & Obst.*, June, 1975, *140*, 925–928. (From: Department of General Surgery, Roswell Park Memorial Institute, New York State Department of Health, Buffalo, N. Y.)

Most pancreatic cysts are either pseudocysts or retention cysts. Cystadenoma and cystadenocarcinoma may also present as cystic swellings arising from the pancreas. If not recognized as such, opportunities for cure may be missed. Cystadenoma repre-

sents four to ten percent of all pancreatic cystic swellings. Roentgenographically, upper and lower gastrointestinal series can aid in demonstrating the size of the lesion, and celiac and superior mesenteric angiograms are particularly helpful because of the vascularity of these tumors. Most are within the body or tail of the pancreas. They have no connection with the pancreatic duct or its branches, and they are multiloculated so that surgical drainage only complicates the subsequent removal.

In this series of five patients, who were predominantly middle-aged women, the presenting symptoms were pain and an epigastric mass without history of pancreatitis or abdominal trauma. Although the tumor is benign, it is known to have association with other malignant lesions as well as multicystic tumors of the liver and kidneys.—*Arch H. Hall, M.D.*

van Heerden, Jonathan A., and ReMine, William H. Pseudocysts of the pancreas: review of 71 cases. *A.M.A. Arch. Surg.*, May, 1975, *110*, 500–505. (Address: Jonathan A. van Heerden, M.B., Mayo Clinic, Rochester, Minn. 55901.)

Cystic lesions of the pancreas are somewhat uncommon but pseudocysts are becoming more common, probably as a result of trauma sustained in automobile accidents. Pseudocysts may form from extravasation of blood, from tissue juices, or from repeated inflammatory reactions in and about the pancreas. The tumor masses may occur in any part of the pancreas and a mass developing in the left upper quadrant of the abdomen after trauma should make one suspect a pseudocyst. Symptoms of pancreatitis are commonly noted and these include dull aching pain in the upper part of the abdomen with anorexia and weight loss and fever. Calcification may be noted in the pancreatic area and jaundice is present in about ten percent of patients where the lesions involve the head of the pancreas.

During the period of 1962 to 1972, 71 patients underwent surgical treatment for pancreatic pseudocyst; internal drainage was performed in 73 percent of these patients as compared to only 20 percent in the series during the previous ten years. During their analysis the authors found that the results during the past decade were superior to those in the previous decade. The treatment of choice is internal drainage either into the stomach or jejunum.

The article is supplemented with several tables and numerous illustrations. Ultrasound, retrograde pancreatography, and angiography are used in the diagnosis of these lesions.

Comment includes a discussion of the many debatable surgical problems concerning the treatment of these lesions. These comments should certainly be a must for the surgeon interested in managing pseudocysts of the pancreas.—*Richard E. Kinzer, M.D.*

Ameriks, John A., Thompson, Norman W., Frey, Charles F., Appelman, Henry D., and Walter, Joseph F. Hepatic cell adenomas, spontaneous liver rupture, and oral contraceptives. *A.M.A. Arch. Surg.*, May, 1975, *110*, 548–557. (Address: Norman W. Thompson, M.D., Outpatient Building, Room C-7554, University of Michigan Medical Center, Ann Arbor, Mich. 48104.)

Hepatic cell adenomas have been reported with increasing frequency during the past decade and spontaneous rupture seems to be a more frequent complication of this tumor. From the University of Michigan Medical Center in Ann Arbor, Michigan comes this report on eight patients with hepatic cell adenomas. They were all women between the ages of twenty-five and forty-four and all had been taking commonly used oral contraceptive agents containing progestogen and estrogen. The authors conclude that the increased incidence of hepatic cell adenomas may well be related to the use of oral contraceptive agents. Three of the patients described developed symptoms from hemorrhage into the peritoneum, two patients had upper abdominal pain from intrahepatic hemorrhage, and palpable tumors were the primary discovery in three asymptomatic patients.

On the basis of their studies, the authors do not believe that liver scans are dependable. They believe that the most valuable roentgenographic examination is hepatic angiography. Several accompanying illustrations demonstrate the detected tumors. Arteriograms were obtained preoperatively in five of their eight patients. The main hepatic artery was normally greatly enlarged and each patient studied had a solitary hypervascular lesion. The angiographic appearance might be useful in distinguishing these tumors from malignant hepatic lesions.—*Richard E. Kinzer, M.D.*

Bernades, P., Callet, B., and Dupuy, R. Étude clinique et évolutive de 102 cas de pancréatite chronique. (Clinical and progressive study of 102 cases of chronic pancreatitis.) *Presse méd.*, Jan., 1975, *3*, 149–152. (Address: Dr. P. Bernades, Centre de Gastroentérologie, Hôpital Beaujon, 100, boulevard du Général-Leclerc, F 92110 Clichy, France.)

The authors used strict criteria in confirming the diagnosis of chronic pancreatitis in the 102 cases presented. Thus, 72 cases had roentgen or surgical evidence, or both, of pancreatic calcifications. Eighty-one patients were chronic alcoholics. In seven patients chronic pancreatitis developed sometime after surgically verified pancreatitis.

Steatorrhea was more prevalent in patients showing pancreatic calcifications. Biliary lithiasis was noted in 17 percent, alcoholic cirrhosis in 17 percent

and gastric or duodenal ulcers in 24 percent.

Sixty-two patients had to be operated on because of complications or progression of the chronic pancreatitis (such as development of acute pancreatitis and pseudocysts of the pancreas, obstructive jaundice, portal hypertension, or cholelithiasis). During a two year period, 70 patients were followed, and of these, 60 percent showed regressive signs. This percentage is the same for those who were operated upon, or treated medically only. In all the cases, abstention from alcohol was found indispensible for good therapeutic results.—*Jirair N. Sarian, M.D.*

Genitourinary System

Raskin, Michael M., Poole, Darrell O., Roen, Sheldon A., and Viamonte, Manuel, Jr. Percutaneous management of renal cysts: results of a four-year study. *Radiology*, June, 1975, *115*, 551–554. (Address: Manuel Viamonte, Jr., M.D., Department of Radiology, Mount Sinai Medical Center, Miami Beach, Fla. 33140.)

The therapeutic management of renal cysts by percutaneous methods was studied comparing Pantopaque instillation and Renografin with air instillation following partial aspiration of cyst fluid. Fifty-six cysts were instilled with Pantopaque and followed at varying intervals over a four year period. Fifteen patients had only Renografin and air injected. The Renografin injected cysts demonstrated random changes in size with most either increasing or remaining the same in size. Using statistical evaluation of the data from the Pantopaque injected cysts, a cyst so injected has a 61 percent probability of being reduced in volume by at least 50 percent within 30 months. The findings thus suggest that Pantopaque alters the natural history of a renal cyst.—*R. F. Kuhnhein, M.D.*

Singh, Manmeet, Marshall, Villis, and Blandy, John. The residual renal stone. *Brit. J. Urol.*, April, 1975, *47*, 125–129. (From: The London Hospital, London, England.)

It has often been said that following removal of a staghorn renal calculus any residual tiny fragments will lead to eventual recurrence of the calculus. For this reason preoperative contact renal films are often made in an attempt to exclude any remaining calcareous particles. In this series of more than 120 pyelolithotomies for staghorn calculi, it was found that recurrence of stone is not inevitable when a fragment is left behind and complete removal does not guarantee that a stone will not recur.

Through a controlled cadaver kidney study the authors have shown that calculi fragments 1×1 mm are usually missed on the Kodak contact film under ideal conditions by the most experienced observers. Even larger fragments are likely to be missed under less favorable operating conditions. However, the residual stones do not always grow but often pass spontaneously or remain unchanged. While it is advisable to take every reasonable precaution to remove calculi as completely as possible, the operative contact renal film would seem of questionable value if it unduly prolongs the operative procedure.—*Arch H. Hall, M.D.*

Foley, W. Dennis, Bookstein, Joseph J., Tweist, Melvin, Gikas, Paul W., Mayor, Gilbert H., and Turcotte, Jeremiah G. Arteriography of renal transplants. *Radiology*, Aug., 1975, *116*, 271–277. (Address: W. Dennis Foley, M.D., Department of Radiology, University of Michigan Medical Center, Ann Arbor, Mich. 48104.)

Sixty arteriograms were reviewed in patients who underwent arteriography for postrenal transplantation dysfunction. Thirty-four studies provided technically adequate demonstration of the cortical angiogram, and these were analyzed to determine the discriminatory value of angiography.

Arteriography was performed by retrograde percutaneous femoral catheterization on the side containing the transplant. The hypogastric artery was selectively engaged in all but two cases and direct 2× magnification was used in all but the first six cases. Three × magnification was used also in the final 14 cases.

Angiograms were analyzed independently by two observers without knowledge of the clinical data. Three major observations were made in each case: (a) estimated renal blood flow; (b) the appearance of the interlobar arteries; and (c) analysis of the magnification renal cortical microangiogram.

Angiography permitted recognition of common causes of post-transplantation dysfunction including acute vasomotor nephropathy, acute rejection, chronic rejection, and obstruction of the ureter, renal artery or renal vein. In addition to its diagnostic value, angiography provided some prognostic information in acute rejection. Irreversible rejection could be predicted on the basis of total absence of outer cortical opacification, indicating cortical necrosis.—*Jon F. Bergstrom, M.D.*

Nervous System

Huckman, Michael S., Fox, Jacob, and Topel, Jordan. The validity of criteria for the evaluation of cerebral atrophy by computed tomography. *Radiology*, July, 1975, *116*, 85–92. (Address: Michael S. Huckman, M.D., Department of Diagnostic Radiology, Presbyterian-St. Luke's Hospital, Chicago, Ill. 60612.)

Two groups of patients over the age of 60 were studied for the presence or absence of cerebral atrophy by computed tomography. The groups consisted of demented and nondemented people. Each patient in one group (35 patients) had a clinical diagnosis of senile dementia and had been examined by a neurologist. The other group (48 patients) consisted of all patients in a series of 300 consecutive computed tomograms.

Criteria for evaluation of cortical atrophy are discussed. These included subjective criteria and more objective evaluation by measuring ventricular size and width of cortical sulci. The findings were similar using either method. Previous studies of cortical atrophy in elderly people as determined by pathologic and pneumoencephalographic methods are compared to the computed tomographic findings. There was close correlation.

Most of the demented patients showed evidence of atrophy and most of the other groups did not show evidence of atrophy. Of particular interest is that two of the demented patients with no computed tomographic evidence of atrophy were subsequently discovered to have treatable illnesses. There was resolution of dementia following appropriate therapy. Another was discovered to have low pressure hydrocephalus. The authors conclude that careful investigation for a treatable illness should be done in those demented patients without demonstrable evidence of cerebral atrophy.—*A. K. Teaford, M.D.*

Gabrielsen, Trygve O., Seeger, Joachim F., and Amundsen, Per. Some new angiographic observations in patients with Chiari Type I and II malformations. *Radiology*, June, 1975, *115*, 627–634. (Address: Trygve O. Gabrielsen, M.D., Department of Radiology, University of Michigan Medical Center, Ann Arbor, Mich. 48104.)

The authors describe the angiographic findings in Arnold-Chiari malformation and corroborate the previous descriptions by Greitz and Sjögren (*Acta radiol.* (Diag.), 1963, *1*, 284–297). In Type I, the medulla oblongata is displaced downward into the spinal canal. In Type II, in addition, the inferior vermis, the brainstem and the fourth ventricle are elongated, with the latter extending for varying distances into the spinal canal. The authors present 13 cases of Chiari Type I and II malformations examined by vertebral and carotid angiography. The angiograms presented are good quality subtractions and there is ample labeling of vasculature.

The pertinent findings are caudal displacement and deformity of the tonsillohemispheric branches of the posteroinferior cerebellar artery (PICA), visualization of small tonsillar branches of the PICA below the foramen magnum and an open, stretched caudal loop of the PICA below the foramen magnum. Definite staining of the tonsil below the foramen may sometimes be seen. It may also be possible in some cases to identify an indentation of the brainstem anteriorly and grooving of the malformed cerebellum posteriorly. All of these same findings could be found in association with an expanding intracranial mass lesion; the obvious difference in the clinical picture would help to differentiate the two.—*W. I. Dittman, M.D.*

Skeletal System

Utsunomiya, Joji, and Nakamura, Tadashi. The occult osteomatous changes in the mandible in patients with familial polyposis coli. *Brit. J. Surg.*, Jan., 1975, *62*, 45–51. (From: Second Department of Surgery, Medical School, and Department of Radiology, Dental School, Tokyo Medical and Dental University, Tokyo, Japan.)

The authors have noted a characteristic endosteoma lesion of the jaw, seen best on panoramic views, which they feel is characteristic for patients with familial polyposis coli. All patients with numerous adenomatous polyps distributed throughout most parts of the large intestine, regardless of familial occurrence or the association of extra colic lesions, are considered to have familial polyposis coli (F.P.C.). Prior to the study, the authors noted that jaw abnormalities were frequently seen in patients with Gardner's stigmata, therefore, they decided to carry out routine roentgenological examination of the oral region on all patients with adenomatous polyps in the colon. The authors considered the following three groups:

1. Polypotic members of F.P.C. families
2. Non-polypotic members of F.P.C. families
3. Cases with intestinal polyposis of other types

These three groups included 29 cases, 19 cases, and six cases, respectively. Colons removed from the above patients showed typical adenomatous lesions in 23 patients. Solitary or multiple malignant lesions were found in ten patients. The remaining five patients were none-operated, but showed characteristic roentgenological and endoscopic features.

Dental roentgenograms on these subjects showed characteristic endosteomas in 27 out of 29 cases with F.P.C. (93.2 percent). These lesions were present in only three of 19 members of F.P.C. families who were free of polyps. Other types of polyposis, including Peutz-Jeghers syndrome, did not show these characteristic lesions. The lesions are described by the authors as radiodense with sharp borders, varying in size from three to ten millimeters in diameter. They are mostly round but sometimes show irregular margins. The degree of radiopacity differs between lesions and within a lesion. The density in a lesion is usually equal to the bony cortex. In seven cases, the lesions were solitary and in 19 cases the lesions were multiple. In F.P.C. patients there was a frequent ap-

pearance of impacted teeth. Patients in F.P.C. families without polyposis showed lesions which were smaller and always solitary.

The authors have noted that the presence of these characteristic lesions of the jaw occur in most patients with F.P.C., regardless of the association with Gardner's stigmata. They suggest therefore, that Gardner's syndrome and simple F.P.C. should not be considered as different etiological categories. They further suggest that F.P.C. has the potential to produce a variety of neoplastic lesions other than the typical colotic lesion, *e.g.*, adenomatosis of the stomach and the characteristic jaw lesions.—*Daniel P. Link, M.D.*

Resnick, Donald, Shaul, Stephen R., and Robins, Jon M. Diffuse idiopathic skeletal hyperostosis (DISH): Forestier's disease with extraspinal manifestations. *Radiology*, June, 1975, *115*, 513–524. (Address: Donald Resnick, M.D., Department of Radiology, Veterans Administration Hospital, 3350 La Jolla Village Drive, San Diego, Calif. 92161.)

Forestier's disease or ankylosing hyperostosis of the spine is characterized by ossification of the anterior and right lateral aspects of the vertebral column, particularly in the thoracic region. The authors described the extraspinal manifestations of Forestier's disease in 21 consecutive cases and suggest that diffuse idiopathic skeletal hyperostosis (DISH) is a more appropriate description of the ossifying diathesis.

All 21 patients had abnormalities of the spine. Extraspinal abnormalities are particularly prominent in the following sites:

Pelvis: Osseous "whiskering" at sites of ligament attachments (iliac crest, ischial tuberosity, trochanters), not unlike that occurring in ankylosing spondylitis and ligament ossification, is frequent.

Heel: Spurs on the posterior or inferior surface, or both, of the calcaneus are common.

Foot: Distinctive bony outgrowths occur along the dorsal surface of the talus, dorsal and medial tarsal, navicular and lateral and plantar portions of the cuboid and base of the fifth metatarsal. Large talar beaks may occur.

Elbow: Olecranon spurs are frequent, well defined and occasionally of considerable size.

Other Sites: Scattered osseous outgrowths occur at other sites of ligament attachment in the body, they are particularly striking about the patella and distal tibia and fibula.

Axial symptoms and signs dominate the clinical picture in this disorder. Mid or low back pain and decreased range of motion are prominent and cervical spine osteophytosis may produce dysphagia due to compression of the esophageal lumen. Peripheral musculoskeletal complaints may be the initial clinical manifestation of the disease. These symptoms are "para-articular" with pain, tenderness and decreased range of motion, and surgical removal of osteophytes may be necessary.

The roentgenographic appearance in the peripheral skeleton is frequently distinctive and allows the radiologist to suggest the correct diagnosis, even in the absence of axial roentgenograms.—*J. L. Montgomery, M.D.*

Tredwell, Stephen J., and O'Brien, John P. Avascular necrosis of the proximal end of the dens: a complication of halo-pelvic distraction. *J. Bone & Joint Surg.*, April, 1975, *57-A*, 332–336. (Address: Stephen J. Tredwell, M.D., Division of Orthopaedic Surgery, University of British Columbia, 2740 Heather Street, Vancouver, British Columbia V5Z 3J4, Canada.)

Roentgenographic evidence of avascular necrosis of the upper part of the dens was observed in 13 of 94 patients treated for scoliosis using Halo pelvic distraction. The diagnosis was made on the basis of demonstration of cystic areas, sclerosis and irregularity in contour of the dens. In this group, roentgenographic evidence of degeneration of the zygoepiphyseal joints of the cervical spine was recognized by narrowing of the joint space and cystic erosion of adjacent bone.

Distraction forces were measured and expressed as a percentage of body weight. Distraction forces were expressed in two ratios: ratio one representing the amount of distraction between the axis and the occiput, and ratio two representing distraction of the atlantoaxial joint. The 13 cases of avascular necrosis of the upper part of the dens all occurred in the group that had had relatively high ratios, permitting the determination of figures above which there was a high incidence of avascular necrosis. The blood supply to the dens is principally from the vessels that enter through the apical alar ligaments, and apparently excessive traction on these ligaments may compromise the vessels and contribute to the avascular necrosis. Stretching of the atlantoaxial ligaments may cause instability. In four of the patients in this series, a separation between the anterior arch of the first cervical vertebra and the dens on forward traction was more than 4 mm. Of these patients, three were in the group with avascular necrosis. This latter finding suggests that the distraction should be applied with the neck in neutral position and not in flexion.

In eleven of the 13 patients, clinical and roentgenographic follow-up of from twelve to 30 months was possible and progressive healing of the avascular lesions was observed. The ultimate clinical significance of the necrosis is not known.—*Lois Cowan Collins, M.D.*

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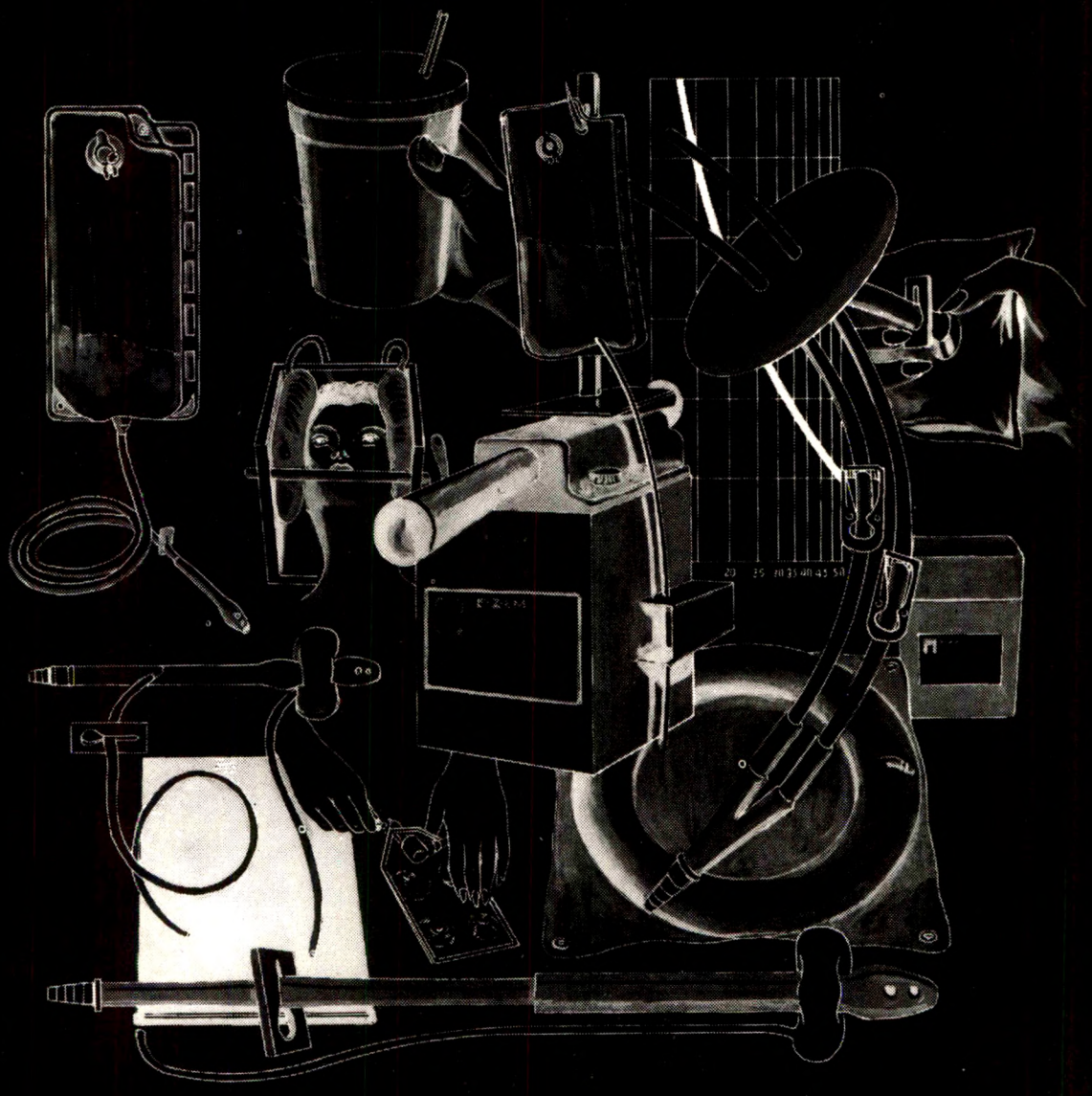

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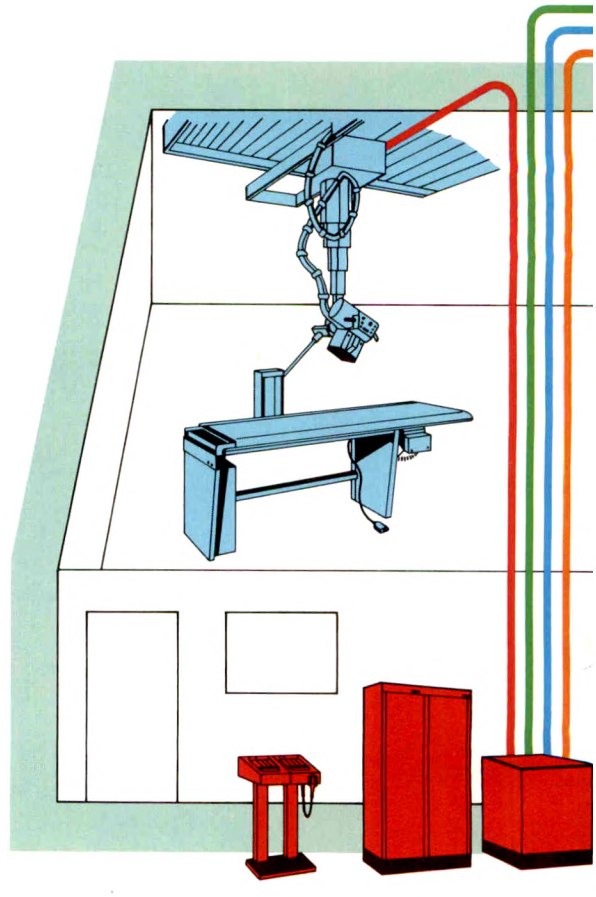

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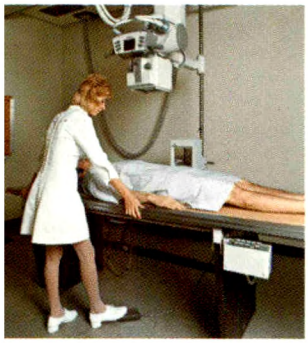

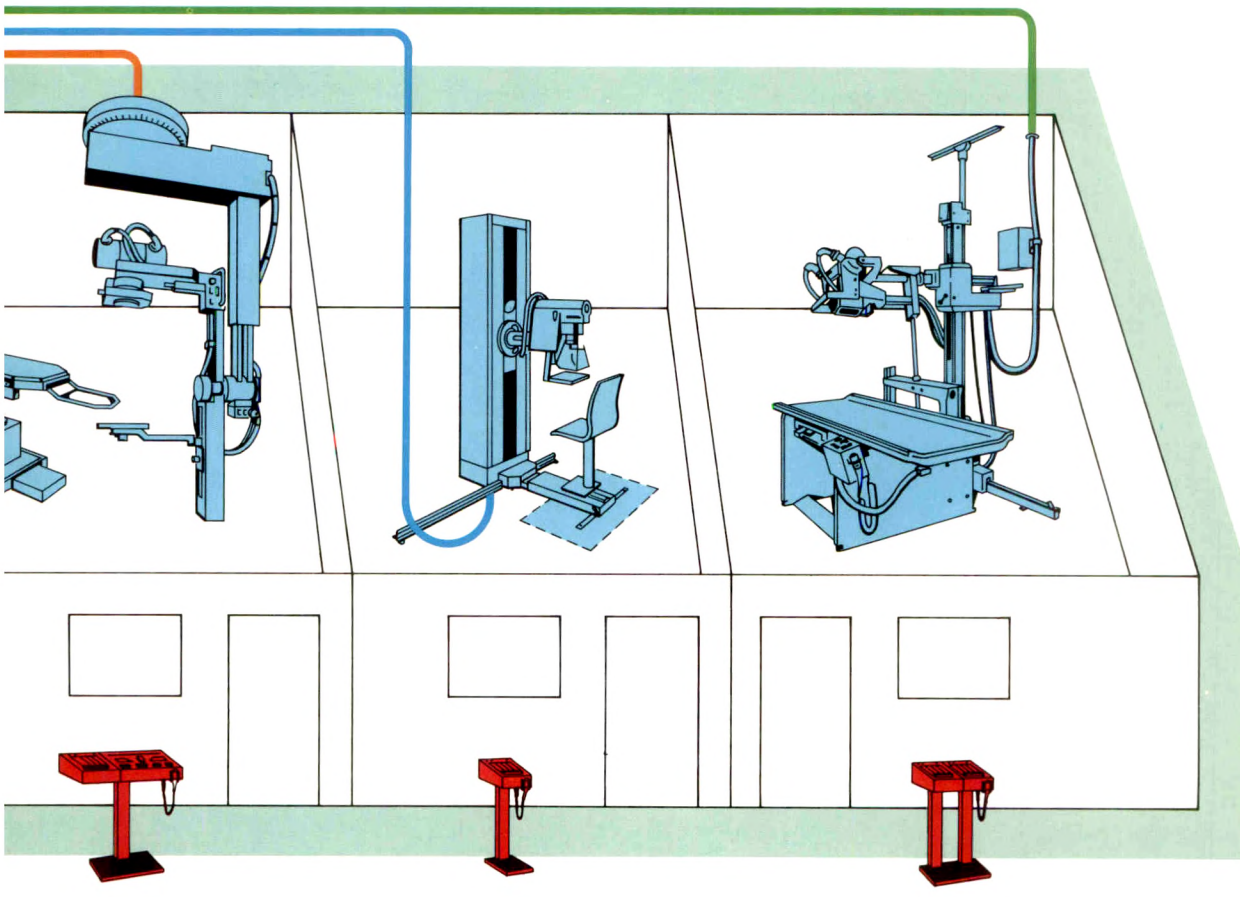

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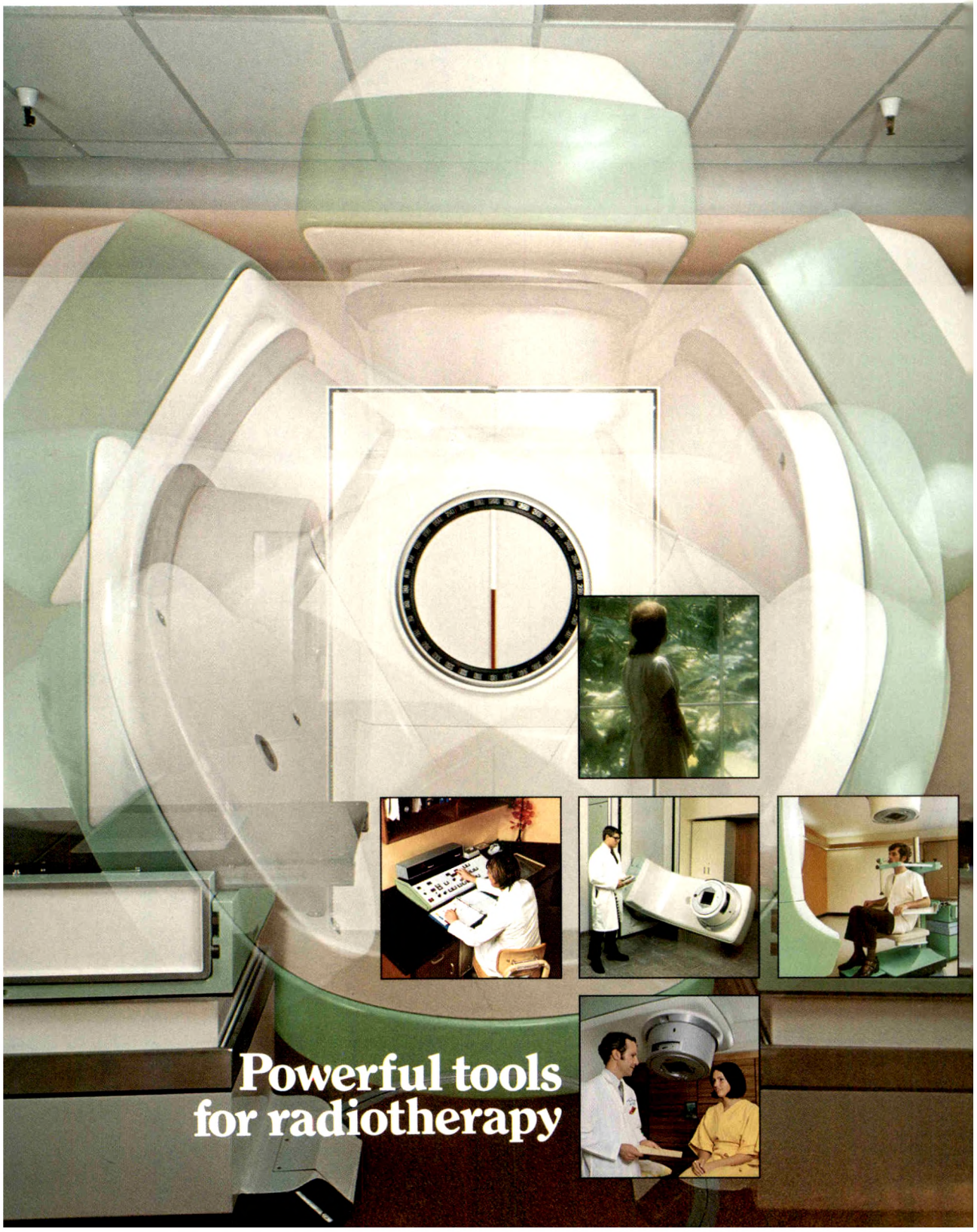

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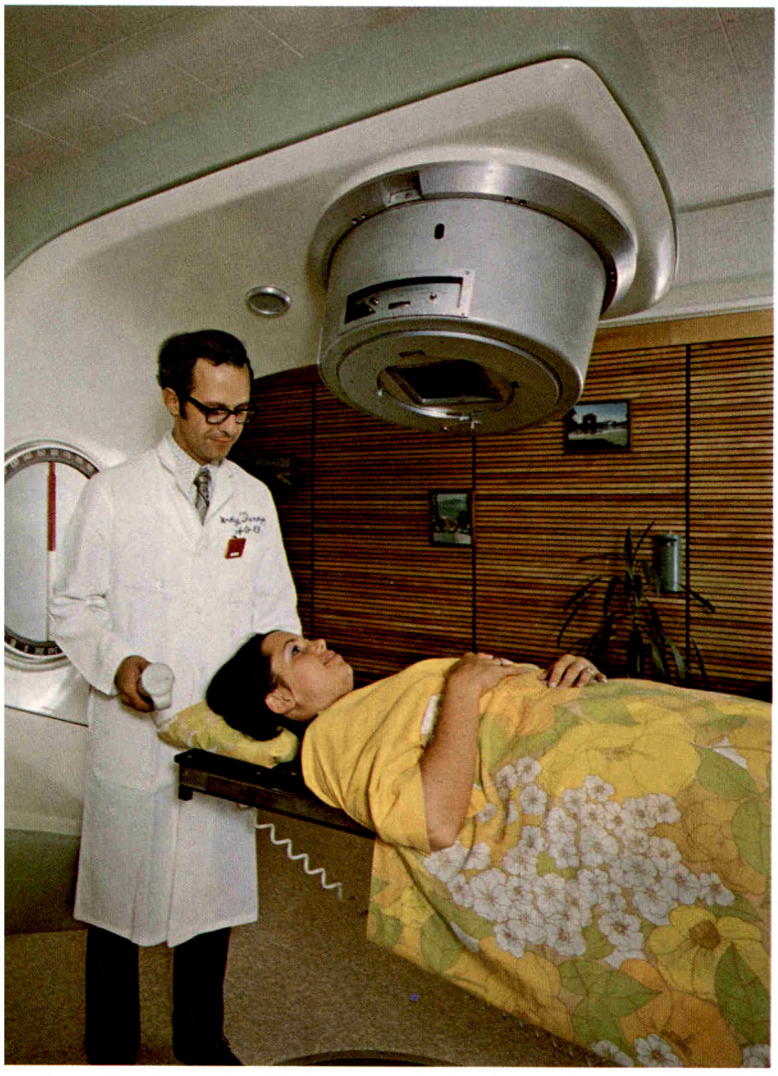

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Clinac 4S

Clinac 4 Performance From a Wall-Mounted Unit

The Clinac 4S has all the desirable Clinac 4 beam characteristics, plus some distinctive features of its own: It provides the largest field sizes routinely achieved by any commercially available medical linear accelerator. Precise set-ups are facilitated by a hand-held pendant, which remotely controls all collimator and gantry motions — and by a wall-mounted digital readout panel that displays the significant x-ray head positions.

Clinac 18

New Keystone for Radiation Therapy Centers

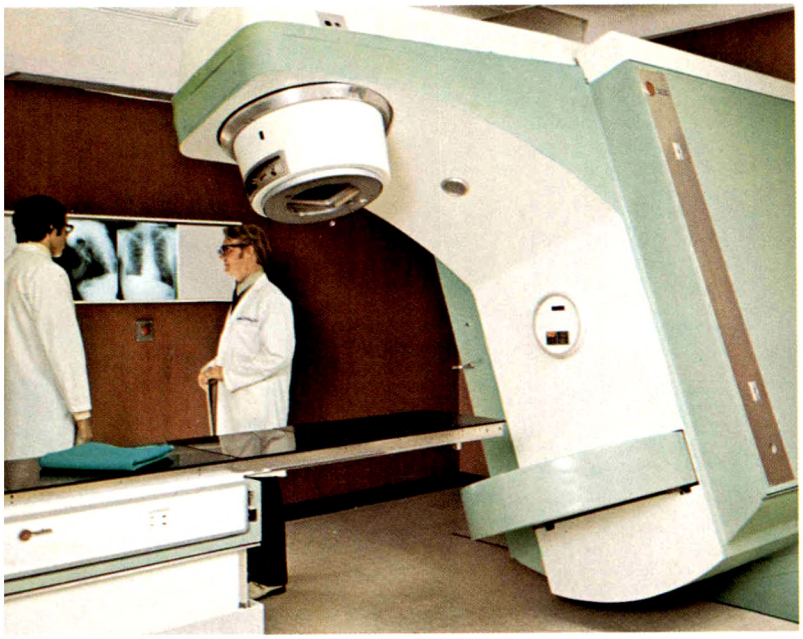
A high energy photon beam at 10 MV and five electron beam energies from 6 to 18 MeV are available from this 100 cm isocentrically mounted machine. A 5 MW klystron tube powers the unit. A fully achromatic beam bending magnet and a gridded electron gun assure stability. Dual dosimetry and independent interrogate circuitry are standard features of this instrument. Because of these clear-cut advantages, the Clinac 18 has been widely accepted as the equipment of choice for progressive therapy centers around the world.

Clinac 12

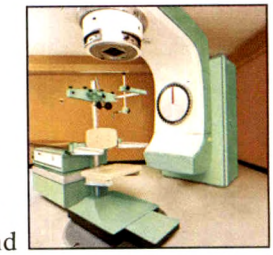
Dual Modality in an Economical Package

Combining the innovative design features of the Clinac 18 with the proven power circuits of the Clinac 4, the Clinac 12 offers dual modality at minimum cost. The machine features an 8 MV photon beam and 6 to 12 MeV electron beams.

Clinac 35
New Opportunities for High Energy Therapy and Research



X-ray energies of 10 and 25 MV, plus electron energies to over 30 MeV, combine to make the Clinac 35 the most powerful medical linear accelerator manufactured today. Dose rates up to 1200 rads/min. at a meter, and a powerful research beam bring new opportunities to the field of radiotherapy and high-power research.



Treatment Chair
Designed for reproducible patient positioning, the treatment chair facilitates arc or multiport therapy in head and neck cases. The chair attaches to Clinac treatment couches for precise motorized control.

Varian's Customer Support Program

Training of Personnel
Varian's week-long training course in Palo Alto teaches equipment operation and much more. Attendees learn to use the Clinac efficiently and to carry out preventive maintenance, trouble shooting and basic repair procedures. Varian's step-by-step operation and maintenance manuals are comprehensive and easy to understand.

World-Wide Service
Varian maintains service offices at strategic points around the world. They are equipped and staffed to meet all routine maintenance and emergency service needs. A member of our service staff is always available to help trouble-shoot a problem on the phone or arrive on the scene within hours.

Users' Meetings
All Clinac owners are invited to attend and participate in periodic Users' Meetings. These seminars, which have attracted as many as 150 therapists and physicists, are a forum for exchanging new data and techniques on the Clinacs.

Come Visit Us.
If you are traveling to California, we invite you to visit us. Our staff will be pleased to discuss and answer any questions you might have about Clinacs. If you cannot come in person, ask our local people to visit you, or write for literature on any or all Clinac linear accelerators.

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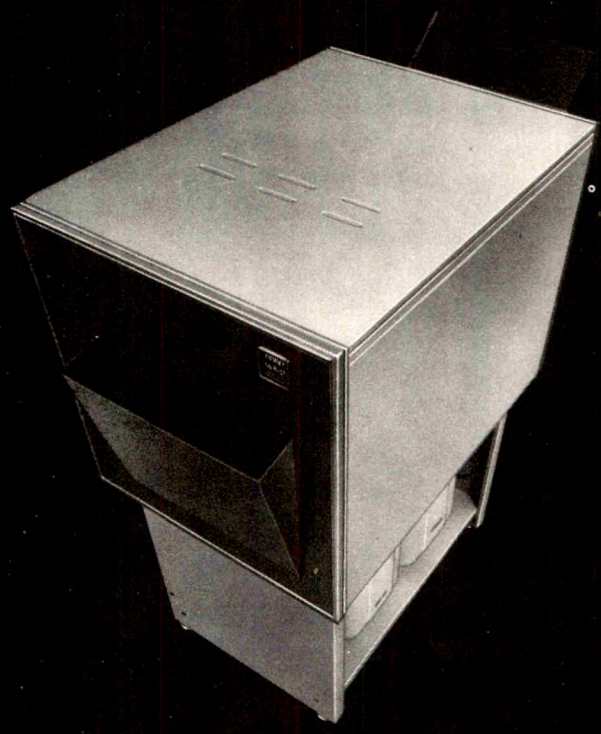

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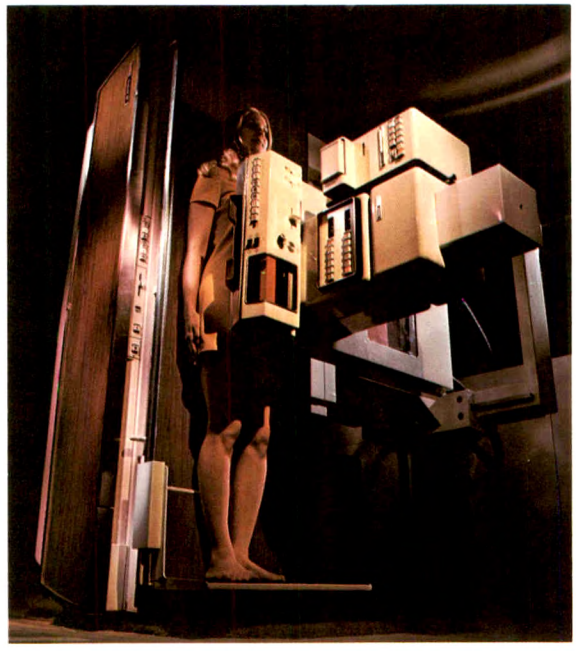

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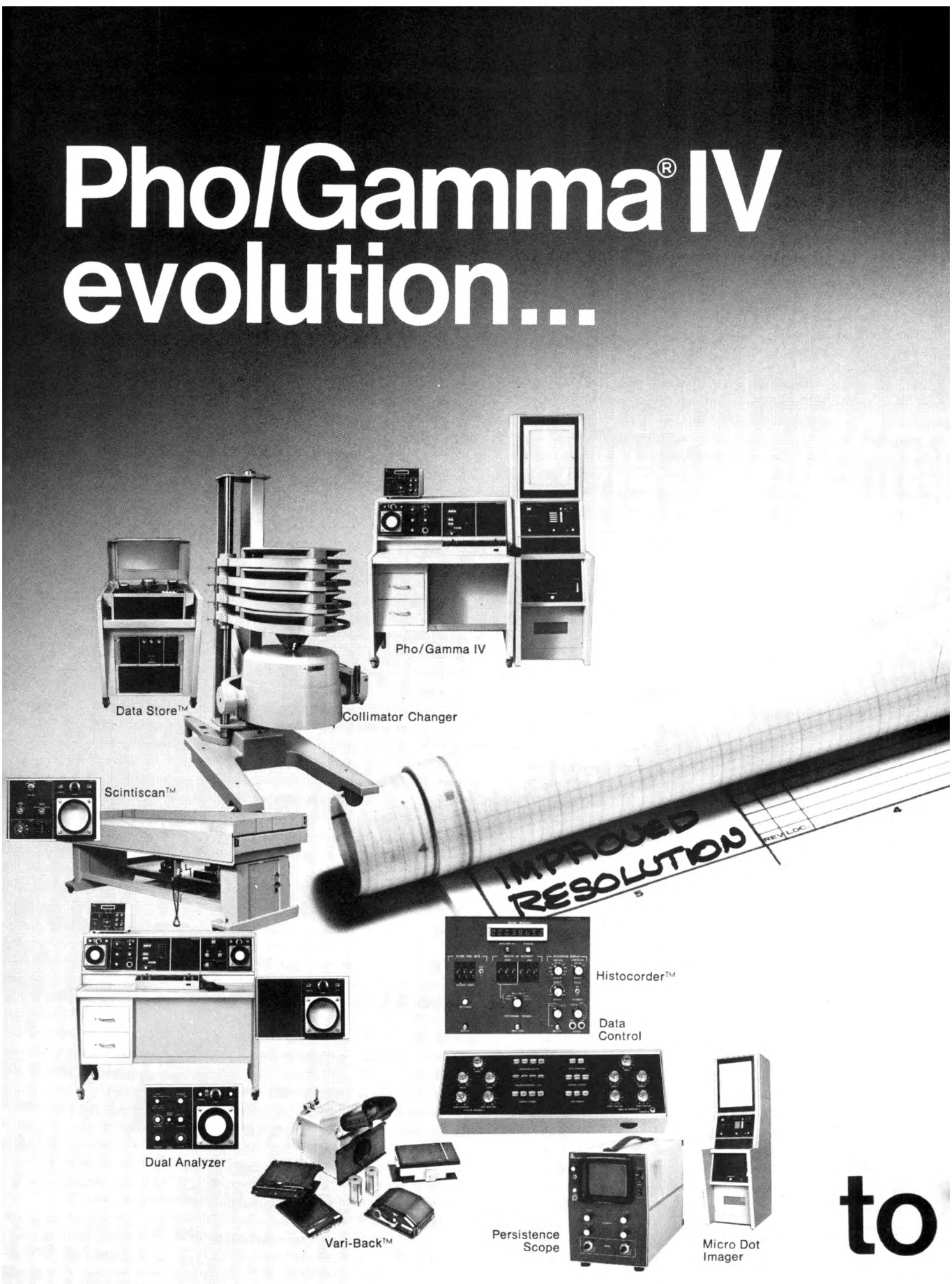

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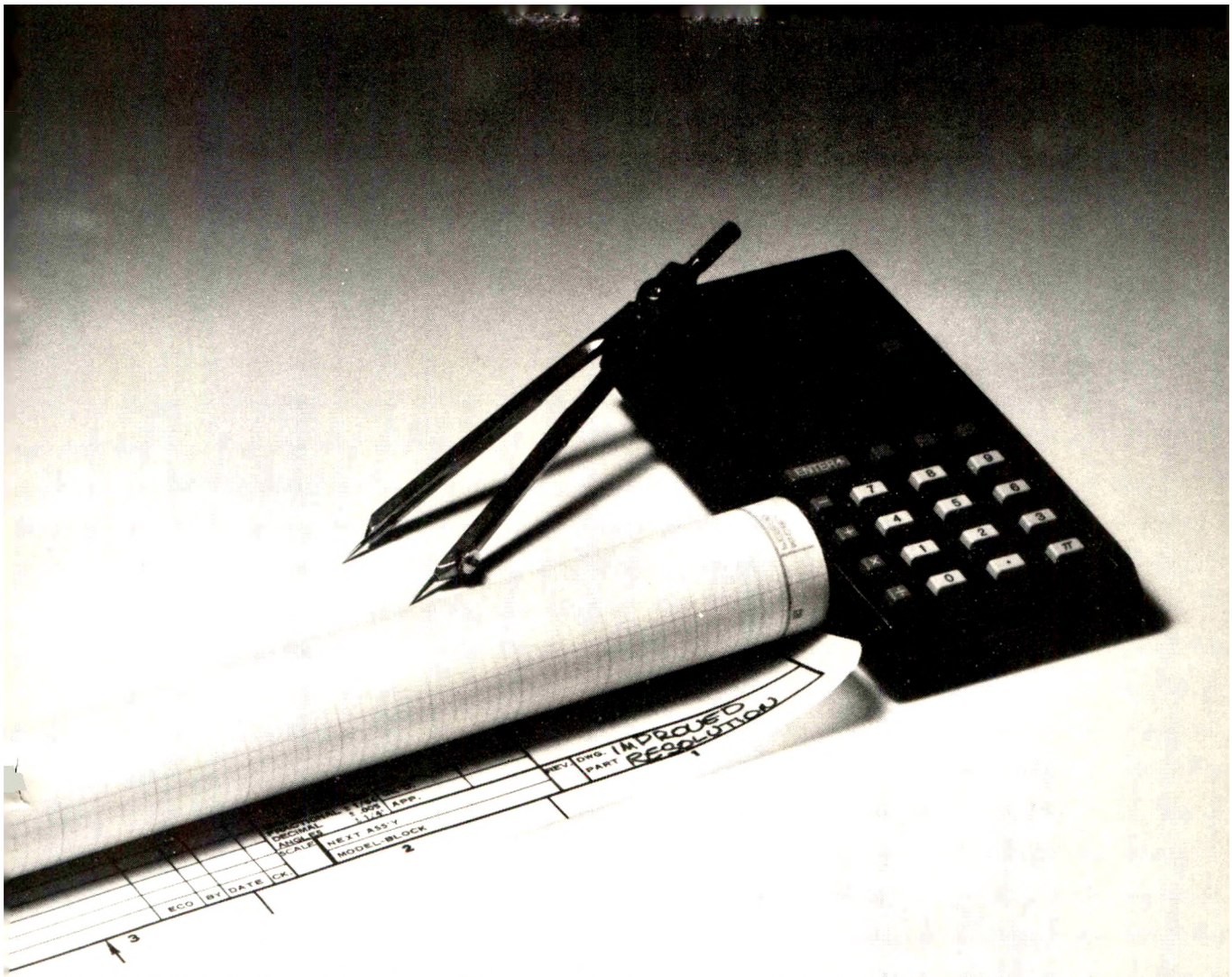

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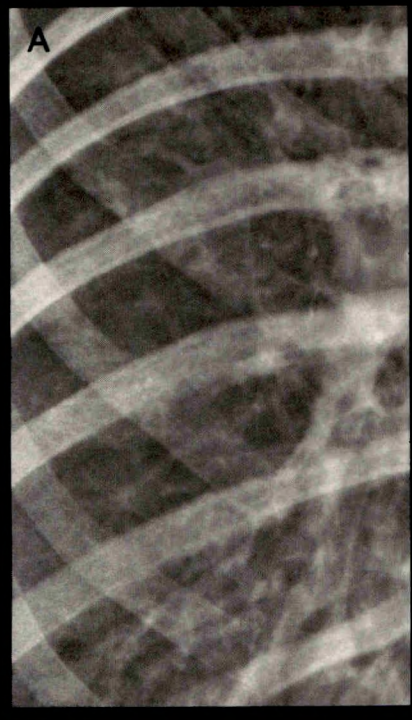
be continued...

The Lungs in Hodgkin's Disease

As a rule, when the lungs are involved in Hodgkin's disease, so are the mediastinal lymph nodes. Of the following examples, all but Case I followed the rule.



CASE I: When first examined, this twenty-year-old female had fever and bilateral cervical adenopathy. At this time, the roentgen study of her chest revealed no abnormalities. Three months later, however, both lungs revealed changes without mediastinal or hilar adenopathy.

FIG. A: A segment of the right hemithorax taken from this patient's original chest examination reveals no abnormalities.

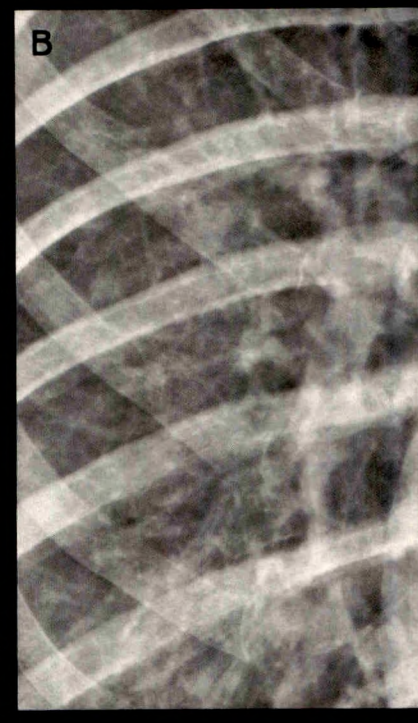
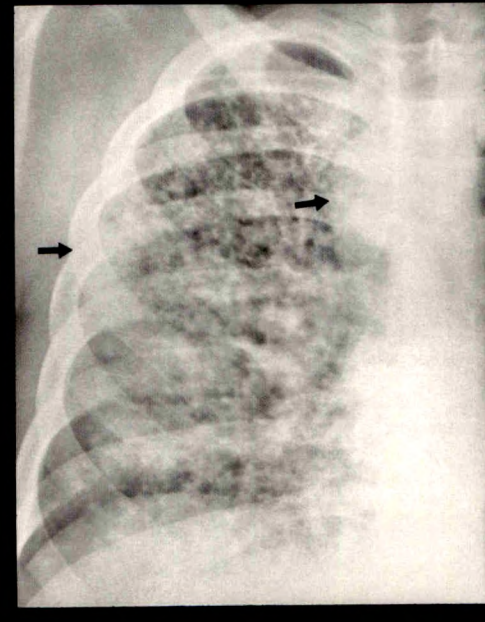


FIG. B: The same segment of the right hemithorax (as demonstrated in Fig. A) three months later. Interstitial and vascular changes are dramatic.

CASE II: A nineteen-year-old male with chills, fever, inguinal adenopathy and weight loss.

It is not difficult to imagine that the infiltrates seen in Case I might have eventually developed a similar degree of parenchymal, mediastinal and pleural involvement (arrows).



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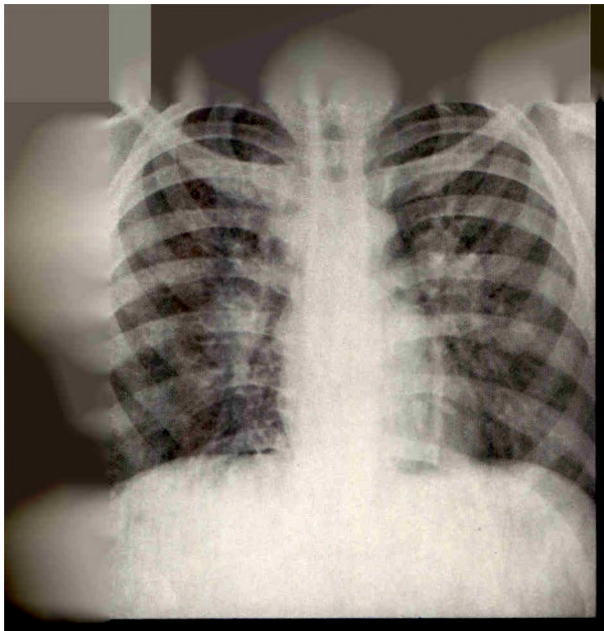
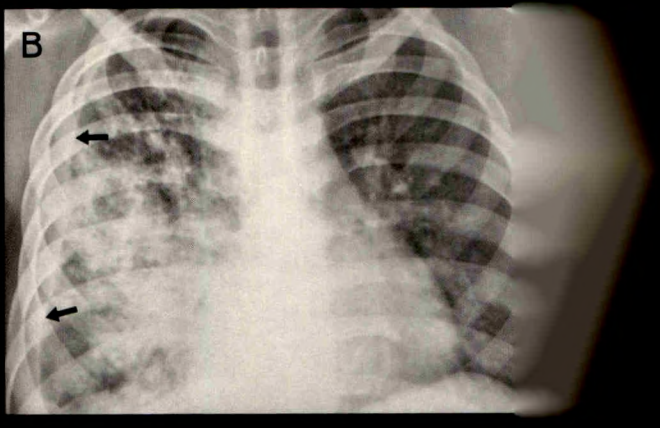
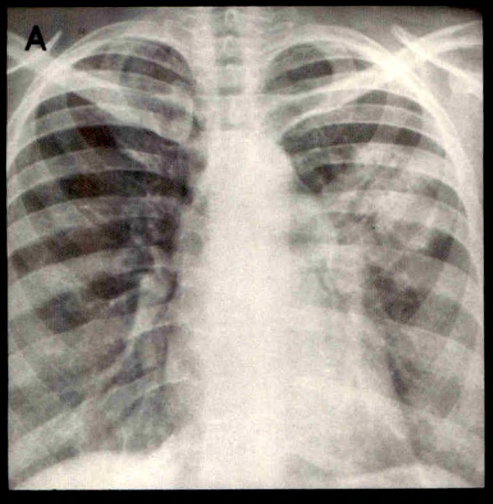
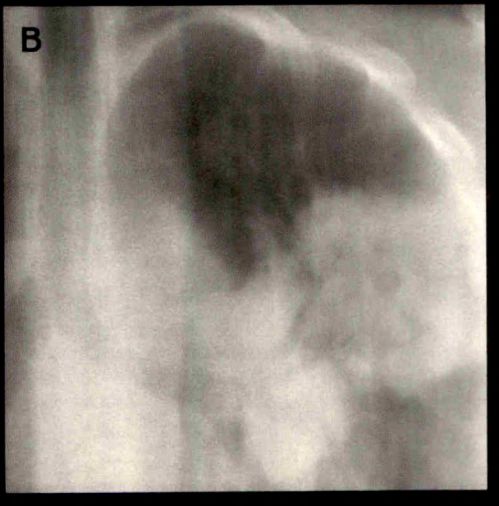


CASE III: A thirteen-year-old boy with fever and wea
ness. The original chest radiograph, Fig. A, revea
mediastinal adenopathy, hilar adenopathy, plus ear
bilateral parenchymal invasion.

Six months later, Fig. B, following radiation therap
and chemotherapy, the right hemithorax reveals p
gressive disease with pleural involvement (arrow
The left hemithorax seems somewhat improved.







IV: A twenty-four-year-old female who suddenly
oped chills, fever, and rusty sputum. Whereas usu-
lung mass associated with hilar and mediastinal
opathy of the type seen in Fig. A suggest broncho-
carcinoma, this patient's age and history favored
e acute process. The shaggy and irregular superior
n of the mass, the bronchial distortion and small
es demonstrated by body section radiography Fig. B,
oteworthy. This proved to be Hodgkin's disease.

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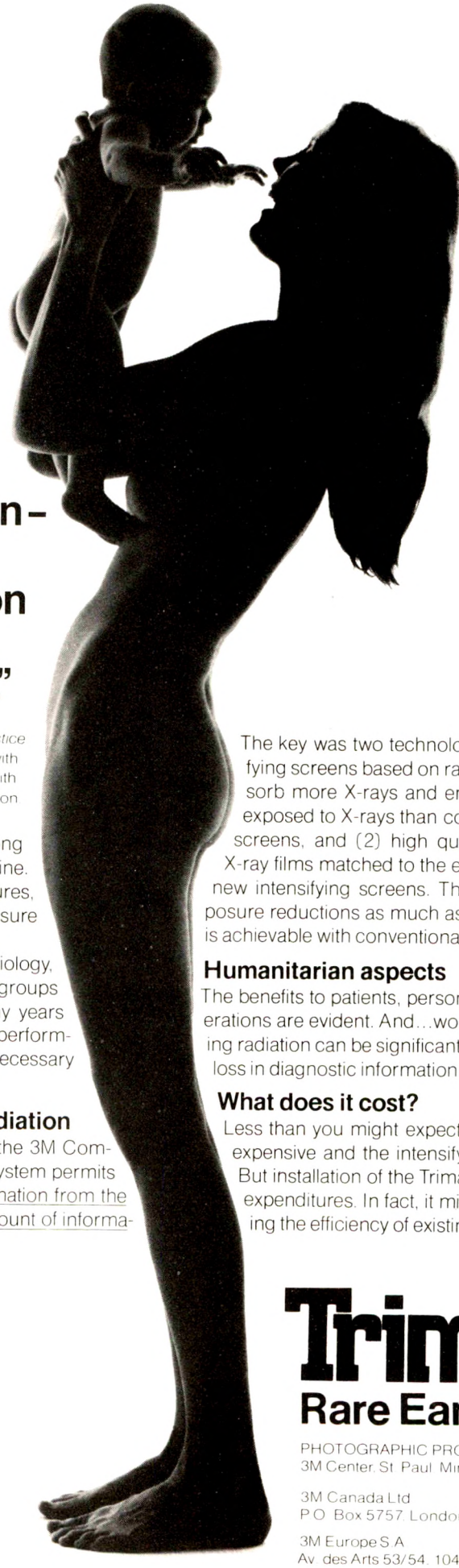

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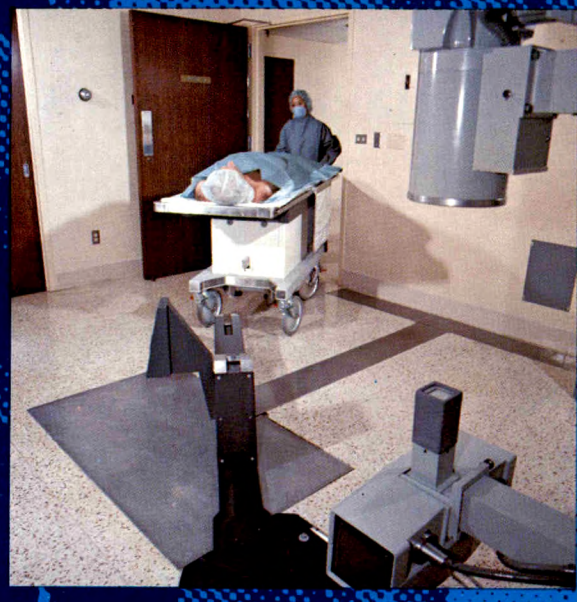

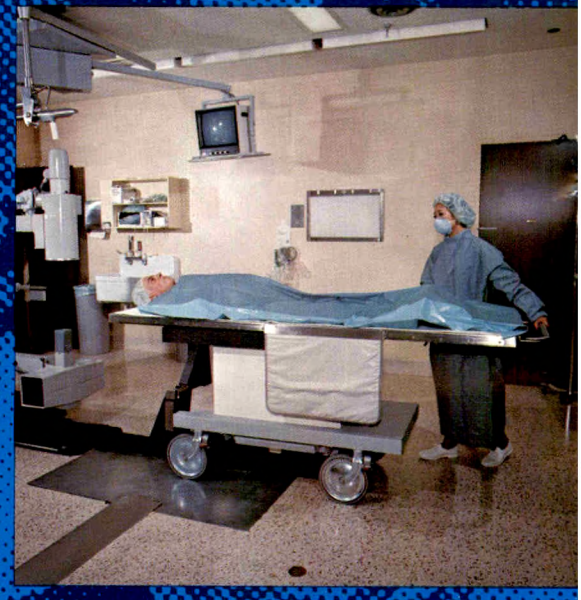

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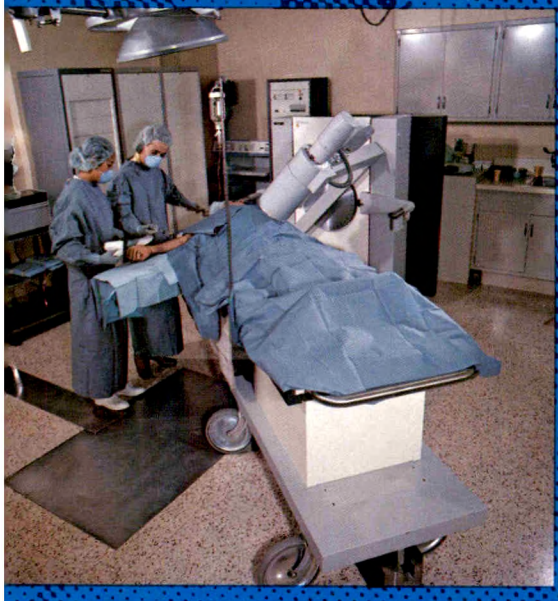

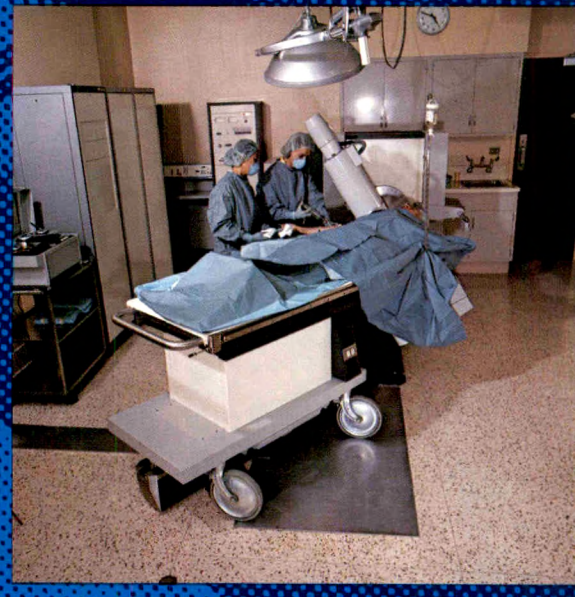

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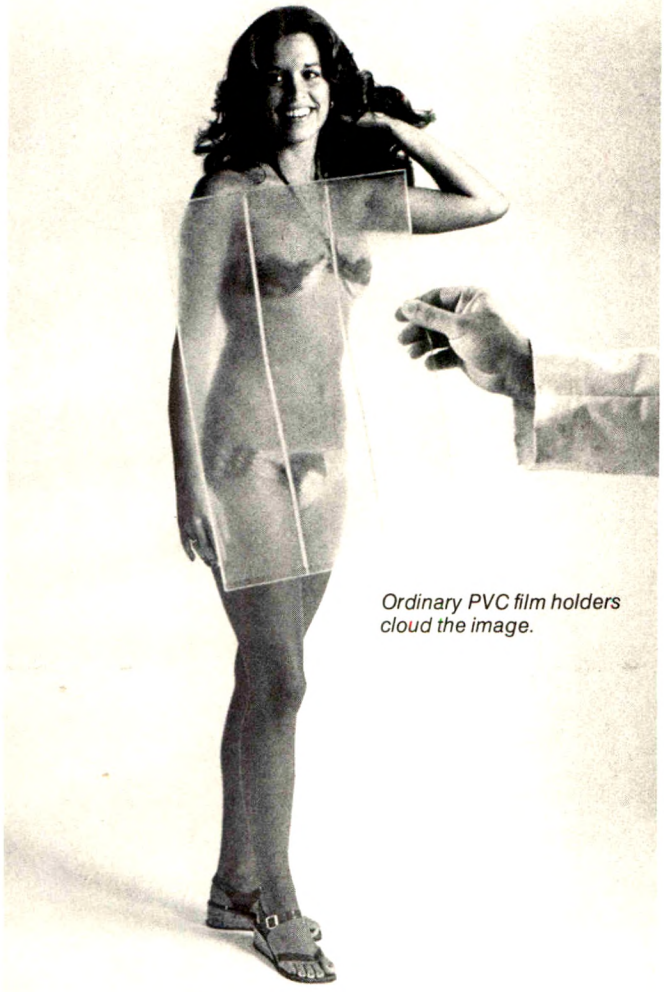

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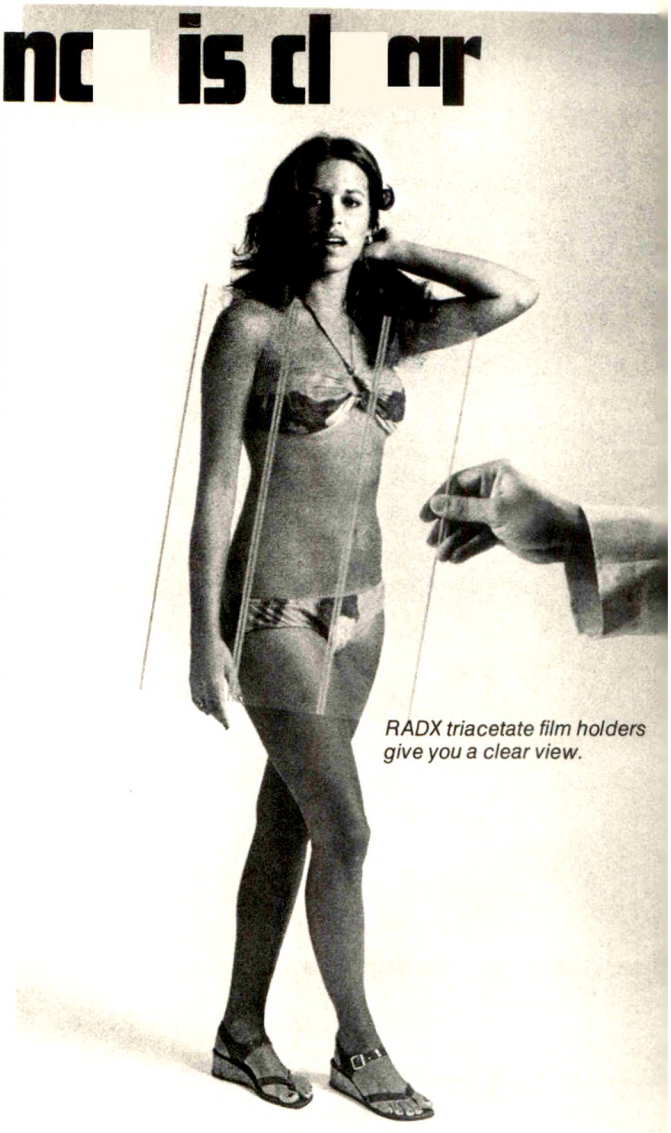

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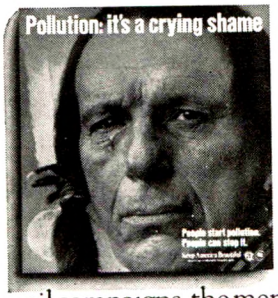

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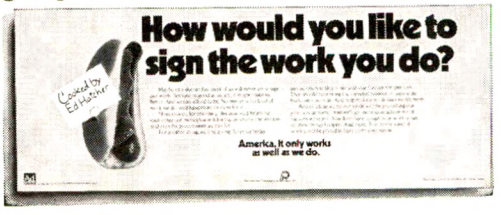

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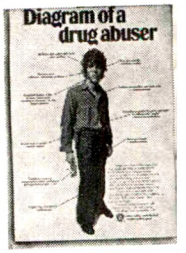

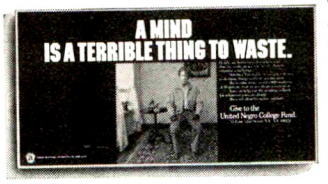

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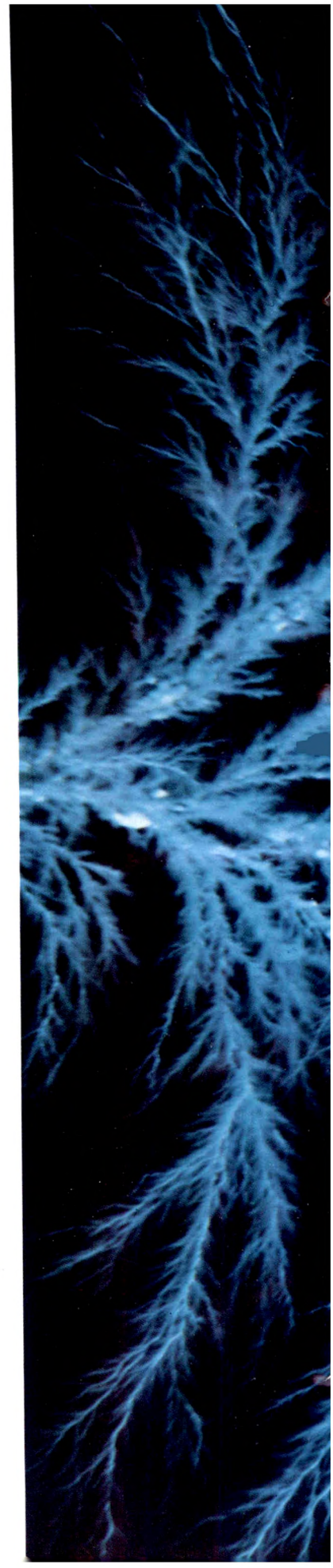

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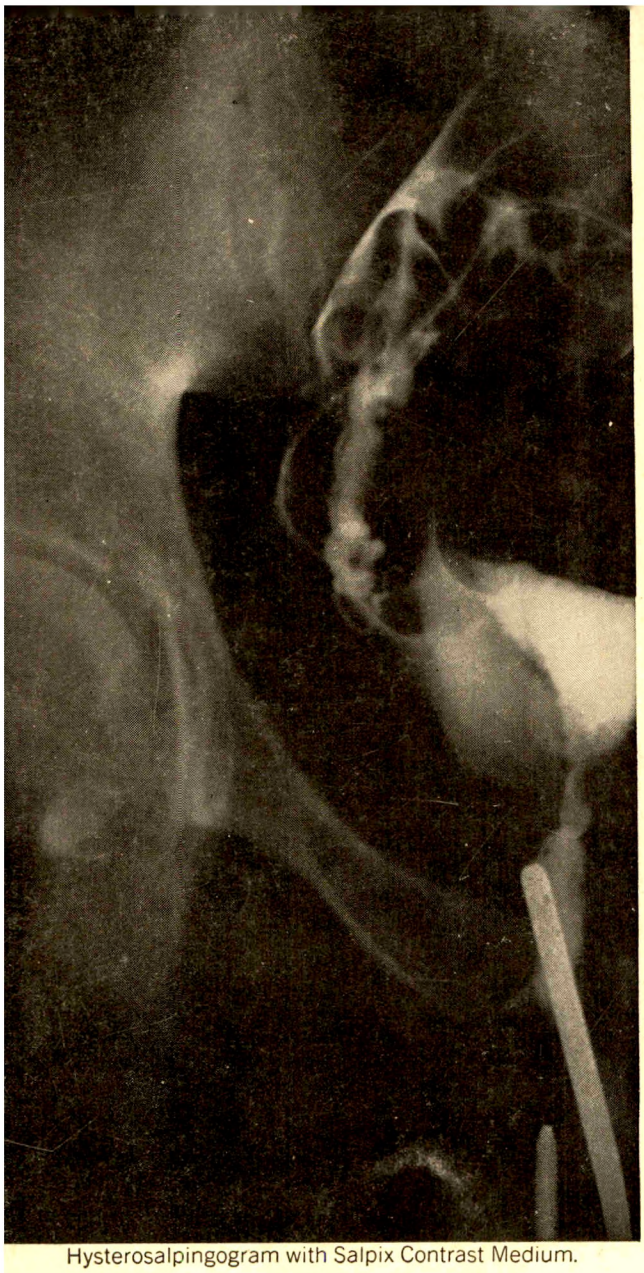

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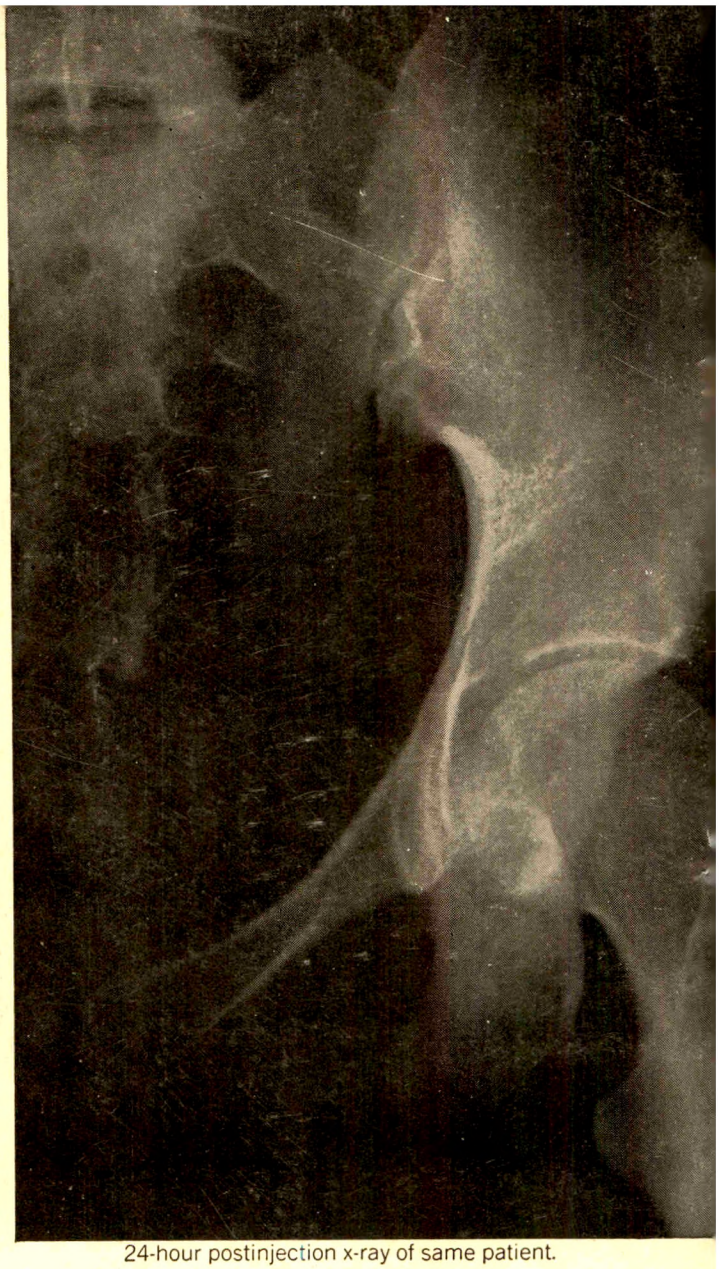

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THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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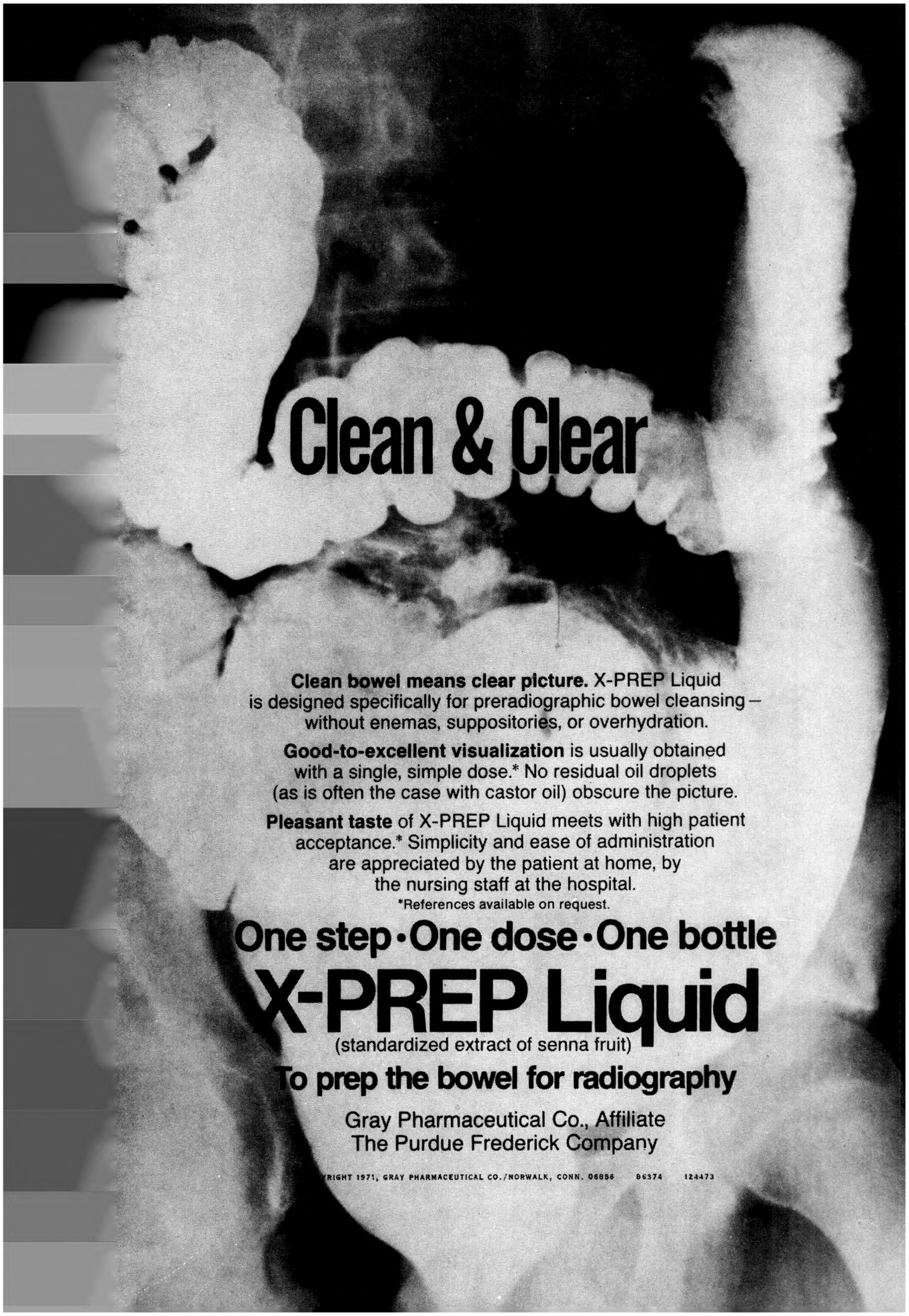

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The SKI Automated Gamma System gives results with or without automatic background subtraction in any of four different forms: (1) concentration derived from an automatically computed standard curve, (2) counts per minute at preset count, (3) counts per minute at preset time, or (4) ratio between standard and sample. In all cases, assay data, sample I.D. numbers and a summary of test parameters are printed on easy-to-file permanent paper tape.

☐ Please send details about The Gamma System.
☐ Please have a representative call for an appointment.
☐ We'd like a demonstration of The Gamma System at no expense or obligation to us.

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Telephone: Area code, number, extension _____

SKI **Smith Kline Instruments, Inc.**
880 West Maude Ave. • Sunnyvale, CA 94086 • (408) 732-6000
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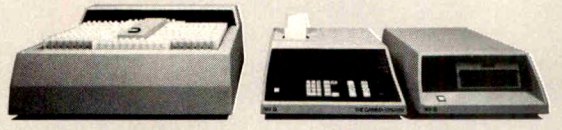

In One Minute a Load-A-Mat® unloads and reloads this many cassettes..

while delivering four radiographs to the processor.

In 15 seconds, the RADX Load-A-Mat can unload and reload an X-ray cassette automatically. And in broad daylight! It attaches directly to your film processor, so that radiographs are ready for viewing in only two minutes. Think what this means . . . no darkroom tie ups. No passboxes. Greater efficiency. Fewer errors. More patient processing.

The remarkable Load-A-Mat accepts four sizes of fresh film in separate light-tight portable magazines. Each stores 200 sheets of standard film furnished by the manufacturer of your choice. Once bulk-loaded into the magazines, the film remains untouched until it comes out of the processor. You're no longer troubled with artifacts, fogging, static or double exposures. No ruined screens either. And the Load-A-Mat lets your processor operate steadily for hours at full capacity.

Two Load-A-Mat models handle four sizes of either KODAK X-OMATIC Cassettes or the new LOAD-A-MATIC Cassettes available with your choice of medium, high speed or super high speed screens. Realize the kind of savings in time and money a Load-A-Mat can bring to your radiology operations.

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The
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is
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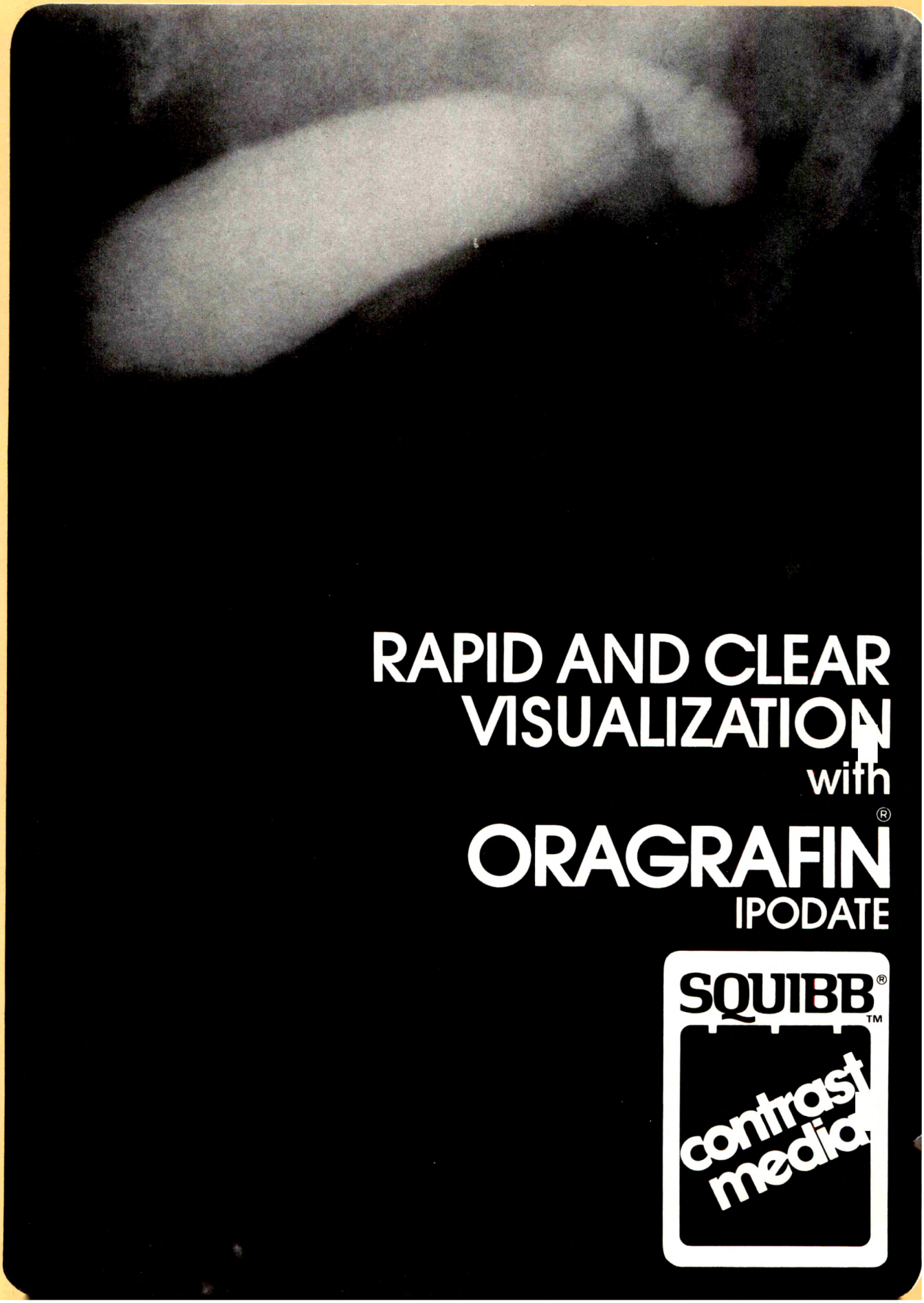
Effectiveness


RAPID AND CLEAR
VISUALIZATION
with
ORAGRAFIN®
IPODATE
SQUIBB®
contrast media™

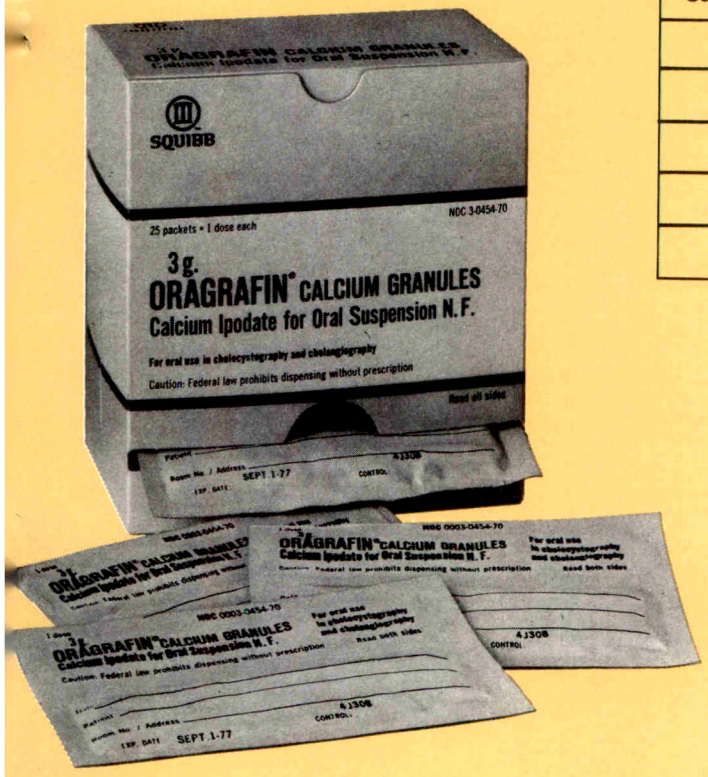
- Visualization of the ducts occurs in 1 to 3 hours and of the gallbladder within 5 hours or less with Oragrafin Calcium Granules (Calcium Ipodate for Oral Suspension N.F.).
- The gallbladder is opacified approximately 10 hours after ingestion of Oragrafin Sodium Capsules (Sodium Ipodate Capsules N.F.).
- Rapid absorption often permits visualization of even poorly functioning gallbladders. An unprepared patient can be studied the same day.
- When patients cannot take capsules, the granules are a convenient, palatable alternative.
- Both salts are usually well tolerated.

Dosage Schedules

FOR ROUTINE OVERNIGHT PROCEDURES Sodium Ipodate Capsules and/or Calcium Ipodate for Oral Suspension			
	Gallbladder Films		Films of the Gallbladder and Biliary Ducts
11 P.M.	6 Capsules (or 12 Capsules)	OR 1 packet of Granules (or 2 packets)	6 Capsules (or 1 packet of Granules, if preferred) and
next morning, 8 A.M.			1 packet of Granules
9 A.M.	Visualization of gallbladder	Visualization of gallbladder	Visualization of ducts and gallbladder
10 A.M.			
11 A.M.			

FOR RAPID, SAME-DAY PROCEDURES Calcium Ipodate for Oral Suspension	
Films of Gallbladder and Ductal System	
8 A.M.	2 packets of Granules
9 A.M.	Visualization of ducts
10 A.M.	Optimal visualization of ducts
1 P.M.	Visualization of gallbladder

See next page for complete prescribing information.



ORAGRAFIN®
CALCIUM GRANULES
Calcium Ipodate for Oral Suspension N.F.

ORAGRAFIN®
SODIUM CAPSULES
Sodium Ipodate Capsules N.F.

the reason is clear

ORAGRAFIN®
CALCIUM GRANULES
Calcium Iodate for Oral Suspension N.F.

ORAGRAFIN®
SODIUM CAPSULES
Sodium Iodate Capsules N.F.

DESCRIPTION
Oragrafin Calcium Granules (Calcium Iodate for Oral Suspension N.F.) and Oragrafin Sodium Capsules (Sodium Iodate Capsules N.F.) are oral radiopaque media for cholangiography and cholecystography. *Calcium* iodate is available in single-dose foil packets providing 3 g. calcium iodate as granules dispersed in flavored sucrose. When mixed with water, the granules produce a pleasantly-flavored suspension. *Sodium* iodate is available in capsules providing 500 mg. sodium iodate per capsule.

The organically bound iodine content of calcium iodate and sodium iodate is 61.7% and 61.4%, respectively.

ACTIONS
Calcium iodate and sodium iodate are absorbed from the gastrointestinal tract, excreted by the liver into the bile, and stored and concentrated in the gallbladder. The calcium salt, which is absorbed somewhat faster than the sodium salt, appears in the ducts as early as 30 minutes after ingestion. Optimal concentration of either salt in the hepatic and biliary ducts occurs within 1 to 3 hours after ingestion in nearly all cases; rarely, maximal opacification may be delayed for as much as 5 hours. Adequate opacification of the ducts usually persists for about 45 minutes. The ducts may be visualized in patients who have undergone cholecystectomy.

The gallbladder is optimally opacified approximately 10 hours after ingestion of either salt. Diagnostically adequate filling, however, often takes place within 5 hours or less after ingestion, particularly with the calcium salt. Thus, studies of ducts and gallbladder may be carried out in as little as 5 hours when necessary or desirable. In this connection, it should be pointed out that both salts have been administered to unprepared patients as well as to previously prepared patients, with no significant difference in results.

INDICATIONS
Calcium iodate and sodium iodate are indicated for cholecystography. They may also be used for cholangiography, although they are not considered the drugs of choice.

CONTRAINDICATIONS
Both preparations are contraindicated in persons who are hypersensitive to iodate salts.

WARNINGS
A history of sensitivity to iodine *per se* or to other iodinated compounds is not an absolute contraindication to the use of these preparations, but calls for extreme caution in administration.

Usage in Pregnancy
The safety of these preparations for use during pregnancy has not been established; therefore, they should be used in pregnant patients only when, in the judgment of the physician, their use is deemed essential to the welfare of the patient.

PRECAUTIONS
Increasing the dosage above that recommended increases the possibility of hypotension.

Anuria may result when these preparations are administered to patients with combined renal and hepatic disease or severe renal impairment.

Renal toxicity has been reported in a few patients with liver dysfunction who were given oral cholecystographic agents followed by urographic agents. Administration of urographic agents should therefore be postponed in any patient with a known or suspected hepatic or biliary disorder who has recently taken a cholecystographic contrast agent.

Gastrointestinal disorders which interfere with absorption and liver disorders which interfere with excretion may result in non-visualization of the hepatic and biliary ducts and the gallbladder.

Contrast agents may interfere with some chemical determinations made on urine specimens; therefore, urine should be collected before administration of the contrast media or two or more days afterwards.

Thyroid function tests, if indicated, should be performed prior to the administration of these preparations since iodine containing contrast agents may alter the results of these tests.

ADVERSE REACTIONS
Sodium iodate and calcium iodate are usually well tolerated. Unwanted effects such as mild and transient nausea, vomiting, or diarrhea sometimes occur but the incidence can be reduced by restricting the dosage to 3 g. and administering only the Granules to patients who may be prone to gastrointestinal reactions. Headache, dysuria, or abdominal pains may occur infrequently as transient disturbances.

Hypersensitivity reactions may include urticaria, serum sickness-like reactions (fever, rash, arthralgia), other skin rashes, and, rarely, anaphylactoid shock. They are more likely to occur in individuals with a history of allergy, asthma, hay fever, or urticaria, and in those who have previously demonstrated hypersensitivity to iodine compounds.

DOSAGE AND ADMINISTRATION
The Capsules should be taken with as little water as possible. They may be swallowed in rapid succession or slowly over the course of ½ hour or more, depending on patient preference.

The Granules should be stirred vigorously into a small amount of water (¼ glass or less) and swallowed immediately. If lukewarm rather than cold water is used, the patient will find the suspension quite palatable and there will be less likelihood of nausea.

A total dose of 6 g. (12 Capsules or 2 packets of Granules) per 24 hour period should not be exceeded.

When an upper gastrointestinal barium study is scheduled, prior administration of either salt will not interfere. Either salt may also be used in conjunction with an intravenous cholangiographic agent.

The recommended dosage and patient preparation for cholangiography and/or cholecystography are as follows:

Routine Cholecystography
On the day before the examination, the patient may, if feasible, eat a high-fat lunch so that the gallbladder will evacuate and refill with opacified bile. The evening meal, which should be light and fat-free, should be eaten 2 hours before the contrast medium is taken. The contrast agent should be administered 10 to 12 hours before the roentgenologic examination. The usual dose is 6 Capsules (providing 3 g. sodium iodate), although up to 12 Capsules may be given; 1 to 2 packets of Granules may be used instead. No other food or drink except small amounts of water should be taken before the examination. A mild laxative or other cathartic may be given, but is not generally necessary.

Roentgenologic examination of the gallbladder is performed the next morning in the usual manner, employing the standard positions. An immediate study of the wet films will determine any need for repositioning of the patient. Administration of a fat meal or other cholecystokinetic agent provides good dynamic studies of gallbladder function. If a barium meal examination is also to be performed, the fat meal may be given simultaneously with or following the barium meal. Gallbladder function studies are then made after the barium meal examination is completed.

Cholangiography
No prior preparation of the patient is necessary. For this procedure, the Granules should be used. They are administered one hour before cholangiography. Although only one packet (providing 3 g. calcium iodate) may suffice, two packets (6 g.) are usually necessary for visualization of the ducts.

Beginning one hour after ingestion of the Granules, and at 15-minute intervals thereafter, films should be taken, preferably with the aid of tomography or laminography. With proper positioning of the patient, residual contrast medium in the gastrointestinal tract is not likely to be a problem, particularly with the more rapidly absorbed calcium salt. Optimal visualization of the ducts is generally achieved between 1½ and 2½ hours after ingestion of the medium.

Combined Cholangiography and Cholecystography
Preparation of the patient and administration of dosage should be as outlined under *Routine Cholecystography*, with a dosage of 6 Capsules or, if preferred, one packet of Granules taken on the evening before examination. On the following morning, one hour before the examination, one additional packet of Granules should be administered.

Beginning one hour after the morning dose, and at 15-minute intervals thereafter, films should be made, preferably with the use of tomography or laminography. While the gallbladder will have concentrated the evening dose of the medium, the additional morning dose will provide opacification of the ducts, and simultaneous visualization may be achieved. When satisfactory films have been obtained, a fat meal may be administered to provide gallbladder function studies.

Rapid Combined Cholangiography and Cholecystography
No prior preparation of the patient is necessary. On the day of examination, the patient should be given two packets of Granules. Beginning one hour later, examination of the ducts should be performed as described under *Cholangiography*. If the gallbladder has not opacified sufficiently by the time the cholangiographic examination is completed, further cholecystograms may be taken after 2 or 3 hours. Dynamic studies of gallbladder function may be carried out in the usual manner.

HOW SUPPLIED
Oragrafin Calcium Granules (Calcium Iodate for Oral Suspension N.F.), single-dose foil packets; boxes of 25 packets (3 g. calcium iodate per packet). Oragrafin Sodium Capsules (Sodium Iodate Capsules N.F.), boxes of 25 vials (6 capsules each vial); bottles of 100; and Unimatic® single-dose packages of 100.

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contrast media
The Reason is Clear

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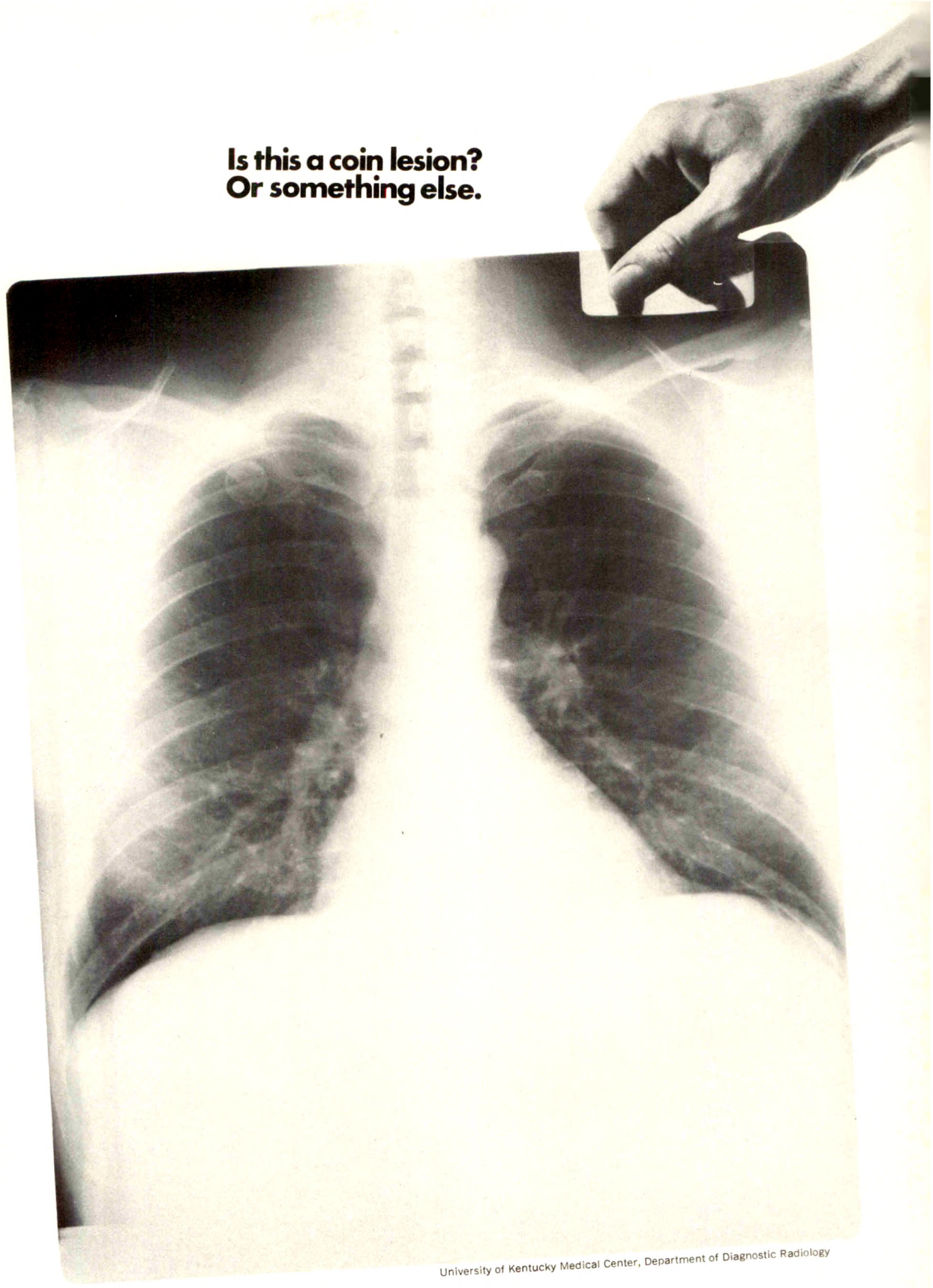
Or something else.



University of Kentucky Medical Center, Department of Diagnostic Radiology

(See GAF® page xxvii)

Intensified
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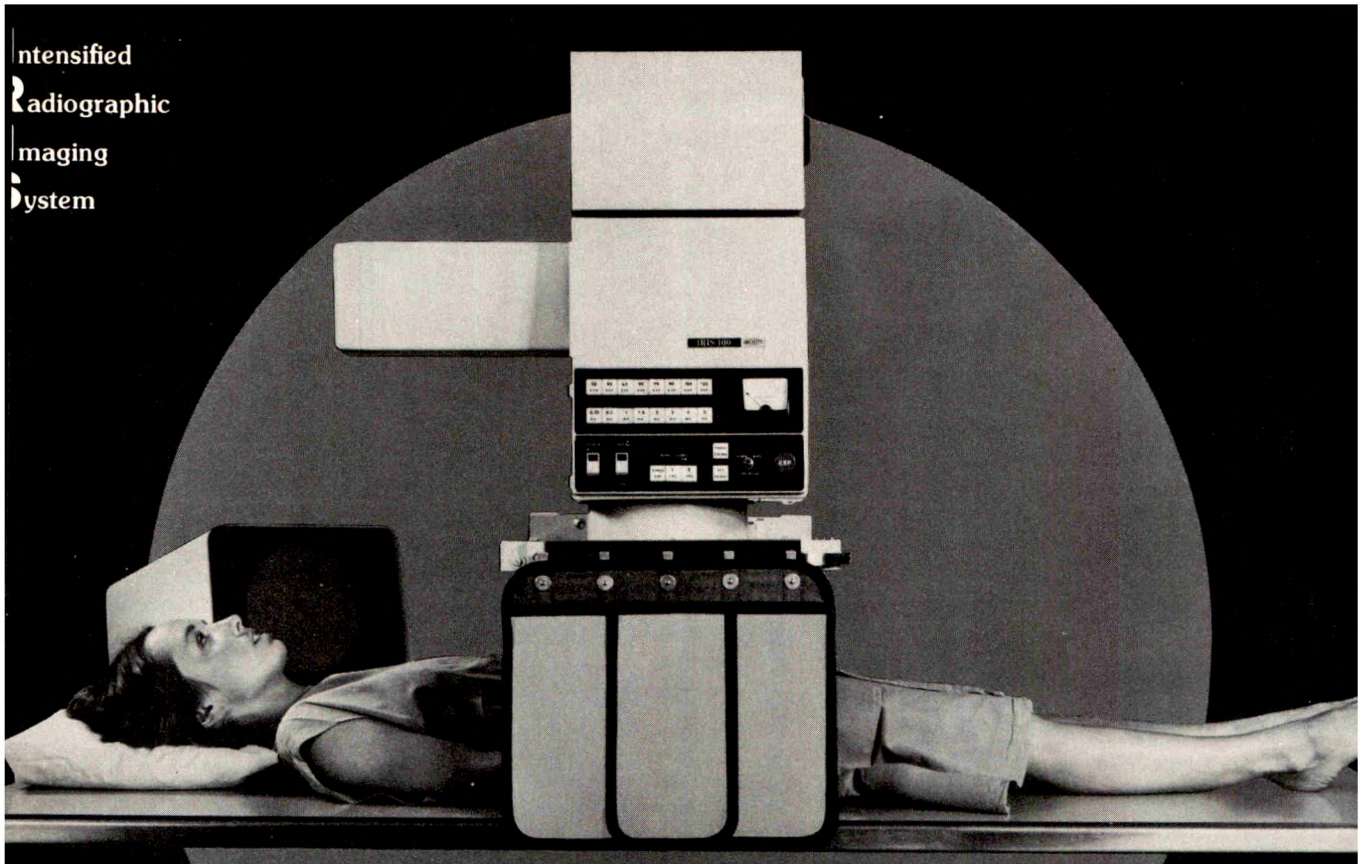

IRIS 100 adds television fluoroscop and 100mm spotfilming to your present x-ray room

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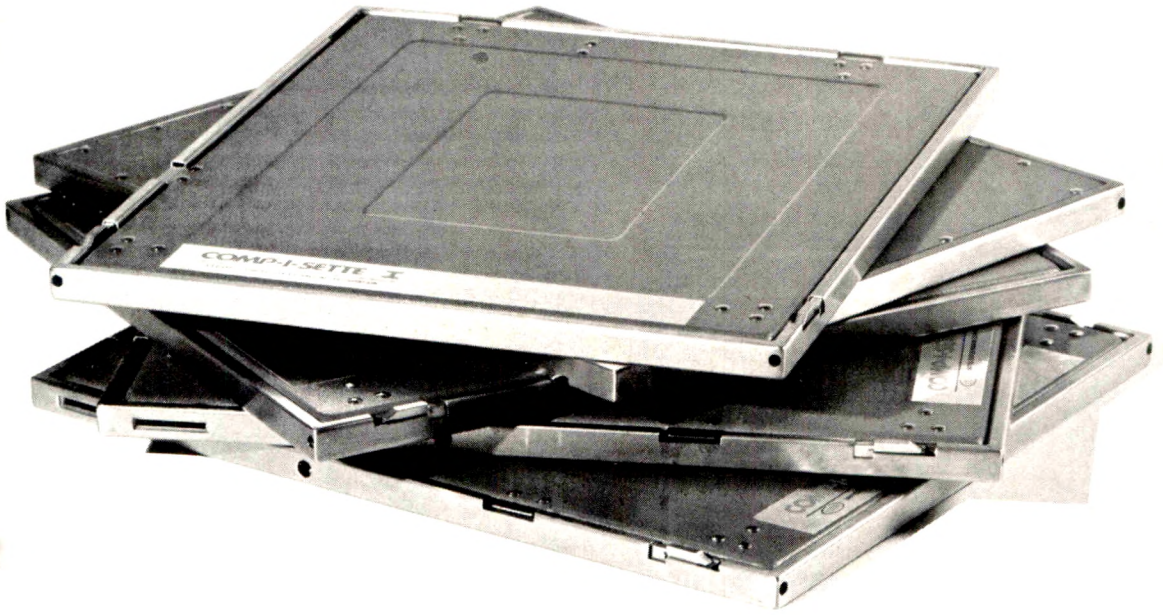
Tough all over



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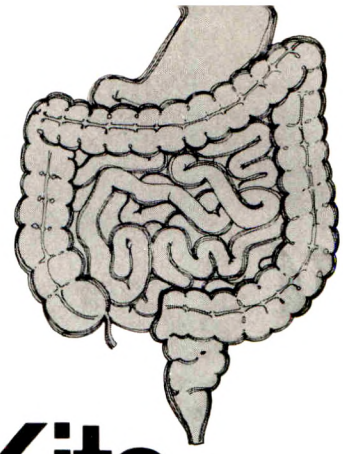
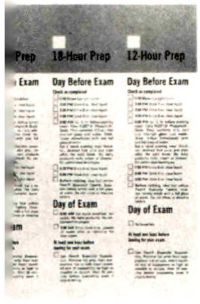
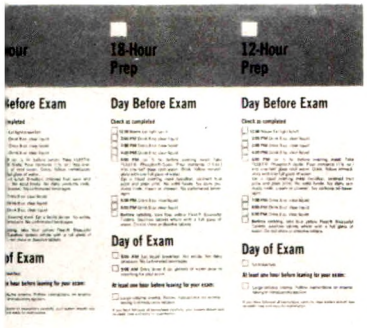


Enema Prep Kits

clearer x-rays, and fewer retakes





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☐ **With effective and balanced methods of prepping**
Utilizing a combination of diet, hydration, and evacuation, the Fleet Barium Enema Prep Kits meet all the criteria for preparing the colon for examination as recommended by the American College of Radiology Ad Hoc Committee on Detection of Cancer of the Colon.[1]

☐ **Available in two convenient forms**
There are two Fleet Barium Enema Prep Kits, so that now you have the option of choosing the final step that you prefer—enema or suppository.

☐ **With easy-to-follow instructions**
Each Fleet Barium Enema Prep Kit supplies clearly written, concise instruction sheets for easy, dependable administration by the nurses in the ward or by your patient at home. Check-off boxes ensure completion of each step.

☐ **With a flexible time-table**
The Fleet Barium Enema Prep Kits also offer three convenient time schedules —12, 18, or 24 hours. So now, you can tailor the prepping to fit better into your schedule and your patient's routine.

"In my experience across the country, talking to technicians and doctors, . . . the greatest defect in colon examinations, . . . is in the preparation of the colon."[2]

For better colon preparation and fewer repeat examinations, use Fleet Barium Enema Prep Kits routinely —as your prepping agent of choice in the Radiology Department and on an outpatient basis.

References:
1. *Detection of Colon Lesions,* First Standardization Conference, Amer. College of Radiology, (Chicago, Ill., 1973), pp. 203-204.
2. *Ibid,* p. 76.

Fleet pharmaceuticals

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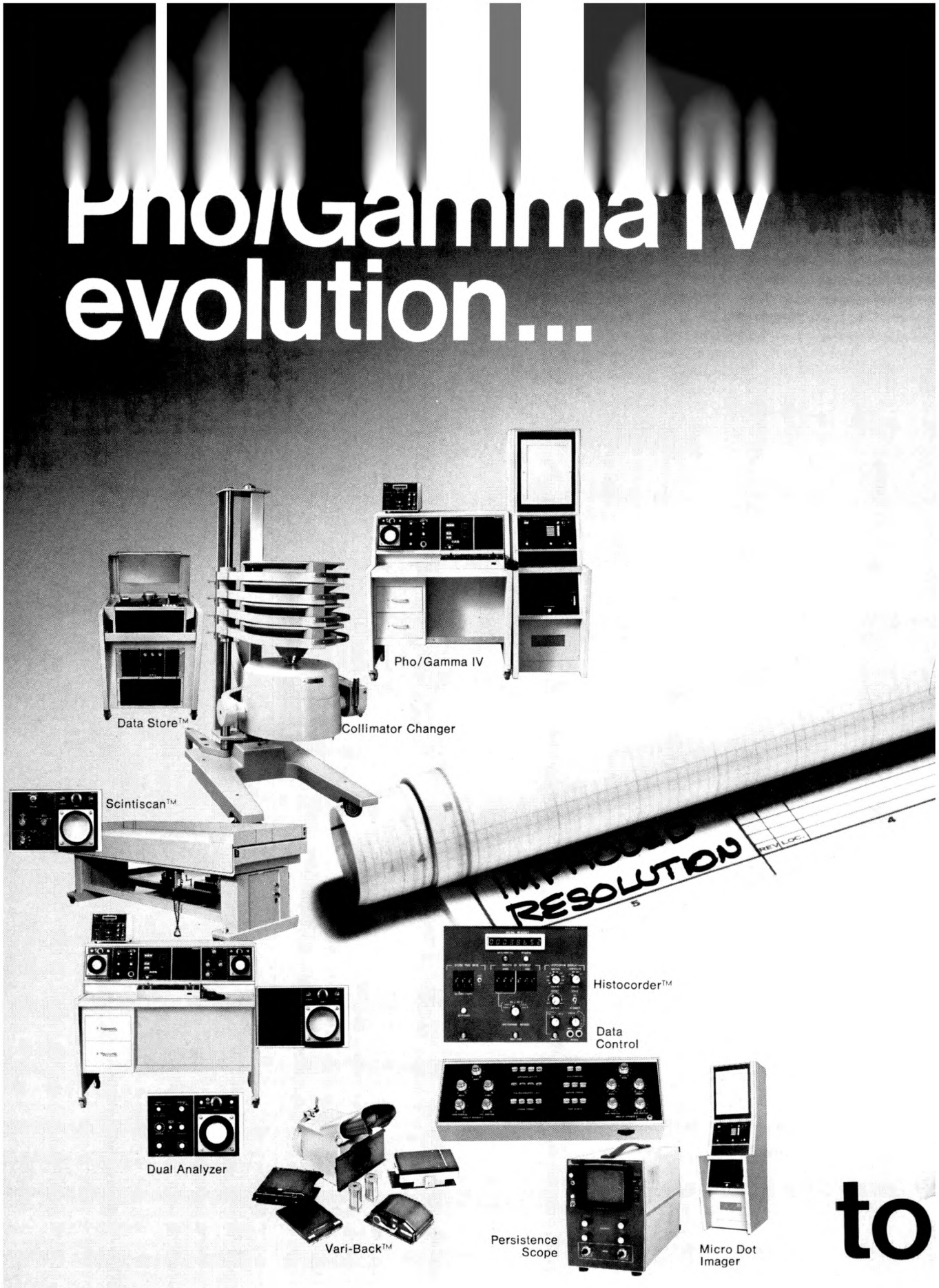
Pho/Gamma IV evolution...



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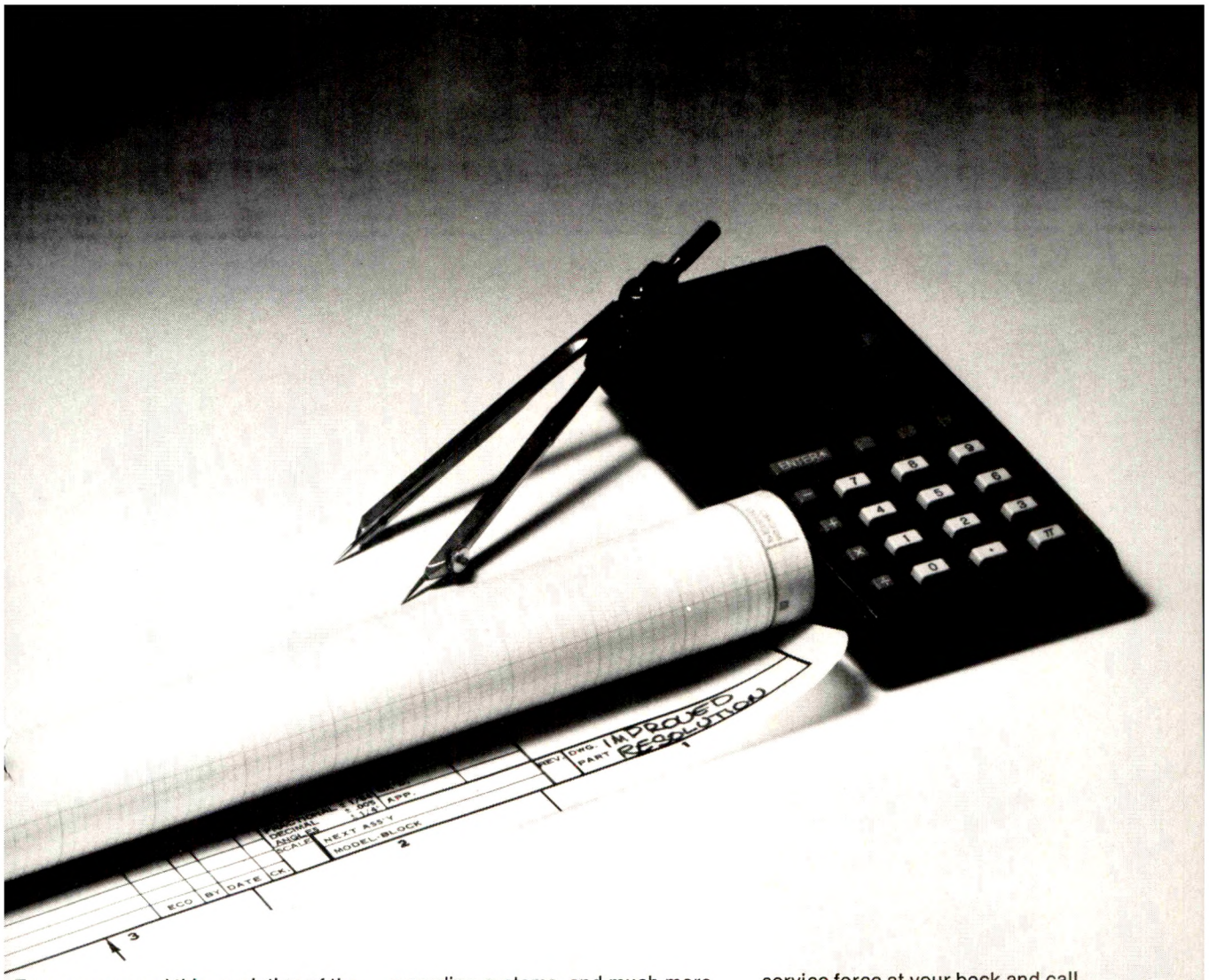

Even as you read this, evolution of the Pho/Gamma IV Scintillation Camera system goes on. Our product development engineers are in daily contact with working laboratories nationwide. And the Pho/Gamma IV is being continuously improved to meet your growing, changing needs.

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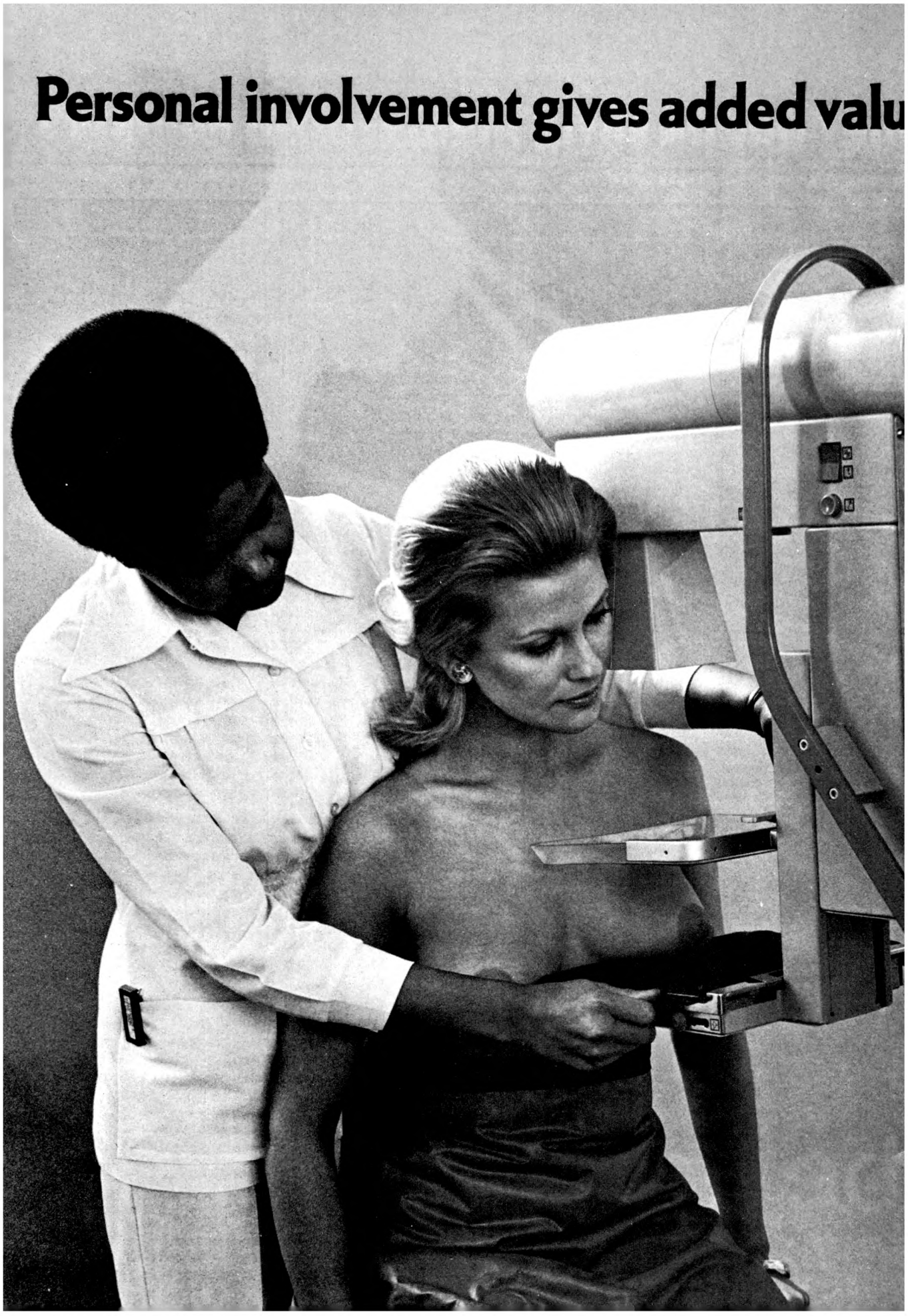
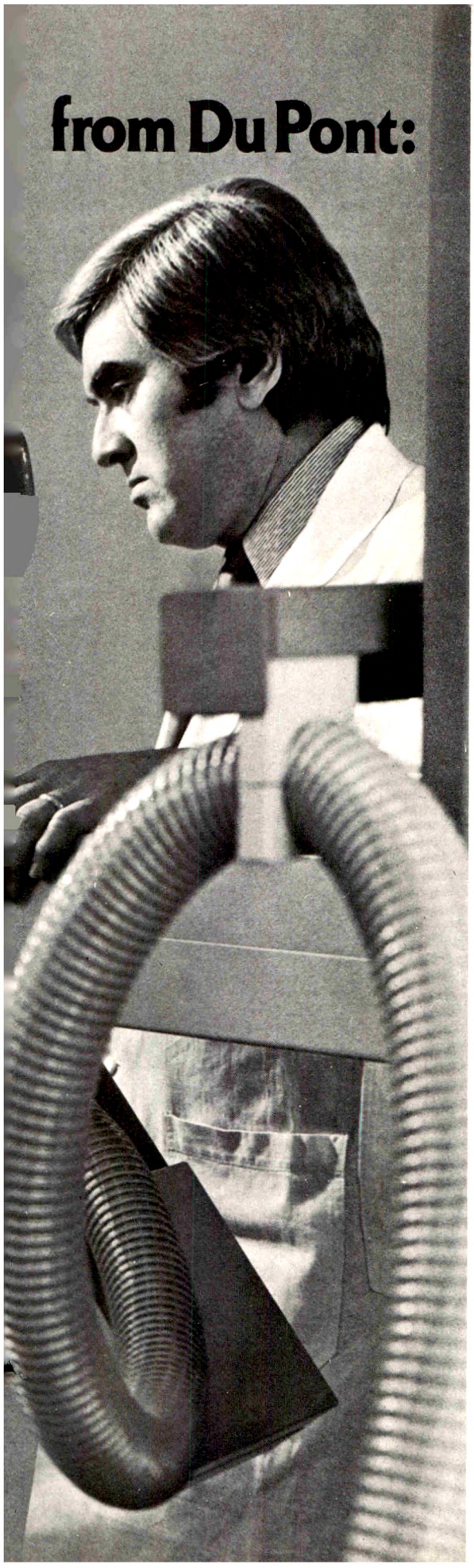

Personal involvement gives added valu



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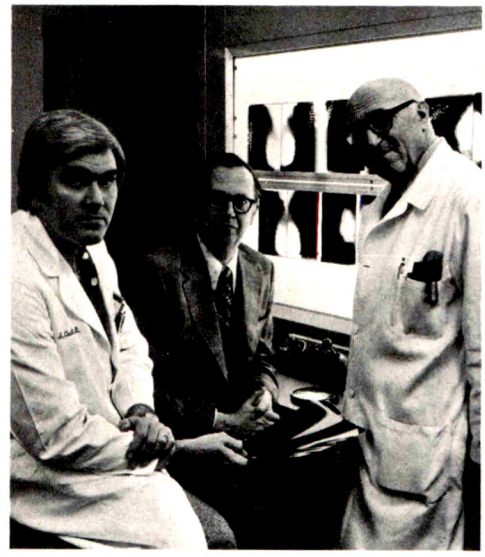

Left to right:

Bernard J. Ostrum, M
Chairman of Departm
of Diagnosis, Albert
Einstein Medical Cent
Philadelphia.

R. E. Wayrynen, PhD.
X-ray Technical Mana
Du Pont Company.

Harold J. Isard, M.D.,
Chairman of Division
of Radiology, Albert
Einstein Medical Cent
Philadelphia.

In the late 1960's, Drs. Isard and Ostrum were looking for a better way to perform mammography—one with lower patient risk than conventional systems. They came to Du Pon for help.

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Phosphotec provides all the nonradioactive components required to prepare ^{99m}Tc-stannous pyrophosphate complex. Each vial contains a sterile, nonpyrogenic lyophilized powder prepared from 40 mg. tetrasodium pyrophosphate decahydrate (equivalent to 23.9 mg. tetrasodium pyrophosphate) and 1.0 mg. stannous fluoride; pH is adjusted with sodium hydroxide or hydrochloric acid. The product does not contain a preservative. At the time of manufacture, the air in the vials is replaced by nitrogen.

Reconstitution of Phosphotec with sterile sodium pertechnetate-^{99m}Tc results in an aqueous solution of Technetium 99m-Stannous Pyrophosphate Complex.

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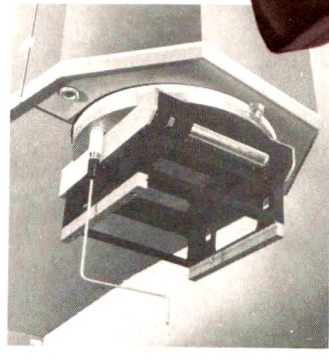

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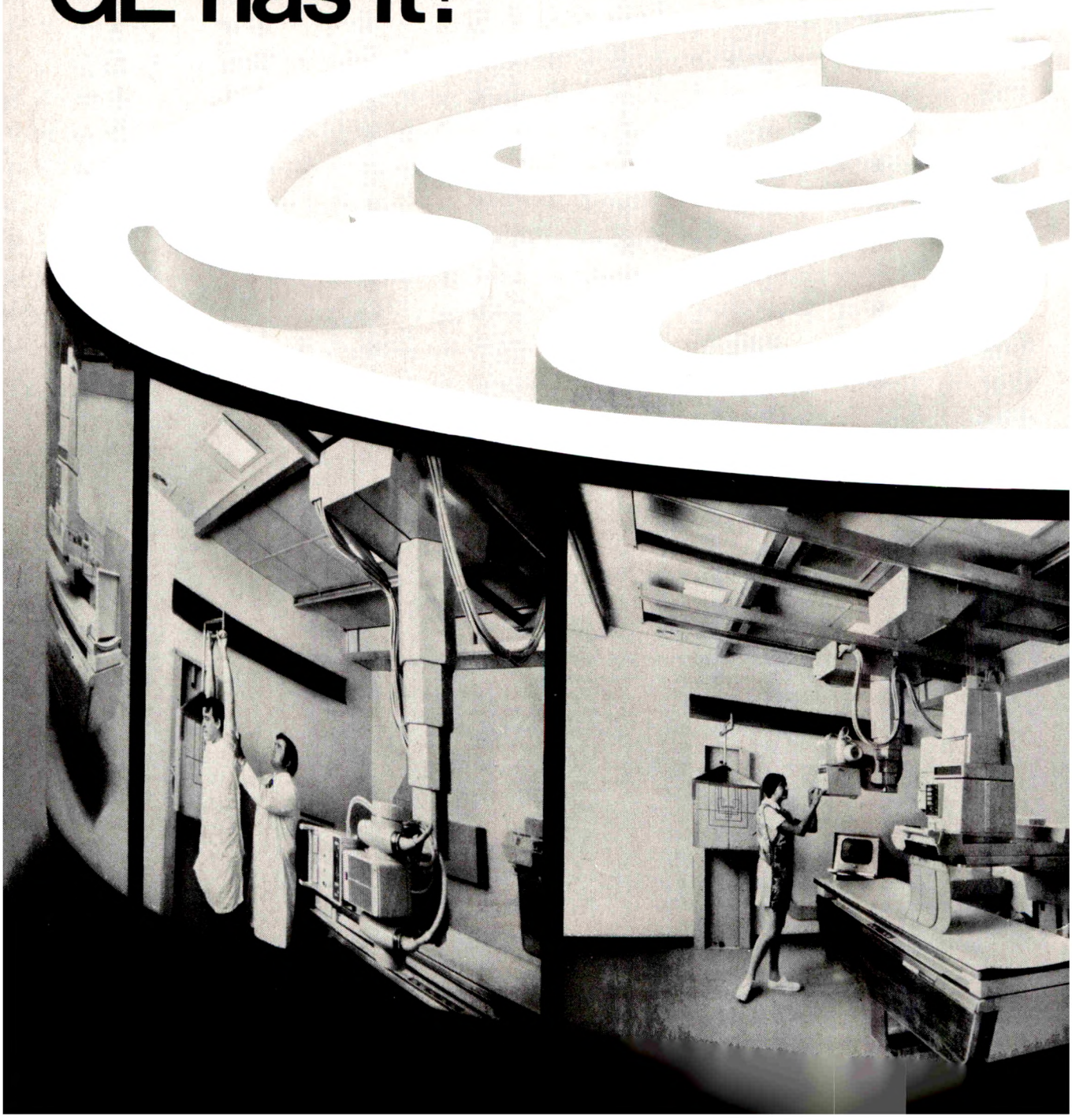

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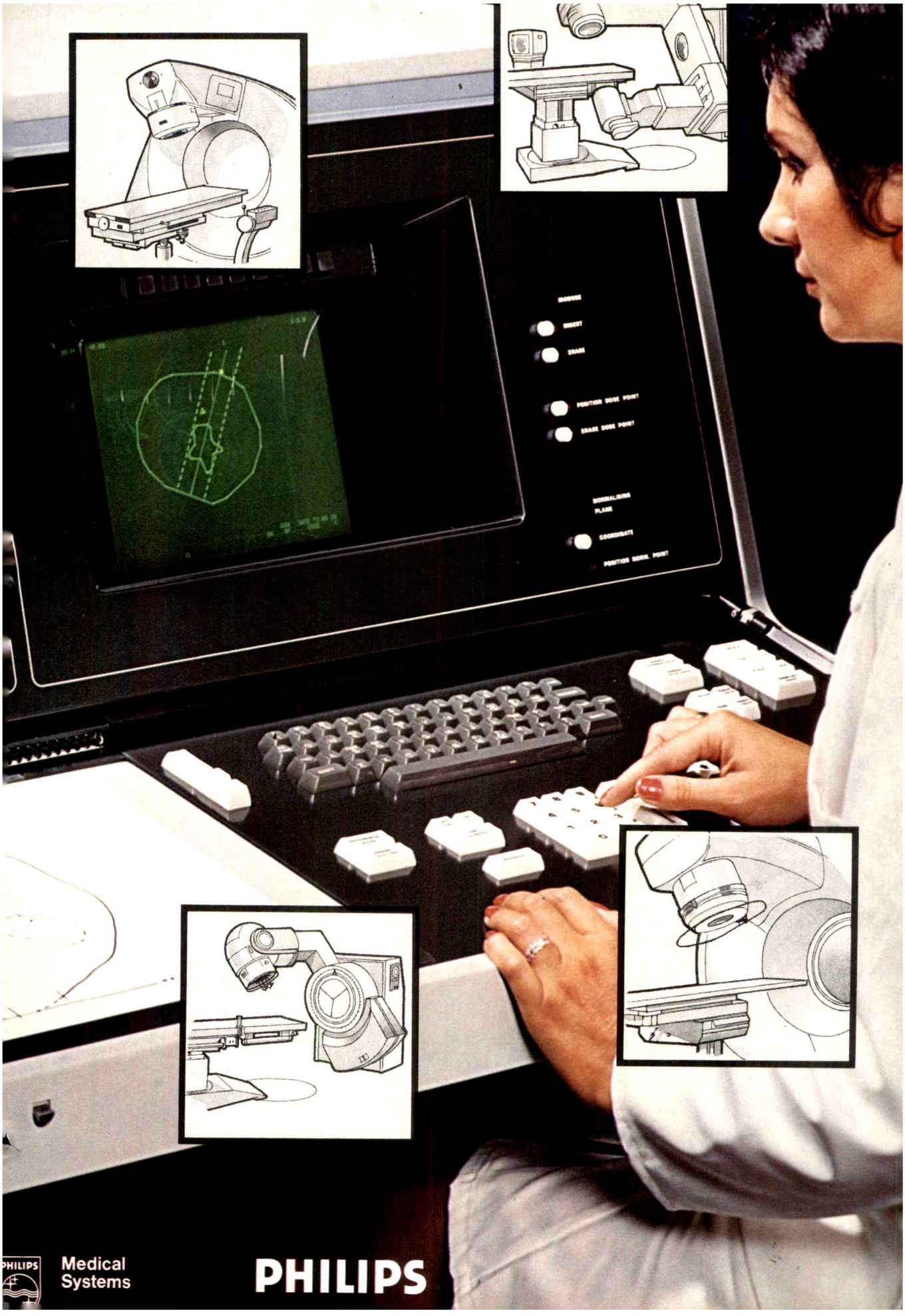
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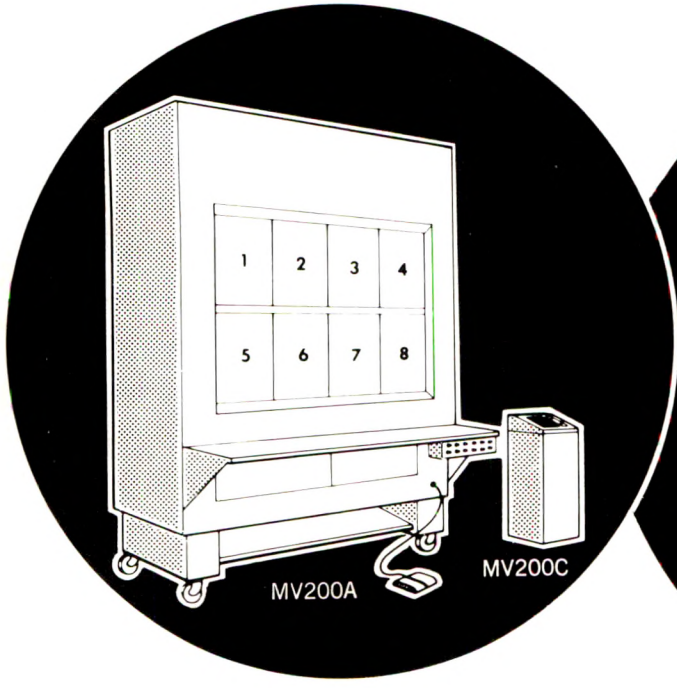

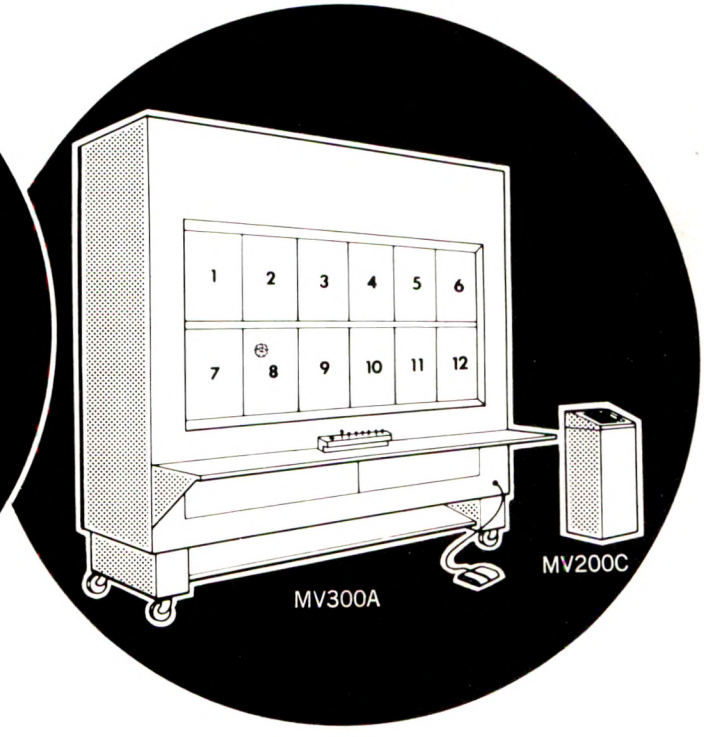

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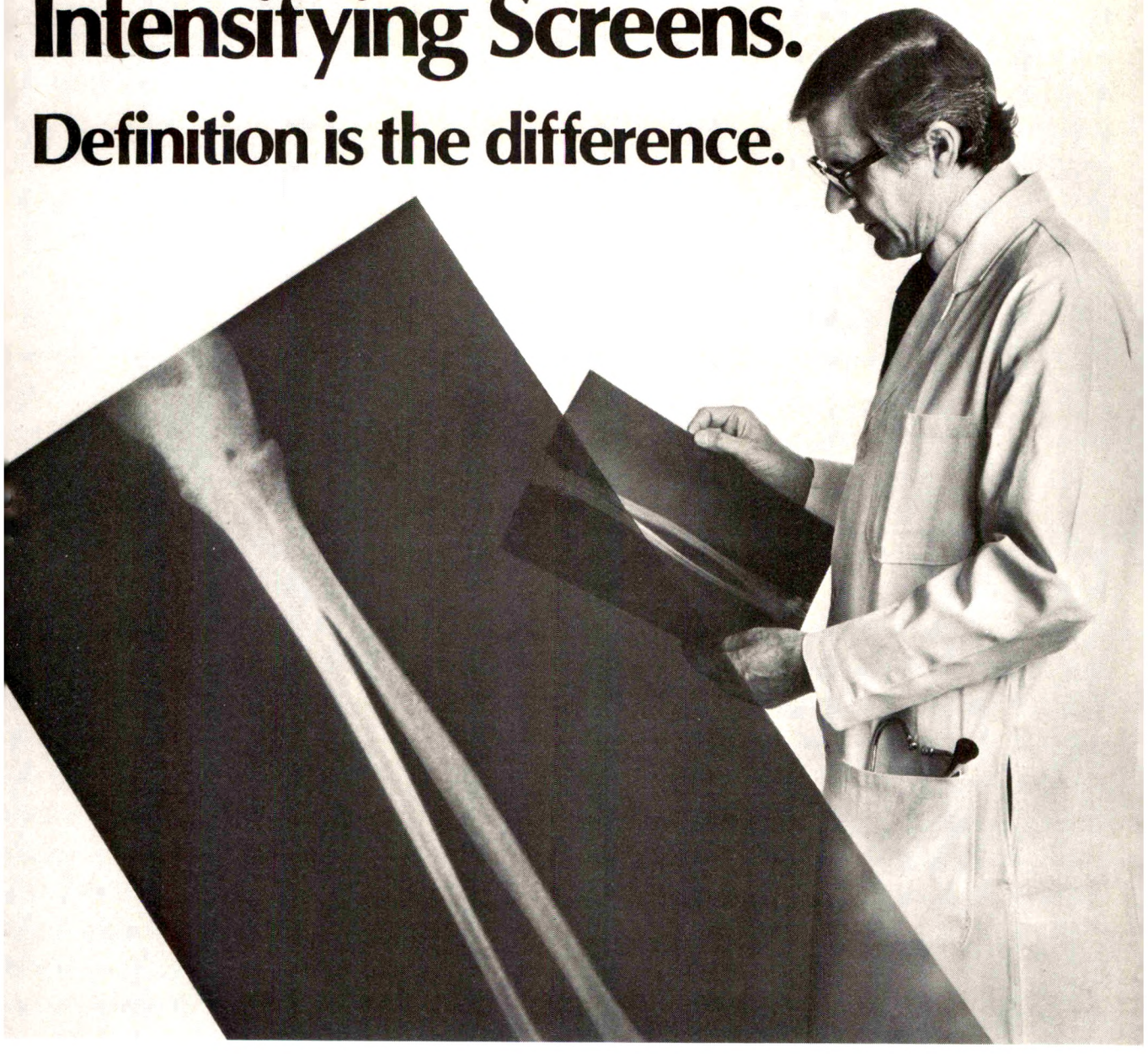

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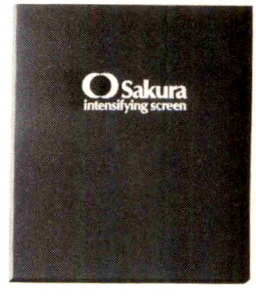

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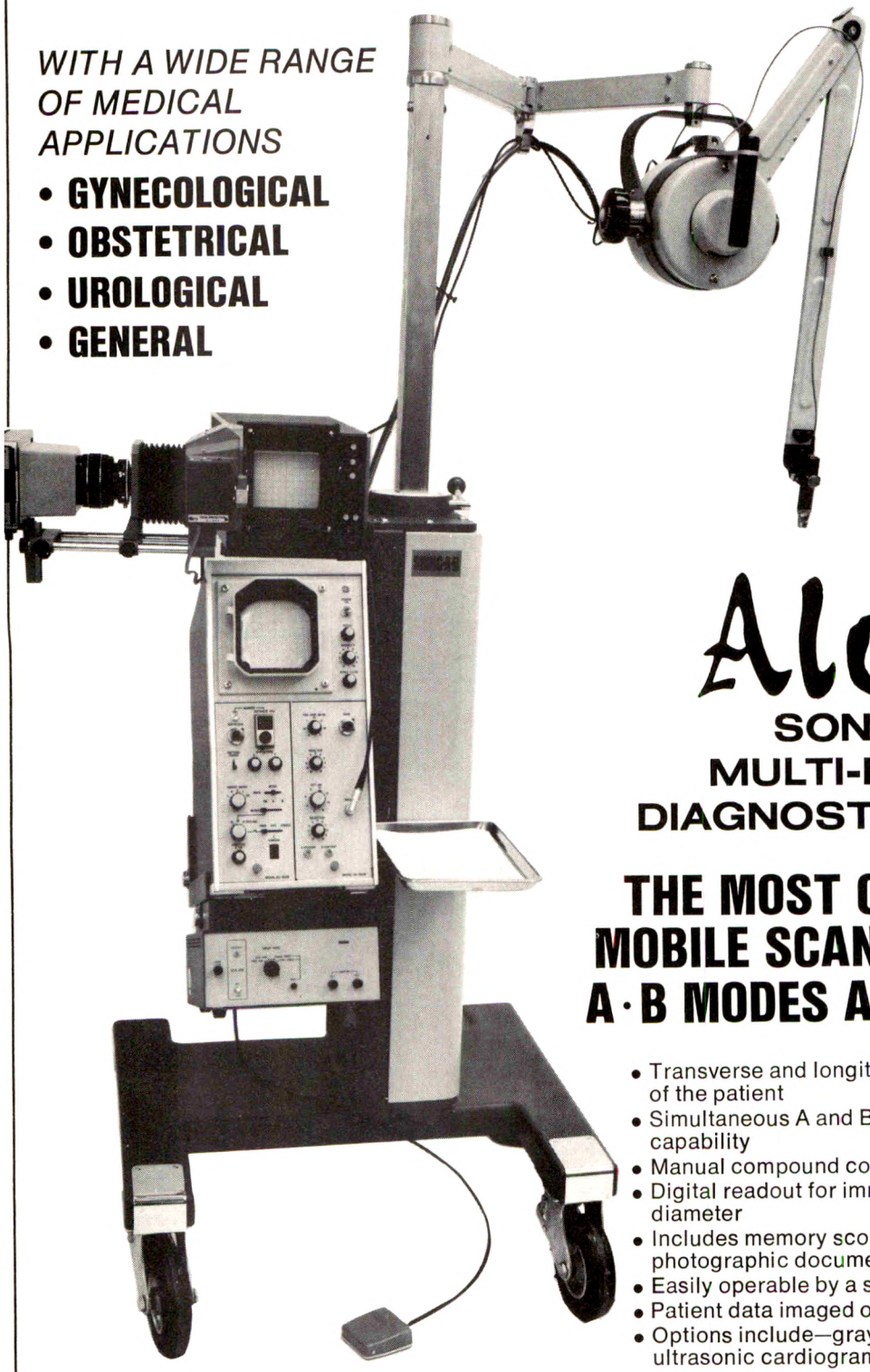

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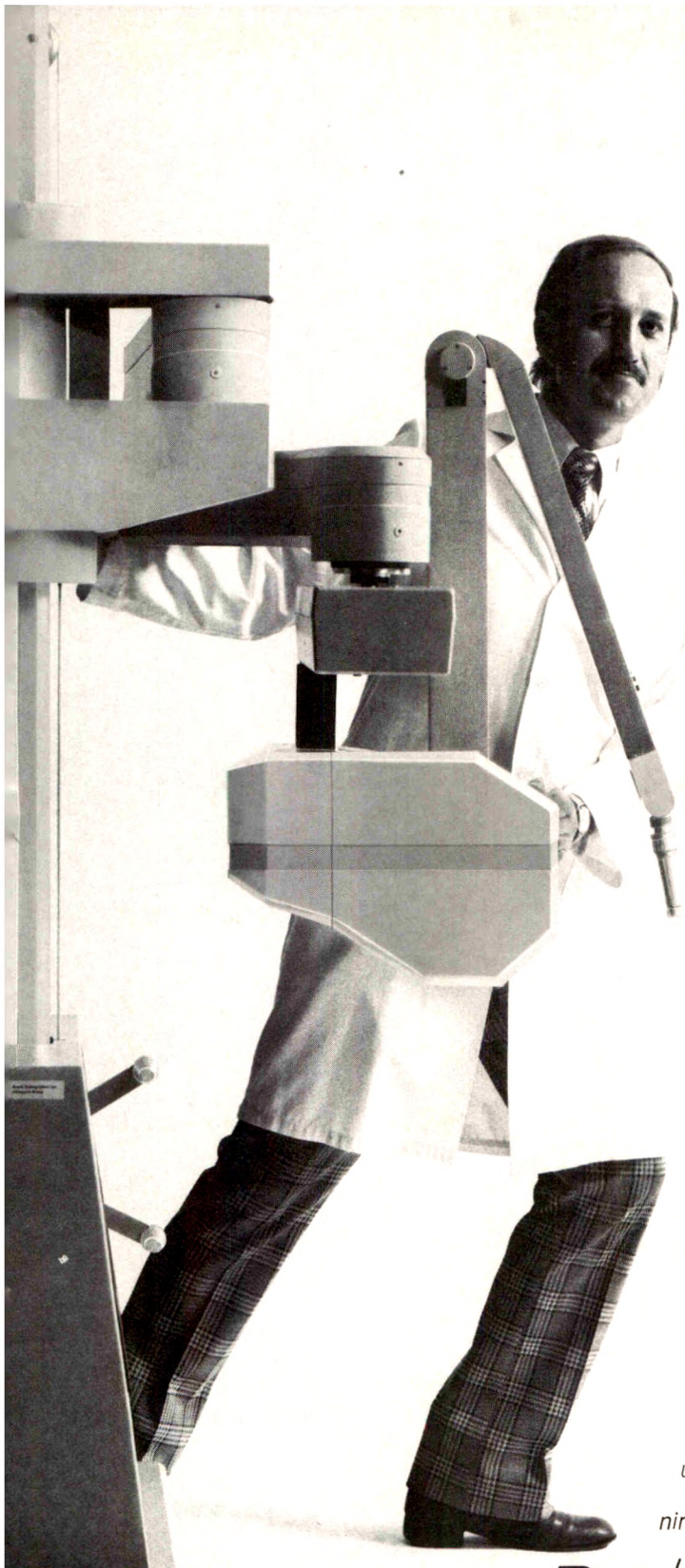

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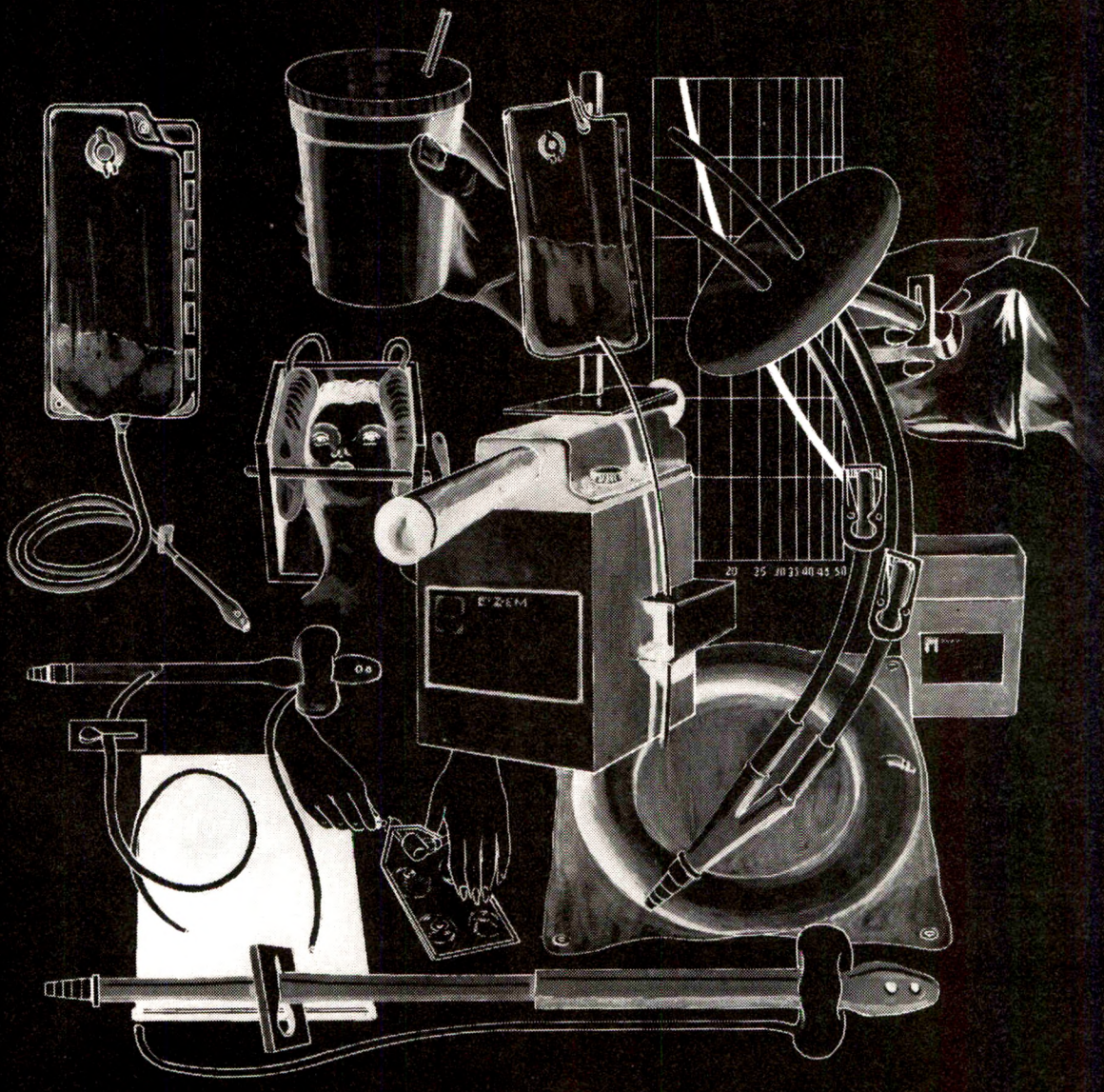

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VOL. 125 NOVEMBER, 1975 No. 3

THE TETHERED CONUS*

By CHARLES R. FITZ, M.D., *and* DEREK C. HARWOOD-NASH, M.B., CH.B.
TORONTO, ONTARIO, CANADA

ABSTRACT:

The tethered conus is a real and treatable entity. It presents with a variable though fairly constant symptom complex. The diagnosis is probably not even considered in many patients with this condition. Recognition of the possibility of this condition by both the clinician and radiologist is needed. Properly performed oil or air myelography is the only method of preoperative confirmation of a tethered conus and should be done in all cases where it is suspected.

THE tethered conus remains a controversial subject which has received scattered attention, primarily in the surgical literature, since the 1950's.[4,7,10,12] Surprisingly it has been the subject of only one radiological report, by Gryspeerdt[5] in 1963. He laid down the important fundamentals of myelography in this condition and related problems. The symptoms and the association with spina bifida occulta were recognized as early as 1918,[3] although authors as recently as 1972 have been unaware of the condition in scoliosis,[13] one of its common presenting forms.

The "syndrome" is known by a variety of names: tethered filum, filum terminale syndrome, cord traction syndrome, tight filum, and low conus. The authors have chosen the term tethered conus because it is the low position of the conus that is important clinically and visible roentgenographically.

The tethered conus presents with various signs and symptoms, primarily upper motor neuron type symptoms of spastic gait, weakness, muscle atrophy, bowel and bladder dysfunction, and often orthopedic conditions such as scoliosis, foot deformities, painful or numb lower extremities, or low back pain. Abnormal Babinski reflexes are often present on one or both sides. Knee and ankle jerks are usually hyperactive but may be decreased. External signs of spinal dysraphism may be present, including hypertrichosis, subcutaneous lipomas, dermoids, and sinus tracts. These occur in roughly half of the patients in our experience.

The same clinical findings are often noted in association with other related con-

* Presented at the Annual Meeting of the Society for Pediatric Radiology, San Francisco, California, September 22–23, 1974.
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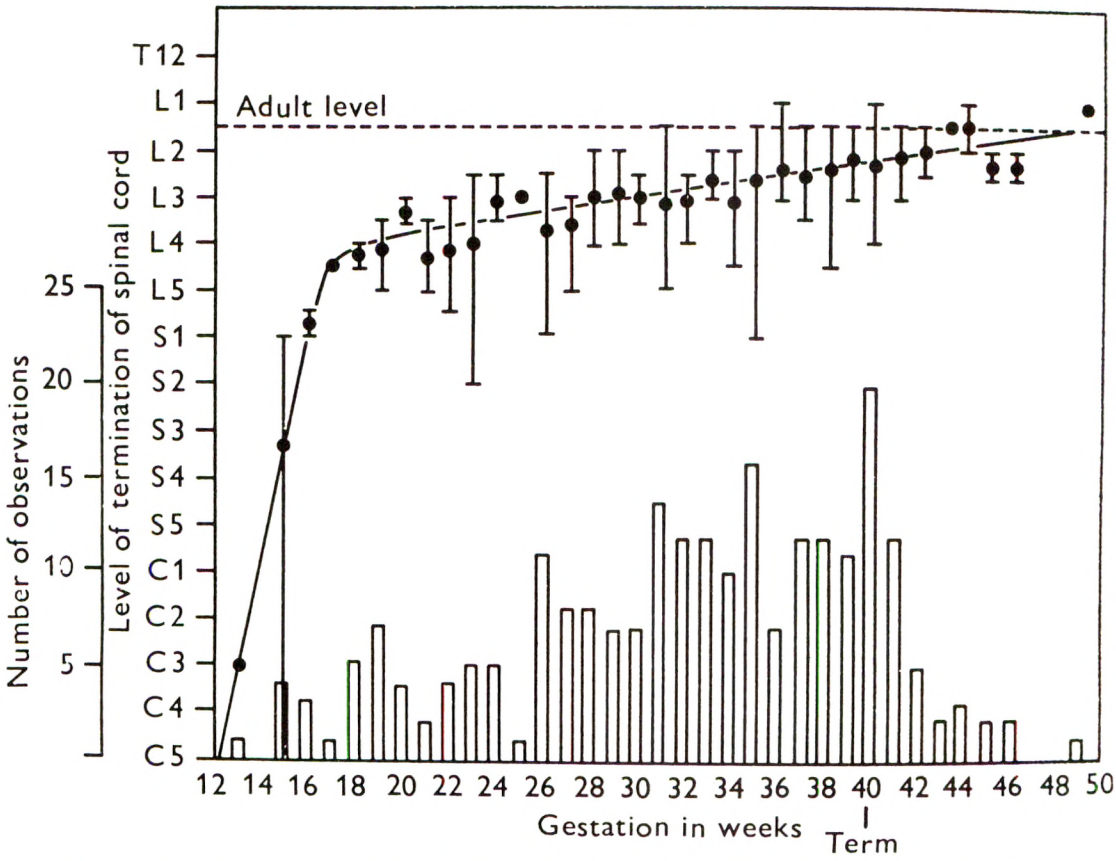


FIG. 1. Note L1–2 level of conus tip may be reached at 31 weeks' gestation. (Reprinted from *The Journal of Anatomy*, 1969, *106*, with permission of the publishers.)

ditions of spinal dysraphism such as diastematomyelia and meningocele. We have excluded these patients in our own series, but have included those with a generalized slight widening of the lumbar dural sac where no discrete outpouching of an intraspinal meningocele was present at myelography. The widened dural sac may be seen without any other abnormality except spina bifida occulta, and is considered by the authors to be the mildest form of abnormality in the spinal dysraphism spectrum. Other intradural abnormalities such as lipomas and dermoids are also seen with the tethered conus. Some authors[8] believe that these "accessory" findings are the true cause of the symptoms. We believe this not to be the case as ten out of 24 in our series had no associated masses or visible band at operation or myelography.

To diagnose a low tethered conus, one must know the normal conus level and how it got there. The human embryo has a distinct tail bud[11] which gradually regresses. By 30–35 mm (nine weeks) it is no longer present externally. The conus and filum which develop from the tail bud are at the lower end of the spinal canal at this stage. Between 30 and 100 mm (nine–16 weeks) there is a rapid ascent of the conus due to faster growth of the lumbar vertebrae than the cord. This is combined with atrophy of the terminal portion of the cord which forms the filum.

Barson[2] measured the conus termination in 252 neurologically normal infants and fetuses of 15 to 49 weeks gestation. He found that as an average, the adult level (L1—2) is reached by 48 weeks gestational age (two months postnatally). It can be at

that level as early as 31 weeks (Fig. 1). In autopsies of older children he also noted conus levels of L3 in a three year old and L2—3 in a twelve year old. James and Lassman[8] found similar results in a smaller group of normal children. Based on these findings and our own experience we think a conus tip below the L2—3 interspace in a child older than five years is abnormal (Fig. 2). By age twelve the conus tip should be at the mid L2 level. In addition, a filum measuring greater than two mm at myelography is probably abnormal. Because the measurement is not corrected for magnification error which varies slightly from case to case, one cannot be absolute about this measurement.

MATERIAL

At the Toronto Hospital for Sick Children, we have seen 24 patients with a myelographically and surgically confirmed diagnosis of a tethered conus having between nine months' and five years' postoperative follow-up. As noted, those with diastematomyelia and meningocele have been excluded from this series. Those having intradural lipomas, cysts, and dermoids have been included. Symptoms are as noted on Table 1. Most children had more than one clinical finding and tended to fall into one or three groups, being seen by either urologists, orthopedic surgeons, or neurosurgeons and neurologists. Our own series has fewer children with scoliosis than some other reported series, presumably because of the increased awareness of the condition on the part of the clinicians other than orthopedic surgeons. Two of these children had been previously diagnosed as having cerebral palsy. All but two had definite spina bifida occulta. The two uncertain cases were older cases in which the plain

TABLE I

SYMPTOMS

Urinary incontinence	5
Other bladder problems	5
Bowel dysfunction	2
Abnormal reflexes	9
Sensory loss	8
Weakness	8
Scoliosis	5
Small limb	3
Low back pain	3

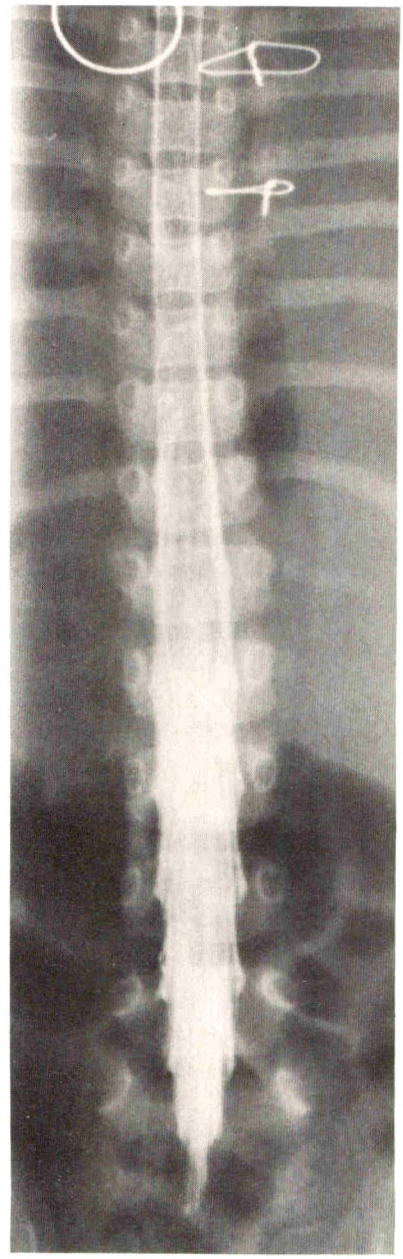


FIG. 2. Normal myelogram. Water soluble postmortem myelogram on infant. Note conus ends at mid L2 level and nerve roots exit in a course nearly parallel to the spinal axis.

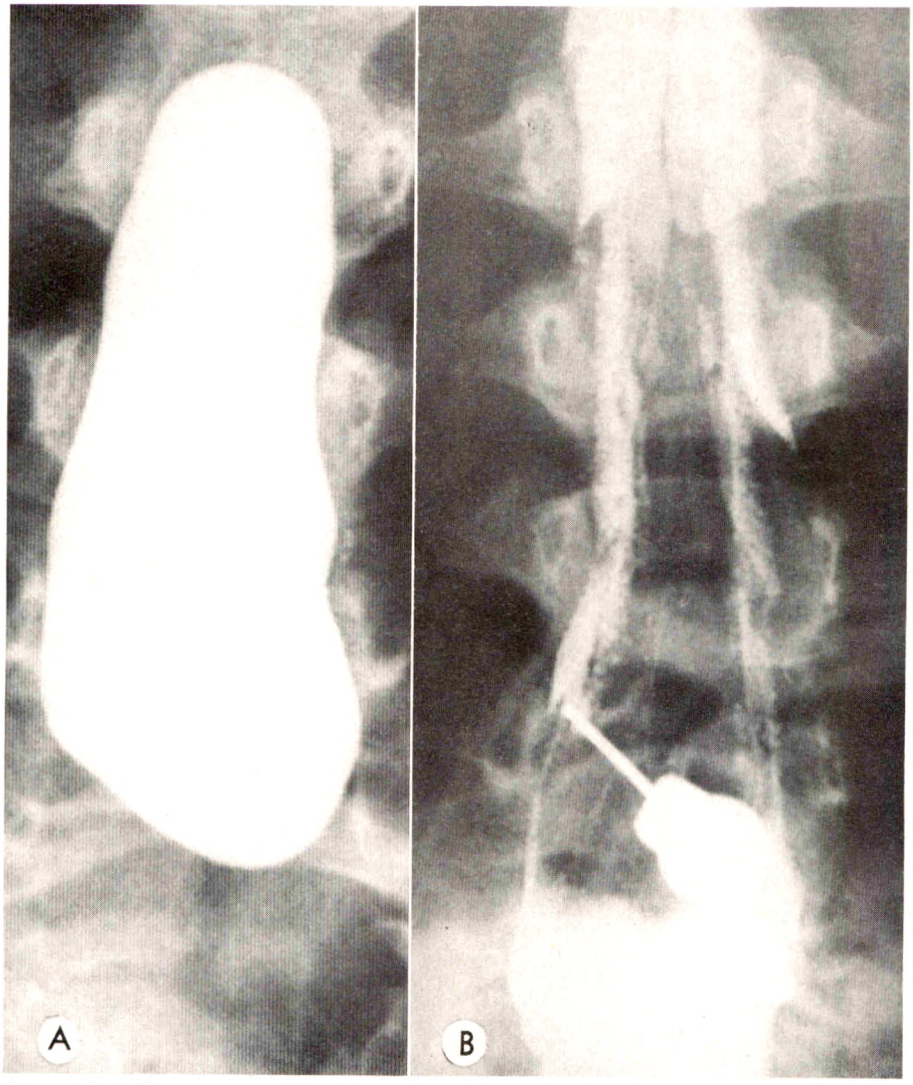


FIG. 3. (*A*) Prone view. Conus not seen in spite of large amount of contrast medium extending to L_5 level. (*B*) Supine view. Conus and attached lipoma easily seen in supine projection. Also note nerve roots course more laterally than normal.

spine films could not be located for review. Considering the evidence of the remaining cases, these were probably also abnormal.

MYELOGRAPHY

Myelography was done in all patients, usually under general anesthesia. Although cisternal puncture has been recommended to avoid piercing a low cord we have successfully used a short bevel lumbar puncture needle in the lumbar area without difficulty. When there is a question of a tethered conus, we try to make the puncture slightly off the midline and have not pierced a thickened filum or conus with the use of this method. Six to 20 cc of Pantopaque were used in the investigations. The amount will vary with the age of the child and the width of dural sac, which is often abnormally large.

The key to the diagnosis is supine myelography. Although the anterior spinal artery generally ends at the conus tip, it is not always seen at that level at myelography because the conus, when tethered, is usually held dorsally in the spinal canal.

Thus it may rise out of the contrast medium column unless a very large amount of contrast material is used (Fig. 3*A*). This large amount in turn, may be so dense as to obscure the artery or the underside of the conus to which the artery is attached. The supine position requires less contrast material and usually allows complete visualization of the conus tip and the filum which also might be obscured by a large amount of contrast material. The anteroposterior position is best for visualization of the conus and filum.

With a large dural sac, the supine position is especially advantageous (Fig. 3*B*).

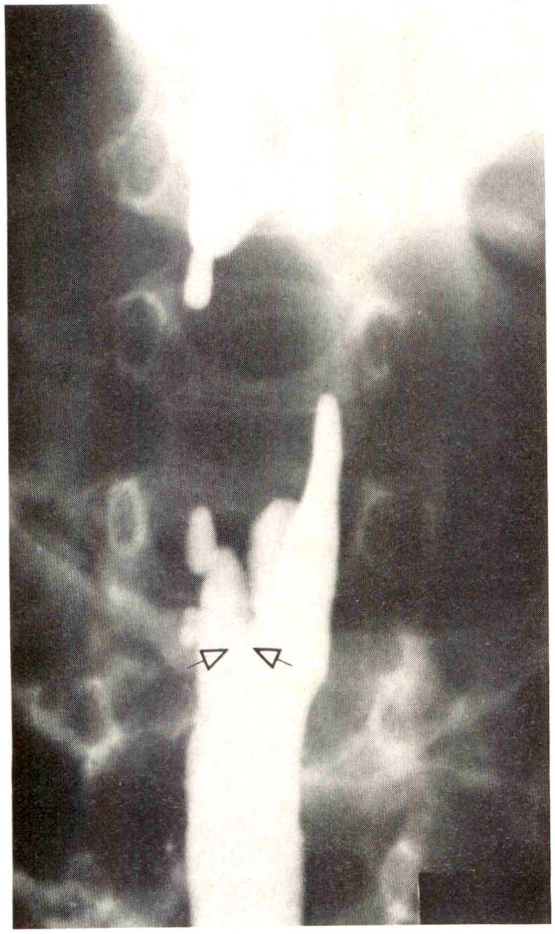


Fig. 4. Low conus with attached cyst. Conus tip at upper level of L3 (open arrows). Irregularity of contrast medium around dorsal and lateral edges of conus suggests additional defect. Attached cyst confirmed at operation.

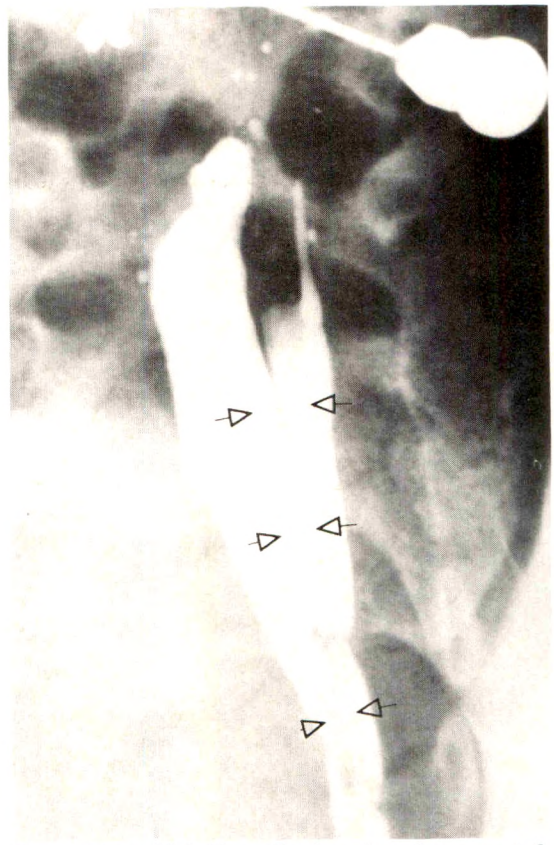


Fig. 5. Very thick filum. Supine view shows no definite end to conus, but continuation of very thickened filum into sacral region.

Small lipomas, cysts, etc., attached to the conus are easily seen (Fig. 4). At times what appears to be a thick filum may in fact be lipomatous tissue around the filum, but this differentiation cannot be made by myelography. At times the conus has no definite tip, but gradually tapers to a very thick filum (Fig. 5). The filum in these cases, though often appearing externally like normal neuronal tissue at operation, contains no viable nerve structures when sectioned.

Other myelographic evidences of a tethered conus such as the more laterally coursing nerve roots are also best seen supine (Fig. 6). This becomes more prominent as the level of the conus is lower. A widened dural sac is also more likely to occur when the conus is quite low at the L4 or L5 level (Fig. 3*A*).

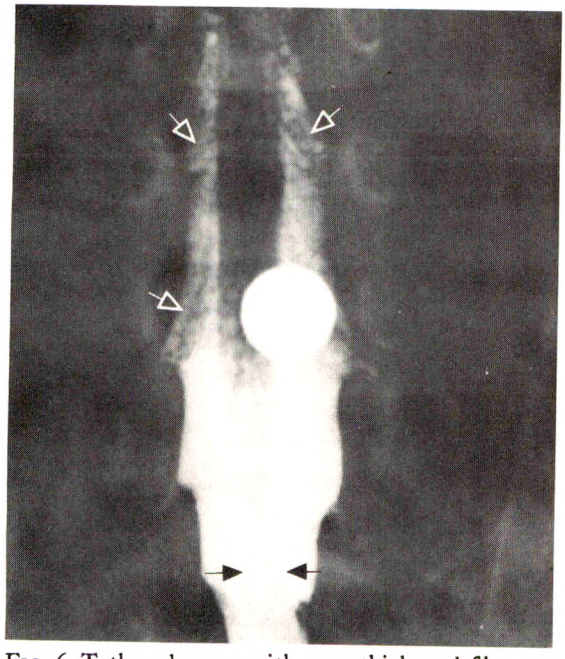


FIG. 6. Tethered conus with very thickened filum as in Figure 5 (black arrows) and well visualized laterally coursing nerve roots (white open arrows). Compare with nerve roots of Figure 2.

At the Hospital for Sick Children we have made a horseshoe shaped pad which allows the patient to be supine without removal of the needle (Fig. 7, *A* and *B*). The patient is first examined in the prone position from caudal sac to posterior fossa to rule out any other abnormalities, particularly diastematomyelia. Following the prone examination the patient is lifted and rolled into the pad for the supine portion of the examination. Some anesthesiologists also prefer to use the pad while the patient is prone, the space in the "U" allowing for better chest expansion than on the table top itself. Supine filming is perhaps more easily done by removal of the needle. In the long run the pad saves time and allows better examination as it: prevents the need for re-puncture to remove the dye; prevents extradural leakage which may occur on needle removal; allows one to add more contrast material if necessary; and lastly, remains as a marker of vertebral body level as in the prone position.

We have used air myelography at times. It has two advantages. One need not remove the contrast material and the supine position is not needed. Over-all we find a preference for oil examination. Because tomography is necessary for air myelography the procedure tends to be longer even though the extra supine examination is not needed. Detail, although adequate, is less than on oil myelography. The filum and nerve roots are harder to see. Although we have not encountered the situation, it is possible that air myelography would be more likely to miss a fibrous diastematomyelia which would also have similar symp-

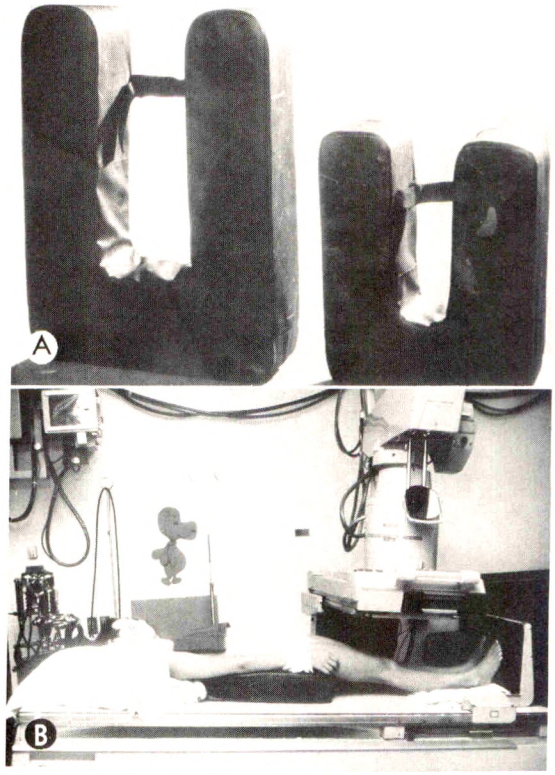
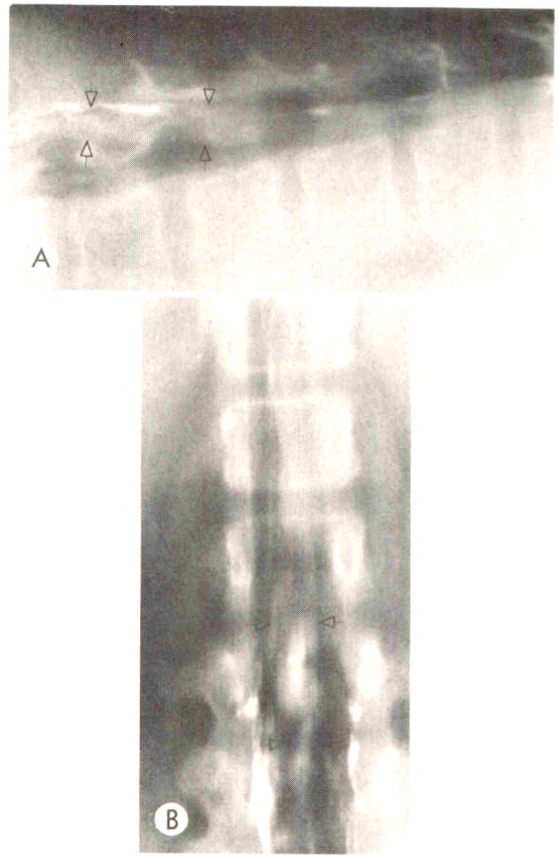


FIG. 7. Supine myelography pad. (*A*) View of large and small pads. Velcro strap at top of "U," keeps the arms of the pad from spreading. (*B*) Larger pad in use with older child.

➤

FIG. 9. Operative release of filum. (*A*) Conus tip and filum with surrounding lipoma exposed. (*B*) Forceps holding cut filum that was at lower end of incision.

FIG. 8. Air myelography. (*A*) Lateral view. Tethered conus (open arrows) outlined with air. Note dorsal location. (*B*) Anteroposterior projection. Conus less well seen. Opaque densities from previous oil myelography.



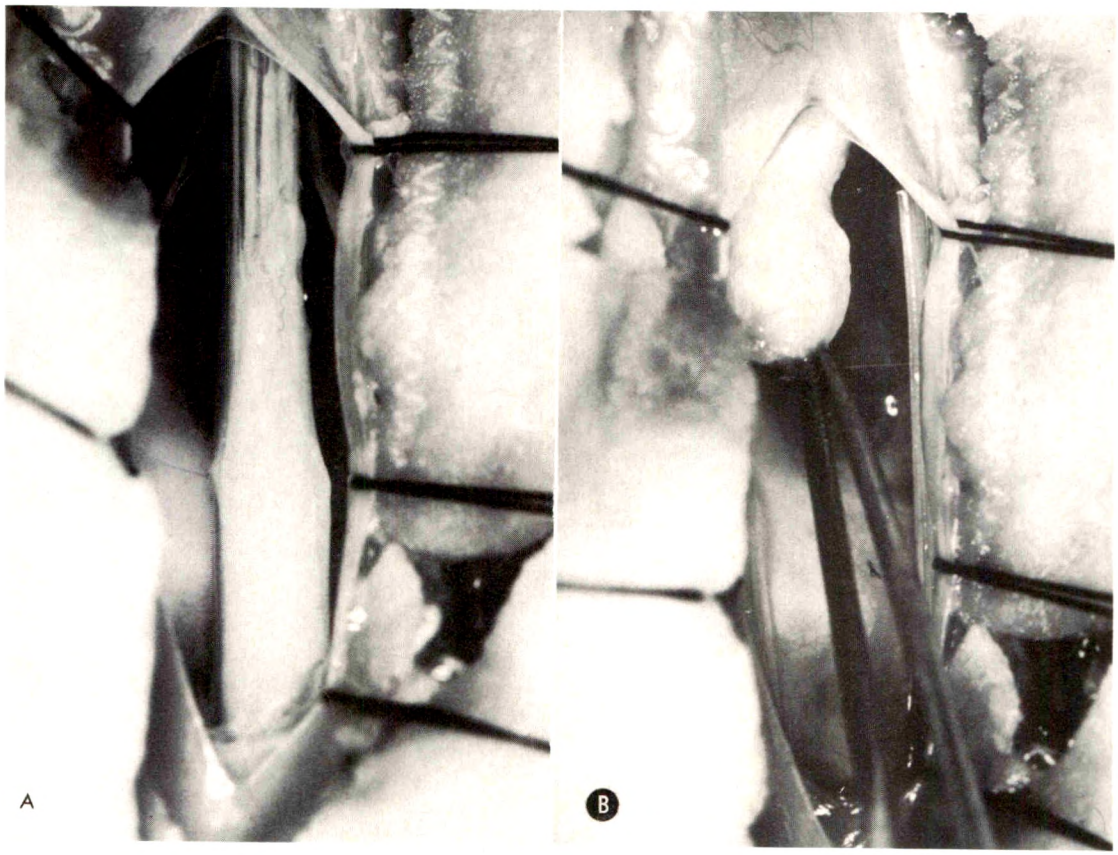
toms, plain film findings, and an associated tethered conus.

In air myelography the lateral view usually gives the best demonstration of the conus and filum (Fig. 8, *A* and *B*).

RESULTS

At operation the filum is identified, two adjacent silver clips are put across the distal end, and the filum is cut between the clips. The clips will usually separate 5 to 25 mm, often dramatically (Fig. 9, *A* and *B*). Postoperative spine films will confirm this separation (Fig. 10).

Operative results are somewhat hard to evaluate in a growing child whose neurological status is normally changing. Table II shows the improvement of individual



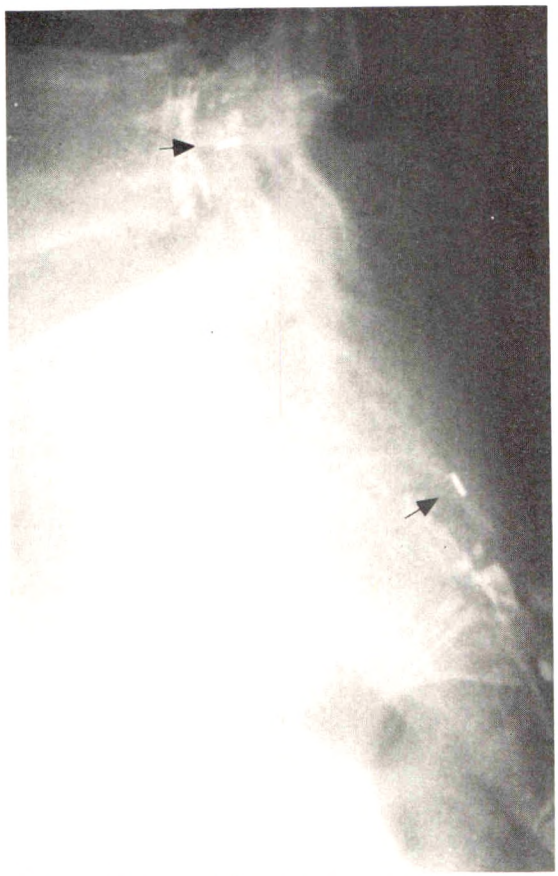


FIG. 10. Postoperative roentgenogram of another patient. Clips (arrows) have separated nearly two vertebral body spaces.

symptoms in our series. Over-all there is a definite improvement of at least some symptoms in our series. At worst there is a halting of symptom progression. Even children with bladder difficulties have improved. There seems to be a better improvement in those with only a tethering without associated intradural masses, possibly reflecting a lesser degree of abnormality.

DISCUSSION

James and Lassman,[9] early advocates of the tethered conus as an entity, now believe that other associated abnormalities, especially intradural or extradural bands, are the true cause of symptoms in these patients. Part of their belief is based on the findings at autopsy of a symptom free and

TABLE II

OPERATIVE RESULTS

MARKED IMPROVEMENT OF INDIVIDUAL SYMPTOMS

Incontinence	2/5
Other bladder problems	3/5
Sensory loss	4/8
Weakness	6/8

presumably neurologically normal 64 year old female having a diastematomyelia at L_4 and conus tethered caudal to this. Because the conus was at a low level in this "normal" individual, they discard it as a cause of symptoms in others. They do not continue the logical sequence and discard diastematomyelia as a cause of symptoms in others even though it was present in this case. Presuming that dural bands are the actual cause of the tethering, the diagnosis is still made by the myelographic finding of a low conus position.

The tethering of the conus either by bands or the filum itself does not give a clear indication of the symptoms. Most likely they are due to a local ischemia caused by stretching. Hilal and Keim's[6] recent publication concerning angiography in scoliosis suggests that tension on the cord may cause arterial stretching and possible ischemia. Autopsy studies also note this.[10] The possibility that stretching of the nerve fibers of the cortical spinal tracts occurs has also been postulated.[14] It has been experimentally shown that the effect of any local stretching of the cord is absorbed within a short distance above or below the level of the stretch.[1] This would account for the symptoms being localized to the lumbar and sacral roots.

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REFERENCES

1. BARRY, A., PATTEN, B. M., and STEWART, B. H. Possible factors in development of Arnold-Chiari malformation. *J. Neurosurg.*, 1957, *14*, 285–301.

2. Barson, A. J. Vertebral level of termination of spinal cord during normal and abnormal development. *J. Anat.*, 1969, *106*, 489–497.
3. Brickner, W. M. Spina bifida occulta. *Am. J. M. Sc.*, 1918, *155*, 473–502.
4. Garceau, G. J. Filum terminale syndrome. *J. Bone & Joint Surg.*, 1935, *35*, 711–716.
5. Gryspeerdt, G. L. Myelographic assessment of occult forms of spinal dysraphism. *Acta radiol.* (Diag.), 1963, *1*, 702–717.
6. Hilal, S. K., and Keim, H. A. Selective spinal angiography in adolescent scoliosis. *Radiology*, 1972, *102*, 349–359.
7. James, C. C. M., and Lassman, L. P. Spinal dysraphism: orthopedic syndrome in children accompanying occult forms. *Arch. Dis. Childhood*, 1960, *35*, 315–327.
8. James, C. C. M., and Lassman, L. P. Spinal dysraphism. In: Spina Bifida Occulta. Butterworth & Co., Ltd., London, 1972.
9. James, C. C. M., and Lassman, L. P. Diastematomyelia and tight filum terminale. *J. Neur. Sc.*, 1970, *10*, 193–196.
10. Jones, P. H., and Love, J. G. Tight filum terminale. *A.M.A. Arch. Surg.*, 1956, *73*, 556–566.
11. Kunitomo, K. Development and reduction of tail and of caudal end of spinal cord. *Contrib. Embryol.*, 1918, *8*, 161–198.
12. Love, G. J., Daly, D. D., and Harris, L. E. Tight filum terminale. *J.A.M.A.*, 1961, *176*, 115–117.
13. Robin, G. C. Scoliosis of spina bifida and infantile paraplegia. *Israel J. Med. Sc.*, 1972, *8*, 1823–1829.
14. Yashon, D., and Beatty, R. A. Tethering of conus medullaris within sacrum. *J. Neurol., Neurosurg. & Psychiat.*, 1966, *29*, 244–250.

ROENTGEN RECOGNITION OF ACUTE PLASTIC BOWING OF THE FOREARM IN CHILDREN*

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ABSTRACT:

Traumatic bowing of one or both forearm bones in children represents an acute plastic deformation of bone. Plastic curvatures have been produced in the dog ulna, but have been recognized only recently in man. The biomechanics and histologic changes of the experimental model explain the roentgenological features found in children with traumatically bowed bones: (1) broad fixed curvatures which hold fractures in angulation and resist orthopedic reduction; (2) absence of periosteal new bone formation following the injury; and (3) partial correction of the plastic curvature by cortical remodeling in young children. In a series of 17 patients, eight children had bowing of the ulna with an angulated fracture of the radius, five had bowing of the radius with an angulated ulna fracture, and four children had both the radius and ulna bowed without fracture. Manipulative reduction in the majority of children with fractures failed to reduce the plastic curvature, causing partial loss of pronation and supination.

ACUTE bowing of the forearm in children is a recently recognized condition.[2] The deformity represents a plastic response to longitudinal stress. Plastic curvatures of bone may be isolated or found in conjunction with conventional shaft fractures and dislocations. This report presents the roentgenographic appearance of plastic curvature of the forearm in children and summarizes the relevant biomechanical and orthopedic features.

MATERIAL

Seventeen cases of plastic bowing of the forearm in children are included in this study. Eight cases have been previously summarized.[2] Three cases were discovered in a review of roentgenograms of children with forearm fractures; clinical information and response to treatment were incomplete in these three patients. There were eight boys and nine girls, ranging in age from 2 4/12 to 13 8/12 years. A new bowing deformity was noticed by the patient, parent, or the examining physician in each case. In two patients, roentgenograms of an earlier radial metaphyseal fracture confirmed the normal appearance of the forearm before the acute bowing deformity (Fig. 3; and 4). The contralateral forearm was normal in all the five cases in which comparison roentgenograms of the uninjured forearm were obtained (Fig. 7).

Fractures were associated with the bowed bones in 13 of the 17 children. Eight chil-

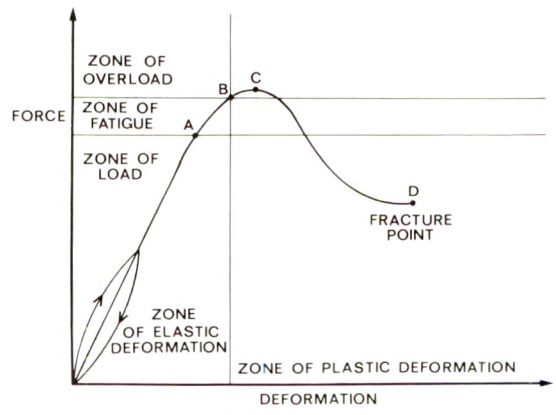

FIG. 1. Graphic relationship of bony deformation (bowing) and force (longitudinal compression), modified after Chamay. The linear response in the zone of elastic deformation and the weakening of bone in the zone of plastic deformation are demonstrated. Further details are in the discussion.

* From the Department of Radiology, Massachusetts General Hospital, Boston, Massachusetts.

TABLE I

SUMMARY OF PATIENTS

Case	Sex	Age	Injury	Fracture	Angulation of Distal Fracture Fragment	Location of Bowing	Convexity of Bow
M. D.	M	13-8/12	Fell down stairs	Midshaft left radius	Anterior	Midshaft ulna	Posterior
A. V.	F	2-4/12	Fell down on hand	Midshaft left radius	Anterior and lateral	Midshaft ulna	Posterior and lateral
S. H.	F	9-9/12	Fell down on hand	Midshaft right radius	Anterior and lateral	Midshaft ulna	Posterior and medial
B. S.	F	7-6/12	Fell onto hand	Midshaft left radius	Anterior and lateral	Midshaft ulna	Posterior and lateral
C. K.	F	6-3/12	Fell on arm	Midshaft left radius	Anterior	Midshaft ulna	Posterior and medial
K. M.	M	4-1/12	Fell off bike	Distal $\frac{1}{3}$ right radius	Anterior	Distal $\frac{2}{3}$ ulna	Posterior
D. K.	M	13-5/12	Fell off bike	Distal $\frac{1}{3}$ left radius	Anterior	Distal $\frac{1}{3}$ radius	Posterior
K. V.	M	7-3/12	Unknown	Midshaft left radius	Anterior	Midshaft left ulna	Posterior
N. B.	F	6-4/12	Fell down stairs	Midshaft right ulna	Medial	Midshaft radius	Lateral
P. B.	F	4-7/12	Fell off fence	Midshaft right ulna	Posterior	Midshaft radius	Anterior
R. P.	M	12-3/12	Fell off fence	Midshaft right ulna	Posterior	Midshaft radius	Anterior
B. W.	M	8-11/12	Fell on arm	Midshaft right ulna	Posterior	Midshaft radius	Anterior and lateral
A. B.	F	7-6/12	Fell on right hand	Midshaft right ulna	Posterior	Midshaft radius	Anterior
M. F.	M	8-8/12	Fell off bike	None	—	Midshaft radius and ulna	Posterior
D. B.	M	11-4/12	Fell off bike	None	—	Midshaft radius and ulna	Posterior and lateral
M. F.	F	6-0/12	Unknown	None	—	Midshaft radius and ulna	Posterior
C. K.	F	6-5/12	Fell on arm	None	—	Midshaft radius and ulna	Posterior and lateral

dren fractured the radius and bowed the ulna, and five children fractured the ulna and bowed the radius. Four children bent both the radius and ulna without fracture. The clinical information and type of injury in each patient is summarized in Table I.

DISCUSSION

All the children in this series had fallen on an outstretched arm, causing acute bowing. To understand the mechanism of this injury, a brief review of some biomechanics of bone is necessary.

Chamay[3,4] produced broad curving deformities of the dog ulna both *in vivo* and *in vitro*. Bowing deformities up to 30° were produced by resecting the radius of healthy dogs and allowing continued ambulation. Similar curves were produced with fresh wet specimens of dog ulna subjected to longitudinal compression. Histologic sections of the resulting curved bones produced in both ways showed multiple oblique microfractures where bone compression was greatest. The microfractures passed from the medullary cavity through the cortex to the periosteum, yet subperiosteal hemorrhage was not a feature. Using static force in a fresh wet preparation of a dog ulna, Chamay[3,4] obtained a stress-strain relationship (Fig. 1). The linear portion of the curve represents the elastic response. Bowing appeared and disappeared with the application and release of force, defining a hysteresis loop. With greater force, the linearity is lost and microfractures appear on histologic sections. With still greater force, more microfractures appear in the concavity of the bowed bone and a maximal strength is attained. Beyond this point, the bone becomes weaker, attains a dramatic curvature, and finally fractures at a substantially submaximal force.

The importance of these experimental data to the human patient is threefold. First, they demonstrate that the limit of an elastic response is not a fracture but rather a defined zone of plastic deformation. Second, the maximal strength of a

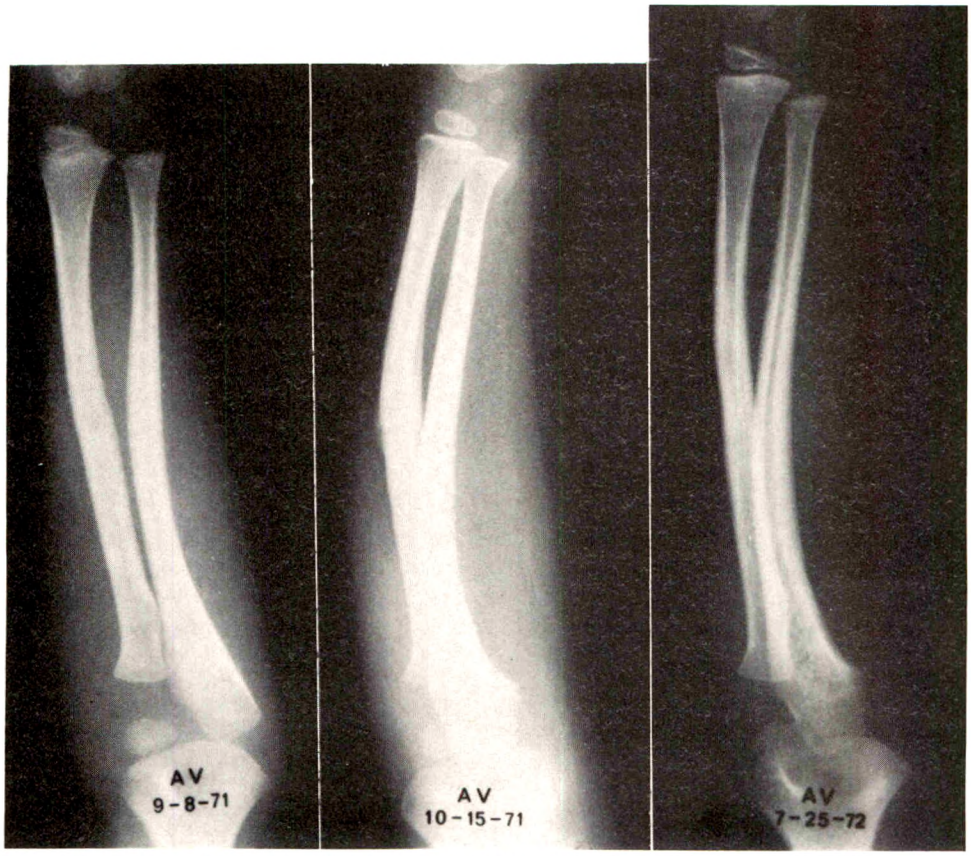

FIG. 2. Anteroposterior projection of the forearm of patient A. V. one week, six weeks and ten months after the injury. Cortical thickening in the concavity of the bowed ulna continues after complete healing of the midshaft radial fracture.

bone (Point C) was found to be approximately 100–150 percent of the dog's entire body weight. It is likely that a force of a similar magnitude is necessary both to produce and to reduce plastic curvatures in the human patient. Third, the duration of the deforming force is critical and is not represented in the stress-strain diagram. If the deforming pressure is continuously applied, every dog ulna will fracture once the maximal strength has been surpassed. However, a plastic curvature is produced if the deforming force is removed just before the fracture. Submaximal forces will cause elastic responses of the bone. Longitudinal forces greater than the maximal strength of the bones, which are applied for long periods, will cause angulated midshaft fractures of both the radius and ulna. Therefore, the relative rarity of plastic curvatures in human forearms is explained by the three criteria for a plastic deformation: (1) the deforming force must be exactly longitudinal (non-longitudinal forces are shearing forces and will cause the commonly seen epiphyseal and metaphyseal fractures); (2) the deforming force must be greater than the maximal strength of the bone; and (3) the duration of the force must be shorter than the time necessary to reach the point of fracture.

In 13 of the 17 children, fractures were associated with the bowed bones. A midshaft fracture of one forearm bone may release sufficient force to prevent fracture of the other bone, which instead is bowed. During a fall, pronation of the forearm with internal rotation at the shoulder will deliver maximal longitudinal force along the radius; a bowed ulna with an angulated

midshaft fracture of the radius may result (8 of 17 patients). Similarly, external rotation at the shoulder with forearm supination may cause a bent radius with an angulated midshaft ulnar fracture (five of 13 patients). A balance of axial forces along the radius and ulna may create two plastic curves without fracture. This is uncommon (four of 17 patients). It is apparent that bent bones are found with shaft fractures, but not with epiphyseal or metaphyseal fractures, which result from shearing forces.

A dislocation of the radius or ulna at the wrist or elbow may also release some longitudinal stress. Blount[1] has seen anterior dislocation of the radial head with a bowed ulna (Monteggia equivalent).

The roentgenologic aspects of bowing of the forearm in children include the recognition of the plastic deformity, comparison with the uninjured forearm, and confirmation of acceptable alignment after orthopedic reduction. A plastic curvature should be suspected in children with a clinical forearm deformity without fracture, an angulated shaft fracture of either the radius or ulna, or a dislocation of one bone at the wrist or elbow. It is important to obtain well exposed and correctly positioned roentgenograms of both forearms that provide anteroposterior projections of both the wrist and elbow in the frontal study and

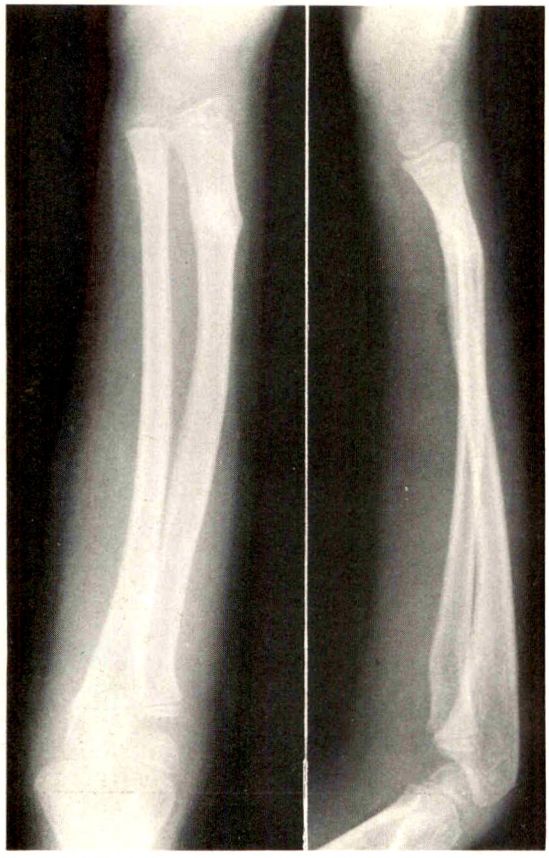

Fig. 3. Frontal and lateral projections of the left forearm of patient K. V. on August 29, 1968. The transverse fractures of the distal radius and ulna, sustained one month before, are healing.

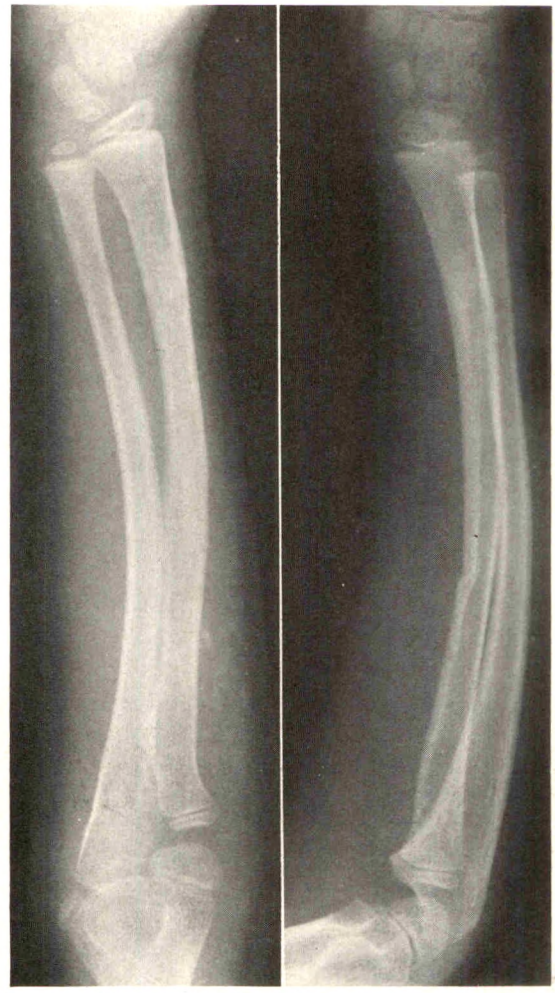

Fig. 4. Frontal and lateral projections of the left forearm of patient K. V. on May 16, 1969, obtained after a re-injury of unknown type. The old radial and ulnar fractures have remodeled and a new fracture is present in the midshaft radius. A new bowing curvature of the ulna can be seen.

FIG. 5. Frontal and lateral projections of the left forearm of patient D. K. obtained after a fall from a bicycle. The fracture of the radial metaphysis was seen, but the subtle bowing of the distal ulna was not recognized.

lateral view of both joints in the other. Occasionally, a child with a bowed forearm may have limitation of the complete supination required for the frontal projection, but diligence in positioning will provide a roentgenogram with minimal overlap of the radius and ulna. The lateral projection of the forearm usually shows the bowing best since most ulnar curvatures are convex posteriorly and most radial curvatures are convex anteriorly (Table 1). The accentuation of the intrinsic curvature of the forearm bones by longitudinal force accounts for the observed consistency in the direction of the radial and ulnar deformities.

Using comparison roentgenograms of the normal arm, subtle deformities may be discovered. Compound curvatures involving different areas of the bone may be recognized. Comparison views allow the measurement of curves to assess the orthopedic reduction.

Sequential roentgenograms of a plastic deformity uniformally reveal no periosteal new bone formation within two to four weeks of the injury. Plastic deformities are broad curvatures which involve the majority of the shaft and blend imperceptibly into the normal bone at either end. These two features distinguish this injury from a greenstick fracture. An unreduced bone is found to contain the original plastic deformity on follow-up roentgenograms (Fig. 5; and 6). In young children, delayed peri-

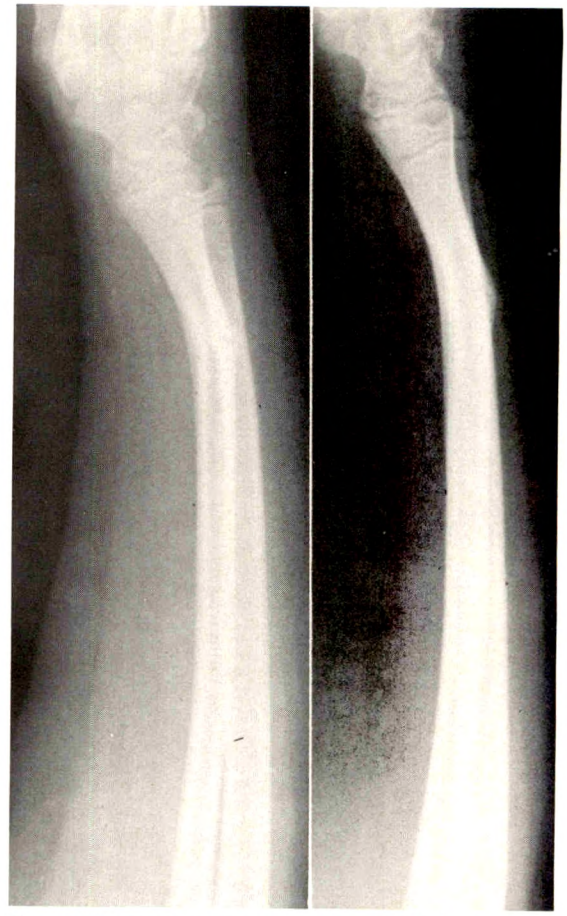

FIG. 6. Lateral projections of the left forearm of patient D. K. five weeks and eight weeks later. The bowing of the distal ulna remains. There is no periosteal reaction. The radial fracture continues to heal.

osteal bone formation is found along the concave surface of the bowed shaft, which slowly remodels (Fig. 2). Less remodeling occurs in older children, little occurs in the teenage patient, and no remodeling occurs in those cases where orthopedic reduction successfully reduced the bowing (Fig. 8). Delayed roentgenograms are necessary to document the adequacy of the orthopedic reduction.

Bowed forearm bones limit pronation and supination. A bowed bone remains bowed, resists attempts at reduction, holds an adjacent fracture in angulation, and prevents relocation of an adjacent dislocation. The force necessary to reduce a plastic deformity is the same as the force required to create it. It is likely that 100–150 percent of a child's total weight is required to reduce these curvatures. These forces are seldom attained by manipulative reduction, which consequently seldom changes the curvature of the bone. Orthopedic reduction in the operating room using vigorous manipulative force and general anesthesia is usually required for a successful reduction of plastic bowing of the forearm. A

Fig. 7. Lateral projections of the injured right and normal left forearm of patient M. F. There is broad bowing of the right radius and ulna with the convexity posterior. (Reprinted with permission of *J. Bone and Joint Surg.*, 1974, *56A*, 611–616.)

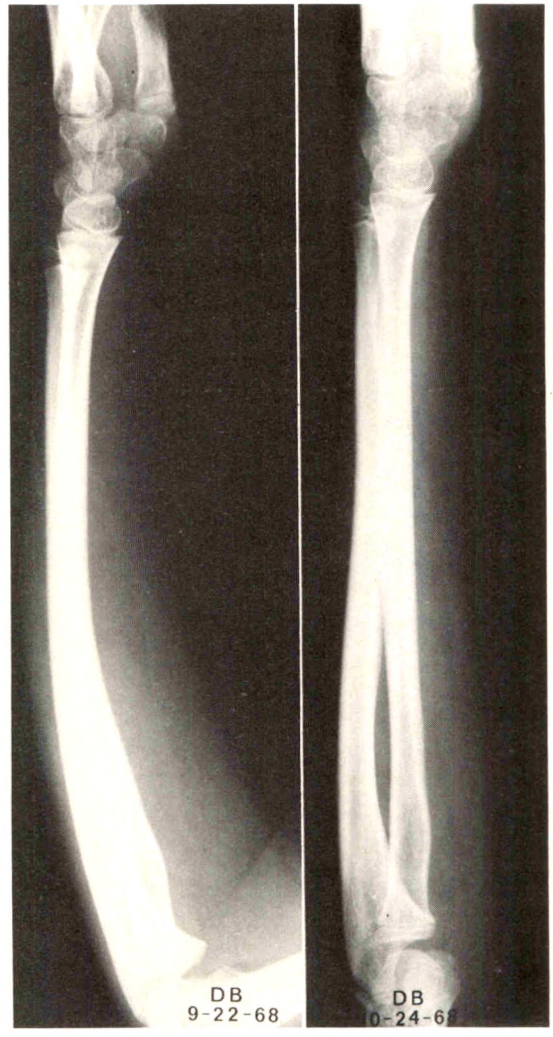

Fig. 8. Lateral projections of the forearm of patient D. B. before and one month after orthopedic reduction under general anesthesia of the bowing deformities of the radius and ulna. No periosteal reaction is seen on the second roentgenogram. (Reprinted with permission of *J. Bone and Joint Surg.*, 1974, *56A*, 611–616.)

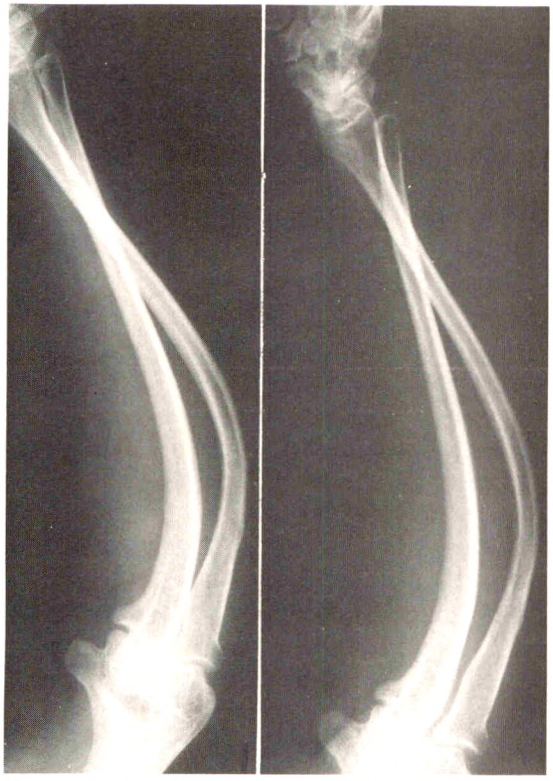

FIG. 9. Two projections of the right forearm of a 20 year old patient with osteogenesis imperfecta. This bowing curvature may be the result of chronic plastic deformation of pathologic bone.

drilling osteoclasis at the apex of the curvature has been proposed to weaken the bone sufficiently to facilitate reduction.[1]

In untreated or incompletely treated cases, bowed bones and angulated fractures may encroach upon the interosseous space, restricting pronation and supination. This commonly occurred in this series. Physiological remodeling of the bowed radial and ulnar shaft in young children may permit a complete range of pronation and supination, but restriction in rotatory movement of the forearm is nearly universal in older children with incompletely reduced plastic deformities.

Several corollaries of the concept of plastic curvature of the forearm in children are apparent. First, it is doubtful that the Salter V fracture of the forearm exists. This fracture presupposes the application of sufficient longitudinal force to injure the epiphyseal plate. As has been demonstrated in the dog and our patients, this force will produce a bowing curvature of the forearm or angulated midshaft fractures. Second, the concept may allow for the development of a plastic curvature of the normal forearm of an adult. In mature bone, it is likely that the slope of the elastic response is steeper and the range of plastic deformation is narrower than in a child or adolescent. However, a longitudinal force of the correct magnitude and duration should produce a plastic curvature. Third, if plastic curvatures occasionally occur in normal bone, it seems reasonable that they should occur more frequently in pathologic bone, because of the lower point of maximal strength, the decreased slope of the elastic response, and the greater range of plastic deformation before fracture. Progressive bowing of the limbs in patients with osteogenesis imperfecta or with extensive fibrous dysplasia illustrates two examples (Fig. 9). Possibly, the plastic curvatures discovered in patients with pathological bones may be corrected with the application of carefully graded reductive forces under general anesthesia, in a manner similar to the reduction of plastic deformities in normal children. Fourth, the lack of intrinsic curvature of the tibia may account for the apparent absence of plastic deformities in the lower leg. In contrast to the forearm, a Salter V fracture may result from a large compressive, longitudinal force.

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REFERENCES

1. BLOUNT, W. P. Personal communication.
2. BORDEN, S. Traumatic bowing of forearm in children. *J. Bone & Joint Surg.*, 1974, *56-A*, 611–616.
3. CHAMAY, A. Mechanical and morphological aspects of experimental overload and fatigue in bone. *J. Biomech.*, 1970, *3*, 263–270.
4. CHAMAY, A., and TSCHANTZ, P. Mechanical instances in bone remodeling: experimental research on Wolff's law. *J. Biomech.*, 1972, *5*. 173–180.

Vol. 125, No. 3

PNEUMATOCELES AS A COMPLICATION OF CHEMICAL PNEUMONIA AFTER HYDROCARBON INGESTION*

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ABSTRACT:

Of 338 children with hydrocarbon ingestion, 134 (40 percent) had pneumonia. Pneumonia was most common after kerosene, furniture polish and lighter fluid ingestion. The pneumonia was most severe, however, with furniture polish. Pneumatoceles occurred in 14 children, most commonly with charcoal lighter fluid ingestion. Pneumatoceles generally appeared late, after the consolidation had cleared. Pneumatoceles were often large, septate and irregular, and sometimes contained fluid levels.

CHILDHOOD poisoning in the United States remains frequent. In 1973 4,974 out of 17,589 poisonings under the age of five were due to petroleum products.[21] The principal complication of all petroleum distillate poisoning is a chemical pneumonitis, with fatalities occurring in four to ten percent of reported cases. Among various pulmonary complications, pneumatoceles have been considered relatively uncommon.[2] This paper describes a retrospective study of the roentgenographic and clinical findings in 338 children who ingested hydrocarbons. Pertinent data about the hydrocarbon pneumonias are outlined and the complication of pneumatoceles emphasized.

MATERIAL AND METHOD

Data were analyzed from 338 children who were treated for hydrocarbon ingestion at Cook County Children's Hospital during the period from March 1964 to December 1972, and who had chest roentgenograms available. Data were distributed according to the type of hydrocarbon ingested: furniture polish, lighter fluid, paint thinner, turpentine, gasoline, acetone, etc. Turpentine and pine oil, which are cyclic terpenes, were included according to the recommendations of the Committee on Accidental Poisonings,[19] since aspiration of these can cause chemical pneumonia.

RESULTS

Of 690 patients with known poisoning during the nine years from 1964 to 1972, 338 children had chest roentgenograms available. The incidence of pneumonia was greatest with kerosene (72 percent) and furniture polish (60 percent) (Fig. 1), and least with pine oil. The average age was under two years (Table 1). Several patients

TABLE I

AGES OF 134 PATIENTS WITH CHEMICAL PNEUMONIA

Substance Ingested	Number of Patients	Age of Patients	
		Mean	Range
		Months	
Furniture polish	58	17.7	3–42
Lighter fluid	25	26.9	14–62
Paint thinner	17	24.0	8–66
Kerosene	17	17.1	11–28
Turpentine	12	24.0	5–52
Miscellaneous	5	21.3	5–41
Total	134	20.8	3–66

* From the Department of Pediatric Radiology and Pediatrics, Cook County Children's Hospital and Hektoen Institute for Medical Research, Chicago, Illinois.

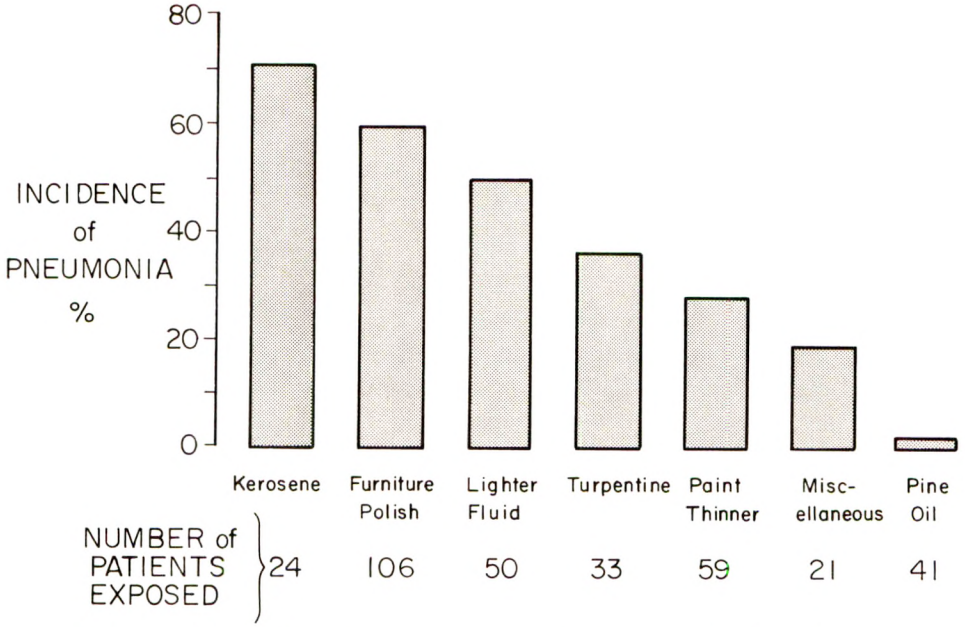

FIG. 1. Incidence of pneumonia in 338 children who ingested hydrocarbons.

under six months were fed hydrocarbons by siblings.

The roentgenograms of the children with roentgenographic evidence of pneumonia were graded according to maximum severity from mild to severe on a scale of one to four (Fig. 2). The more severe pneumonias occurred with ingestion of furniture

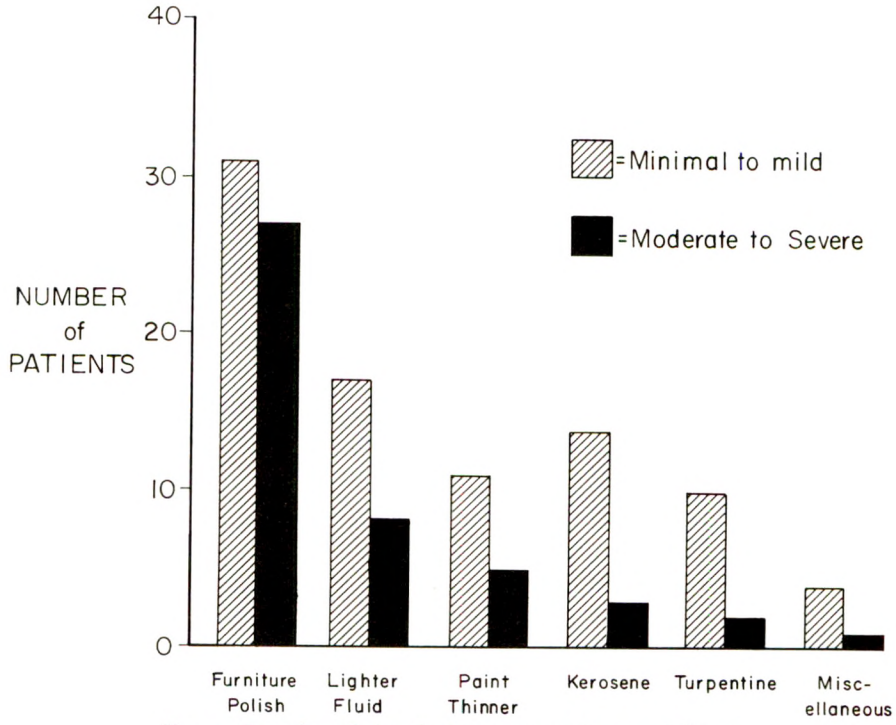

FIG. 2. Severity of chemical pneumonia in 134 children.

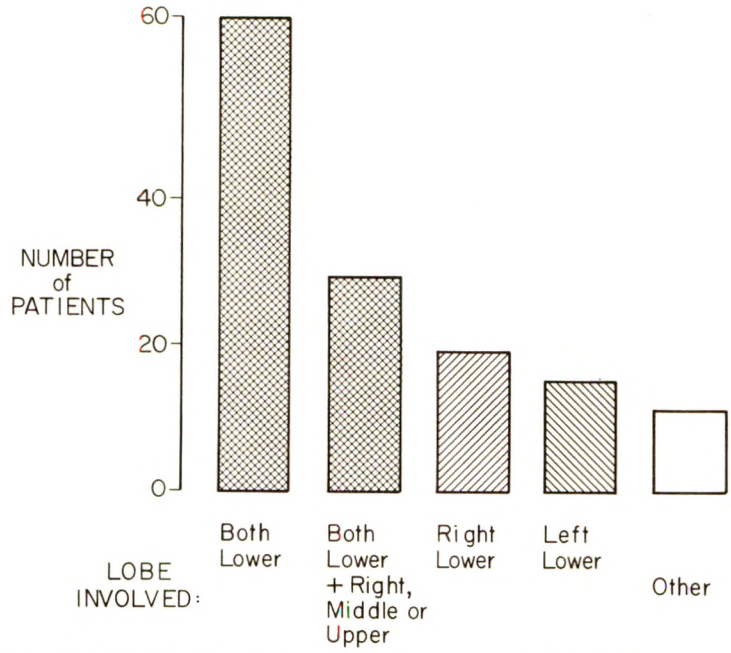

FIG. 3. Pulmonary lobe involved by chemical pneumonia in 134 patients.

polish (46.5 percent) while least severe with kerosene and turpentine ingestions. In every instance except three, at least one lower lobe was involved (Fig. 3), and in the majority, both lower lobes alone or in combination with right middle or upper lobes had pneumonic consolidations (66.4 percent). Three patients had involvement only of the right middle lobe.

The mean clearance time was 14 days for all chemical pneumonias excluding kerosene. Only three patients with kerosene ingestion were followed to clearing, which occurred within five days. Definite pneumatoceles occurred in 14 patients, due to ingestion of lighter fluid in six, paint thinner in four, furniture polish in two and

TABLE II

INCIDENCE OF PNEUMATOCELES IN PATIENTS WITH HYDROCARBON PNEUMONIA

Substance Ingested	Number of Patients	
	With Pneumatoceles	With Pneumonia
Furniture polish	2	58
Lighter fluid	6*	25
Paint thinner	4	17
Kerosene	1	17
Turpentine	0	12
Acetone	1	1
Miscellaneous	0	4
Total	14	134

* Significantly different from incidence of pneumatoceles with all other hydrocarbons ($P < 0.01$) and with furniture polish ($P < 0.001$).

TABLE III

INCIDENCE OF CYSTS, OTHER THAN PNEUMATOCELES, IN PATIENTS WITH HYDROCARBON PNEUMONIA

Substance Ingested	Number of Patients	
	With Cysts	With Pneumonia
Furniture polish	9	58
Lighter fluid	2	25
Paint thinner	2	17
Kerosene	1	17
Turpentine	3	12
Acetone	0	1
Miscellaneous	0	4
Total	17	134

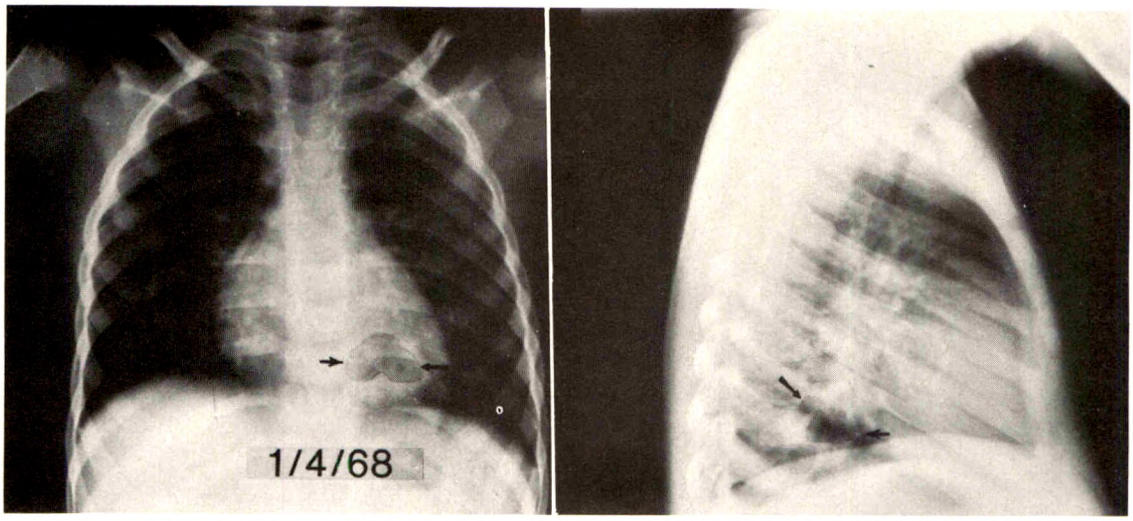

FIG. 4. Group of oval, somewhat irregular, pneumatoceles in left lower lobe in which fluid levels are present (arrow). An additional round pneumatocele is seen in the right middle lobe.

kerosene and acetone in one each (Table II). Chi square analysis of these data shows a significant difference of lighter fluid as compared with all other hydrocarbons ($P < 0.01$) and with furniture polish ($P < 0.001$). Tiny cysts not large enough to be classified as pneumatoceles appeared in 17 children, nine of whom had ingested furniture polish (Table III). Cardiomegaly occurred during the acute episode in two patients, and one patient died.

Many of the pneumatoceles became large, their diameter exceeding 10 mm in seven patients and 20 mm in three (Table IV). Many were irregular or oval rather than round (Fig. 4). Compartmentalization in pneumatoceles was common; in several cases the enlarging or coalescing areas of breakdown could be followed on serial films.

TABLE IV

DISTRIBUTION OF PNEUMATOCELES BY LONGEST DIAMETER

Diameter	Less than 10 mm	10 to 19 mm	20 to 32 mm	Total
Number of Patients	4	7	3	14

Fluid levels were seen in three cases (Fig. 5). Pneumatoceles usually appeared late, during or after clearing of the consolidation, with an average appearance time of 9.5 days and a range of two to 21 days. In four instances the pneumatoceles were visible on the first day of ingestion, although in each case they became more clearly visible as the consolidation cleared (Fig. 6). Three patients were followed more than 30 days after initial ingestion of hydrocarbon. Of these, two had not cleared the pneumatoceles by 41 and 42 days; one was cleared within six months (Fig. 7).

There were numerous instances of tiny cystic areas appearing in sites of consolidation. The majority of these measured between two and seven mm in diameter and were multiple (Fig. 8). Those followed to clearing took two to 15 days to disappear. There were two instances where preceding roentgenograms established the preexistence of these cysts. Most of them are part of the clearing process in the lung, possibly representing cleared groups of alveoli or acini. None progressed to frank pneumatoceles.

DISCUSSION

Pneumatoceles are cysts that originate

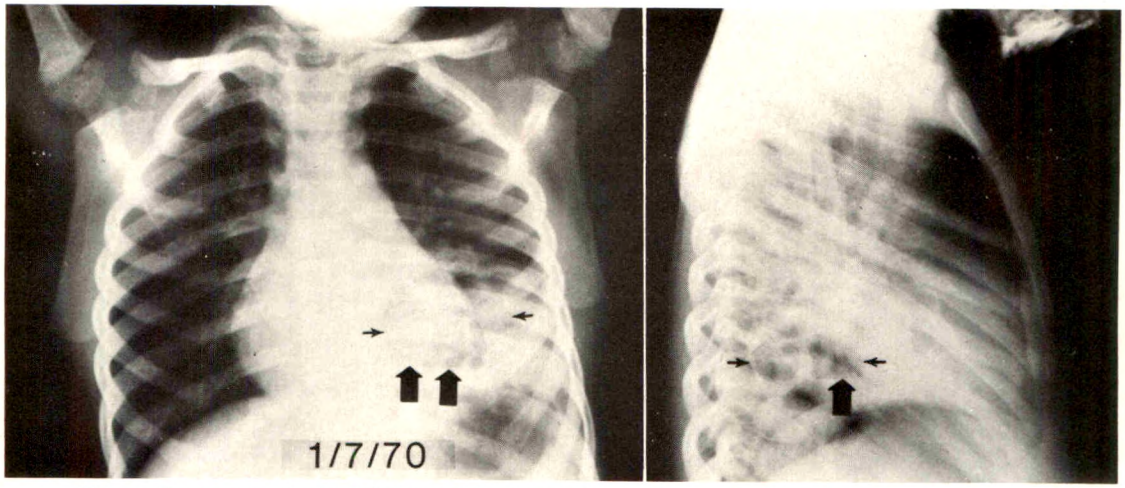

FIG. 5. Pneumatoceles in left lower lobe appearing 13 days after ingestion of paint thinner. Septa and fluid levels are present (arrows).

from a part of lung which has undergone necrosis and which is supplied by a bronchus having an edematous thickened wall. A check-valve mechanism results, which causes the cyst to accumulate air and, thereby, gradually enlarge. Pneumatocele is a well known complication of many pulmonary infections, the most common of which is that due to *Staphylococcus aureus*. It has also been described sporadically as part of hydrocarbon ingestion. In 14 cases of pneumatocele occurring in hydrocarbon-induced pneumonias, previously reported in the English and French literature,[2–4,6,14,15,18,20,22,23] involvement was always limited to lower lobe of one or both lungs; and pneumatoceles appeared only when the patients were clinically well, with the appearance time averaging 8.5 (range three to 15) days after ingestion.

Because of this and the small number of pneumatoceles reported since 1955, it has been suggested that it is either an uncommon event[3] or not detected because roentgenograms are usually no longer taken when the child is well and in the recovery phase of the illness.[18]

In our experience pneumatoceles were most frequently seen in ingestion of charcoal lighter fluid, paint thinner, or furniture polish. Only one example each in kerosene and acetone ingestions occurred,

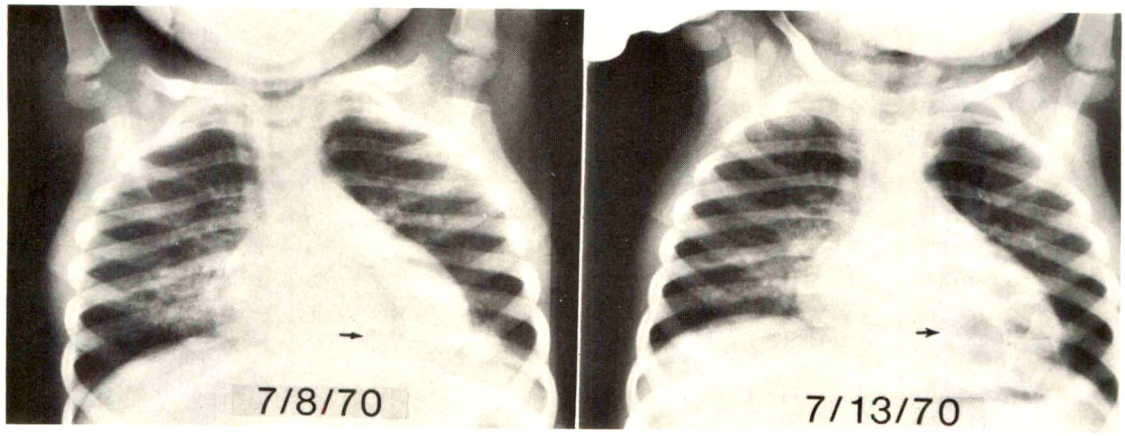

FIG. 6. On the first day, a cystic area communicating with the left lower lobe bronchus is visible (arrow) Five days later there is clearing of the consolidation and appearance of several pneumatoceles.

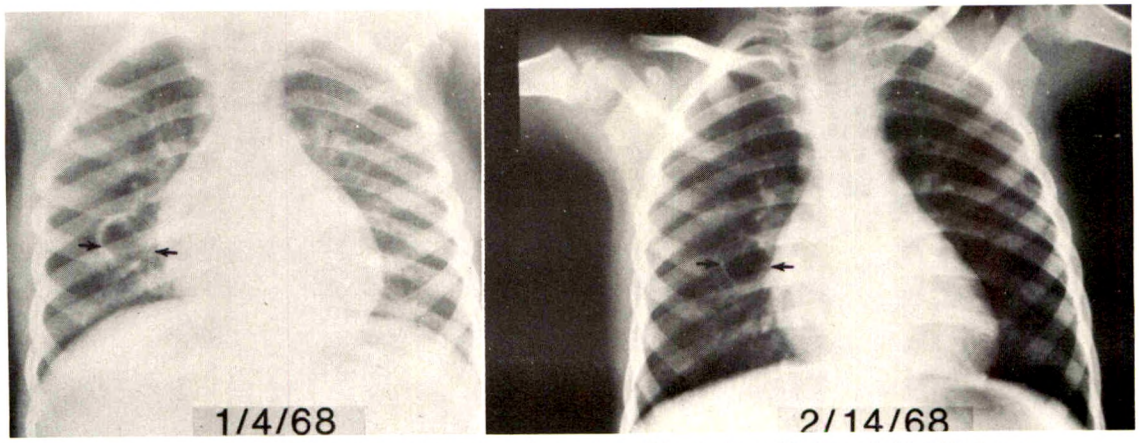

FIG. 7. A child followed for six weeks after onset of ingestion of lighter fluid still shows pneumatocele in right middle lobe (arrows).

and none among 33 cases of turpentine poisoning.

Interestingly, a relatively larger number of pneumatoceles occurred when the hydrocarbon ingested was charcoal lighter fluid, which is a petroleum distillate, naptha, with a low viscosity and surface tension. Gerarde's analysis[10] of the effect of hydrocarbon on the lung has demonstrated increased risk of aspiration and lung damage with decreased surface tension, decreased viscosity, and increased toxicity of the hydrocarbon aspirated. Low surface tension allows rapid spread while low viscosity permits penetration into distal airways. Formation of pneumatoceles suggests destruction of alveoli and pulmonary capillaries.[5,9,10] This is logical in view of the histopathology of lung described in children dying from hydrocarbon aspiration.[7,8,12,16] Findings include: hemorrhagic pulmonary edema; atelectasis; interstitial inflammation; necrosis of bronchial, broncheolar and alveolar tissue; vascular thromboses; a necrotizing bronchopneumonia with polymorphonuclear exudate; and formation of hyaline membranes lining the alveoli. These findings resemble hyaline membrane disease of the newborn. Animal studies of surface tension of pulmonary surfactant demonstrated a significant rise when intratracheal or intravenous furniture polish was administered. The lung is affected by resulting instability of alveoli with atelectasis and edema; distal airway closure also occurred.[11]

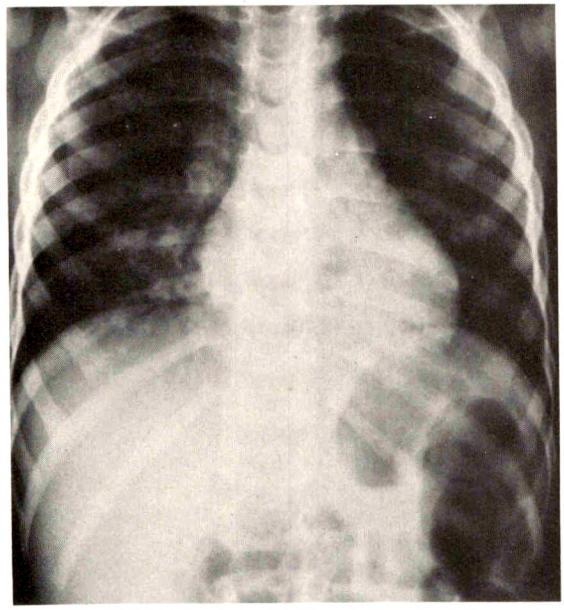

FIG. 8. Tiny round cysts in both lower lobes cleared on following examination.

It has been suggested on the basis of histological observation of destruction and repair of lung tissue in kerosene pneumonia in rats that lung parenchymal changes may persist for long periods, even after roentgenographic clearing.[1,13] The presence of pneumatoceles in this study, 40 to 60 days

after ingestion, tends to confirm this impression.

The pulmonary lesions are caused not by gastrointestinal absorption but by aspiration of the hydrocarbons. This conclusion arises from the results of numerous animal studies.[10,11,13,16,23] Factors involved include the small amount swallowed (estimated as a teaspoonful),[17] the rapid onset of symptoms, and the predominance of lower lobe involvement.

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REFERENCES

1. Ashkenaze, A. E., and Berman, S. E. Experimental kerosene poisoning in rats: use of C14-labelled hendecane as indication of absorption. *Pediatrics*, 1961, *28*, 642–649.
2. Baghdassarian, O. M., and Weiner, S. Pneumatocele formation complicating hydrocarbon pneumonitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *95*, 104–111.
3. Campbell, J. B. Pneumatocele formation following hydrocarbon ingestion. *Am. Rev. Resp. Dis.*, 1970, *101*, 414–418.
4. Castex, P., Courvalin, J. C., Foulon, E., and Faure, C. L. Pneumopathies bulleuses par ingestion de p'etrole (ou de ses derives). *J. de radiol., d' électrol. et de méd. nucléaire*, 1970, *51*, 716–707.
5. Done, A. K. Poisoning from common household products. *Pediat. Clin. North America*, 1970, *17*, 569–581.
6. Dragsted, P. J., and Rodbro, P. Pseudocysts of lungs in kerosene poisoning. *Dis. Chest*, 1965, *48*, 87–90.
7. Eade, N. R., Taussig, L. M., and Marks, M. I. Hydrocarbon pneumonitis. *Pediatrics*, 1974, *54*, 351–357.
8. Foley, J. C., Dreyer, N. B., Soule, A. B., Jr., and Woll, E. Kerosene poisoning in young children. *Radiology*, 1954, *62*, 817–829.
9. Gerarde, H. W. Toxicological studies on hydrocarbons: V. kerosine toxicol. *Appl. Pharmacol.*, 1959, *1*, 462–474.
10. Gerarde, H. W. Toxicological studies on hydrocarbons: IX. aspiration hazard and toxicity of hydrocarbons and hydrocarbon mixtures. *A.M.A. Arch. Environ. Health*, 1963, *6*, 329–341.
11. Giammona, S. T. Effects of furniture polish on pulmonary surfactant. *A.M.A. Am. J. Dis. Child.*, 1967, *113*, 658–663.
12. Griffin, J. W., Daeschner, C. W., Collins, V. P., and Eaton, W. L. Hydrocarbon pneumonitis following furniture polish ingestion. *J. Pediat.*, 1954, *45*, 13–26.
13. Gross, P., McNerney, J. M., and Babyak, M. A., Kerosene pneumonitis: experimental study with small doses. *Am. Rev. Resp. Dis.*, 1963, *88*, 656–663.
14. Guignard, J., Rousseau, L., and LeGouas, J. J. Pneumopathies de l'enfant par ingestion de petrole. *Ann. paediat.*, 1969, *45*, 1060/284–1065/289.
15. Gwinn, J. L., and Lee, F. A. Pneumatocele formation following hydrocarbon pneumonitis. *A.M.A. Am. J. Dis. Child.*, 1974, *127*, 875–886.
16. Huxtable, K. A., Bolander, R. P., and Klaus, M. Experimental furniture polish pneumonia in rats. *Pediatrics*, 1964, *34*, 228–335.
17. Jones, D. V., and Worth, C. E. Volume of swallow. *A.M.A. Am. J. Dis. Child.*, 1961, *102*, 427.
18. Neuhauser, E. B. D. Pneumatoceles following aspiration of hydrocarbons. *Postgrad. Med.*, 1971, *49*, 57–59.
19. Press, E. Co-operative kerosene poisoning study: evaluation of gastric lavage and other factors in treatment of accidental ingestion of petroleum distillate products. *Pediatrics*, 1962, *29*, 648–674.
20. Skarbeck, A. Pulmonary cysts following ingestion of household paraffin. *Arch. Dis. Childhood*, 1955, *30*, 457–459.
21. Tabulation of 1973 reports, bulletin. National Clearing House for Poison Control Centers, 1974, May–June.
22. Talwatte, S. N. B., and Soysa, P. E. Radiologic findings in poisoning from kerosene: observations made in childhood cases in Ceylon. *Clin. pediat.*, 1970, *9*, 422–424.
23. Wolfe, B. M., Brodeur, A. E., and Shields, J. B. Role of gastrointestinal absorption of kerosene in producing pneumonitis in dogs. *J. Pediat.*, 1970, *76*, 867–873.
24. Wolfe, R. E., Adams, F. H., and Desilets, D. T. Pneumatoceles complicating hydrocarbon pneumonitis. *J. Pediat.*, 1967, *71*, 771–814.

PULMONARY VENOUS AIR EMBOLISM IN HYALINE MEMBRANE DISEASE*

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ABSTRACT:

A newborn infant with the respiratory distress syndrome in whom pulmonary venous air embolism (PVAE) developed as a complication of positive pressure therapy is reported. The underlying pathophysiology in this disorder is probably the development of alveolar-capillary fistulae secondary to unduly high intrabronchial pressures. An increased awareness by radiologists and clinicians of PVAE as a potential complication of aggressive respiratory therapy will result in more frequent recognition of this uncommon but lethal disorder.

POSITIVE pressure respirators are commonly employed in newborn intensive care units to treat infants with respiratory distress. Two major attendant complications, pneumomediastinum and pneumothorax, occur frequently and are easily recognized. Their pathophysiology has been well elucidated by Macklin.[12] In contradistinction, pulmonary venous air embolism (PVAE), a dire complication, is only occasionally encountered. However, it should be considered whenever rapid deterioration of an infant on a respirator occurs. PVAE has been reported sporadically, both clinically and experimentally, the latter data suggesting that it may occur more commonly clinically than generally recognized, especially as a complication of mechanical respirators in neonates.[1,9,11,15,17,19]

REPORT OF A CASE

P.C., a one hour old premature female infant, was admitted to St. Paul Children's Hospital with severe respiratory distress. Physical examination at the time of admission revealed a 1,164 gram infant with multiple congenital anomalies including; bilateral cleft lip and palate; bilateral fifth digit skin tags; low set ears; and microcephaly. These findings were compatible with the clinical diagnosis of Trisomy 13–15 syndrome. The patient was cyanotic on 100 percent oxygen by mouth, and demonstrated grunting respiration with sternal retraction.

After the infant was intubated and placed on a PR-2 respirator, both umbilical arterial and venous catheters were inserted. Laboratory values at admission on 100 percent O_2 included pO_2 89, pCO_2 37.3, and pH 7.27. The admission chest roentgenograms demonstrated very severe hyaline membrane disease with no significant aeration of either lung (Fig. 1).

Because of a continuing decline in pO_2 the patient was placed on a Bourns volume respirator four hours after admission in an attempt to maintain adequate pulmonary ventilation. The delivered volume was maintained at 32 milliliters at a rate of 30 respirations per minute. Although the peripheral pulmonary pressure could not be accurately determined, the respirator generated a high pressure of 70–85 cm H_2O in its tubing. Despite use of the high pressure respirator, the pO_2 continued to decline to 48, and the pCO_2 increased to 46.6 at eleven hours after admission.

At 13 hours after admission, the patient suddenly developed blanching of the right arm and the heart rate decreased to 30–40 per minute. An attempt to withdraw blood for gas determinations yielded only air from both the umbilical arterial and venous catheters. Careful inspection demonstrated no leak in either of the catheter systems. An immediate roentgenogram revealed poor aeration of both lungs and extensive collections of air within the cardiac chambers, aorta, and in numerous major arter-

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ial and venous branches including the hepatic veins (Fig. 2).

Shortly thereafter the baby died. Postmortem roentgenograms made some eight hours after death showed complete resorption of the intravascular air. No cardiac or intravascular air was present at autopsy and no site of capillary-venous communication was demonstrable.

DISCUSSION

Pulmonary interstitial emphysema (PIE) results from alveolar rupture and usually precedes the complications of PVAE, pneumomediastinum, and pneumothorax. The high ventilatory pressures currently employed in many neonatal intensive care units to treat infants with severe respiratory distress, increase the probability of all three complications.[12,20] Spontaneous PVAE most likely results from formation of

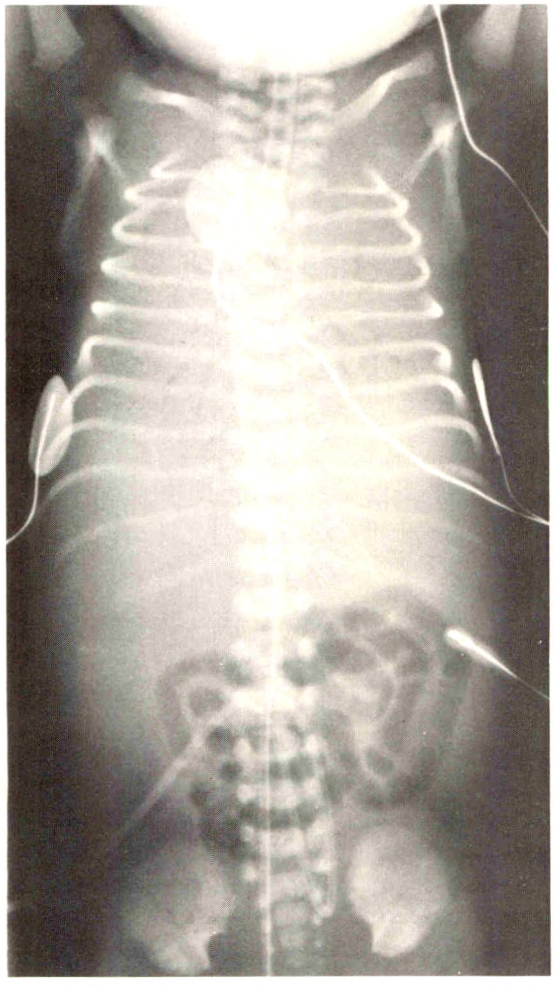

Fig. 1. Both lungs demonstrate the characteristic reticulo-granular pattern and extensive air bronchogram of severe hyaline membrane disease one hour following birth.

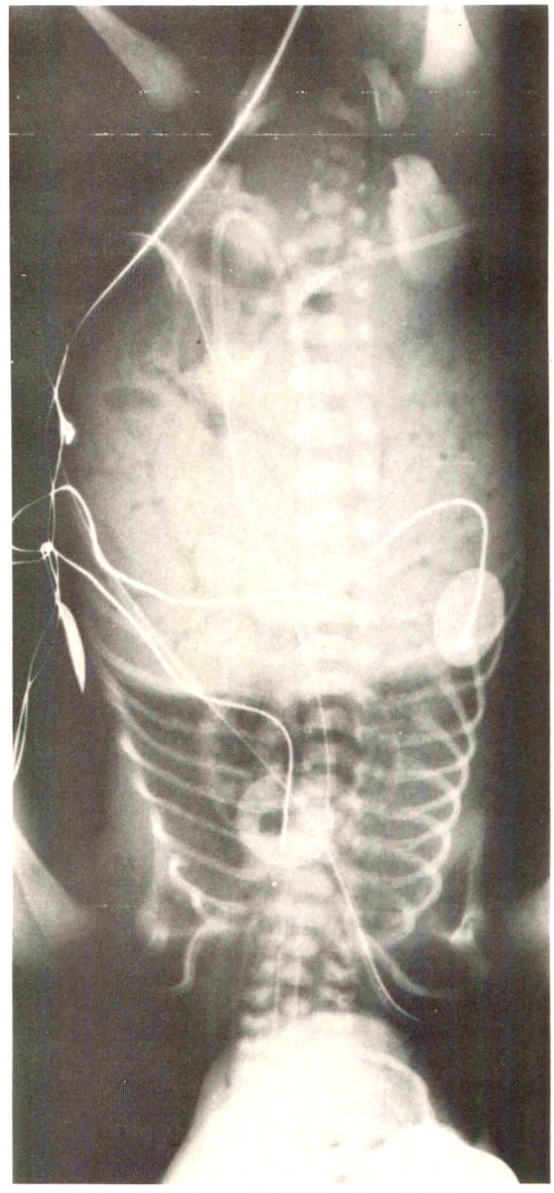

Fig. 2. A roentgenogram obtained at 13 hours of age demonstrates extensive air collections in the cardiac chambers. There is widespread hepatic vein air accumulation, quite different in distribution from that encountered in portal vein gas embolism. There is also gas in the mesenteric veins, left jugular vein, and in the subclavian vessels bilaterally. The increased radiolucency of both lungs is due to interstitial air.

alveolar-capillary fistulae, sometimes less correctly termed bronchovenous fistulae. Two major facts favor this site of fistulization after alveolar rupture: (1) the juxtaposition of blood and air, separated by only a few microns of endothelium, a common basement membrane, and the alveolar epithelium; and (2) a dense mesh of capillaries surrounding each alveolus.[4]

Normally the pulmonary venous pressure exceeds the bronchial pressure and this gradient must be reversed if PVAE is to occur.[3,11,13] As intrabronchial pressures increase, the pulmonary venous pressure also increases but at a slower rate, resulting eventually in reversal of the normal pressure gradient, and thus favoring PVAE should a fistula occur. PVAE does not depend on the absolute pressure present in the bronchioles, but on a transpulmonic (intrabronchial-intrapleural) pressure differential of 60–70 cm H_2O.[3] Perhaps concurrent pneumothorax, through a resultant increase in intrapleural pressure, may exert a therapeutic influence from this standpoint, although detrimental in other respects.

Although demonstration of the fistulous sites by postmortem microangiography should be possible, it has yet to be documented. Other evidence for alveolar-capillary fistulae exists, however: Macklin,[12] using a gelatin carmine particle technique, demonstrated sites of microscopic alveolar rupture in his investigation of interstitial air dissection. Later, the Macklins[13] postulated rupture could also easily occur into capillaries as well as into the neighboring interstitium. In addition, Lenaghan and co-workers'[10] acrylic bronchial casts of dog lungs with air embolization were normal, indicating that fistualization occurred distal to the terminal bronchioles. Further support for the existence of alveolar-capillary fistulae was provided by Bowen *et al.*,[2] who ventilated postmortem lungs and observed air bubbles traveling centripetally in pulmonary veins. They also noted that barium placed in the trachea was later demonstrable outside the bronchi in microscopic vessels and lymphatics.

PVAE is not a unique complication of infants on positive pressure respirators, but has also been observed after the strenuous respiratory efforts associated with whooping cough, asthma, and vigorous resuscitation procedures. It has been noted also following introduction of a needle into the chest for cautery pneumonectomy, irrigation of empyema cavities, artificial pneumothorax, and diagnostic pleural taps.[3,19] In the healthy lungs of submarine escape trainees, various degrees of air embolization may occur, presumably due to cyclic spurts of air passing through alveolar-capillary fistulae that open and close with respiration.[9,11] The noncompliant lungs of hyaline membrane disease, especially when splinted by interstitial air, promote continuous patency of any such fistulae which develop.[5,13] PVAE is also more common in shock lung, presumably for the same reason.[10]

PVAE is evidenced clinically by air in the retinal vessels, sharply defined areas of pallor on the tongue (Liebermeister's sign), marbling of the skin, and elevation of the blood pressure due to vasoconstriction from peripheral air emboli. In addition, air may be aspirated from umbilical catheters or may be seen bubbling from an incision over an area of cutaneous embolization.[5,18,16,19]

The symptoms, signs, and mode of death in PVAE are variable, depending upon the patient's posture at the time of embolization and shortly thereafter. Since intravascular air is relatively insoluble, air buoyancy becomes a major factor in determining the sequelae. If the patient is in the Fowler's position, air buoyancy causes cerebral air embolism, and neuromuscular sequelae dominate. On the other hand, if the patient is in the Trendelenburg position, air rapidly embolizes to the coronary arteries, and cardiovascular sequelae dominate, usually terminating in ventricular fibrillation. In the horizontal position encountered in most patients, both neuro-

muscular and cardiovascular sequelae occur and the mode of death is variable.[5,19] The described sequelae of intravascular air suggest that patient positioning could logically be employed as a mode of therapy for PVAE, but in most cases this is ineffective. With the patient in the Trendelenburg and left posteroanterior position, the coronary arteries and carotid arteries are both maximally protected from air embolization. Ideally this position should be assumed prior to embolization. However, since the air is sometimes transiently arrested in the aortic arch, and since PVAE may be sporadic rather than continuous, this position should probably be assumed whenever the diagnosis is suspected.[5]

Roentgenographically in PVAE, air may be demonstrable in all cardiac chambers and in the arteries and veins of both systemic and pulmonary circulations. This wide distribution of air can result from an intracardiac shunt or perhaps intravascular air can also traverse the pulmonary capillary bed, as in dogs, resulting in the occurrence of both right and left ventricular air.[19] Both portal and hepatic venous air may occur in PVAE. Differentiation is possible, although academic, since the prognosis is poor in either event. Hepatic vein air collects centrally in the upper region of the liver with the venous radicles converging toward the inferior vena cava. In contradistinction, portal gas is usually evident peripherally, but can occur centrally with the venous radicles converging more caudally in the region of the hepatoduodenal ligament. This distribution should not be confused with biliary air which tends to remain centrally located within the liver. Quisling *et al.*[14] recently described the rapid spontaneous accumulation of postmortem intravascular air in some infants with hyaline membrane disease and observed that the amount actually increased in the hours following death. Other authors, including ourselves, have noted that rapid absorption of the intravascular gas in PVAE may occur after death.[6,19,20] Since postmortem roentgenograms may be misleading, close correlation between the clinical and roentgenologic findings is of the utmost importance in establishing the diagnosis of PVAE.[18,19]

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Our special thanks to Mrs. Norma Brandon for assistance in preparation of the manuscript.

REFERENCES

1. Blumberg, N., and Latowsky, L. W. Cerebral air embolism, subcutaneous emphysema, and spontaneous pneumothorax in tuberculous patient: report of unusual case. *Dis. Chest*, 1940, *6*, 211–214.
2. Bowen, F. W., Chandra, R., and Avery, G. B. Pulmonary interstitial emphysema with gas embolism in hyaline membrane disease. *Am. J. Dis. Child.*, 1973, *126*, 117–118.
3. Chiu C-J., Golding, M. R., Linder, J. B., and Fries, C. Pulmonary venous air embolism: hemodynamic reappraisal. *Surgery*, 1967, *61*, 816–819.
4. Copenhaver, W. M. Bailey's Textbook of Histology. Fifteenth edition. Williams & Wilkins Company, 1964, pp. 448–449.
5. Durant, R. M., Oppenheimer, M. J., Webster, M. R., and Long, J. Arterial air embolism. *Am. Heart J.*, 1949, *38*, 481–500.
6. Gregory, G. A., and Rooley, W. H. Gas embolism in hyaline membrane disease. *New England J. Med.*, 1970, *282*, 1141–1142.
7. Grosfeld, J. L., Boger, D., and Clatworthy, H. W. Hemodynamic and manometric observations in experimental air-block syndrome. *J. Pediat. Surg.*, 1971, *6*, 339–344.
8. Gwinn, J. L., Lee, F. A., and Weinberg, H. Radiological case of month. *Am. J. Dis. Child.*, 1973, *126*, 63–64.
9. Kinsey, J. L. Air embolism as result of submarine training escape. *U. S. Armed Forces M. J.*, 1954, *5*, 243–255.
10. Lenaghan, R., Silva, Y. J., and Walt, A. J. Hemodynamic alterations associated with expansion rupture of lung. *Arch. Surg.*, 1969, *99*, 339–343.
11. Liebow, A. A., Stark, J. E., Vogel, J., and Schaefer, K. Intrapulmonary air trapping in submarine escape training casualties. *U. S. Armed Forces M. J.*, 1959, *10*, 265–289.
12. Macklin, C. C. Transportation of air along

sheaths of pulmonic blood vessels from alveoli to mediastinum. *Arch. Int. Med.*, 1939, *64*, 913–926.

13. Macklin, M. T., and Macklin, C. C. Malignant interstitial emphysema of lungs and mediastinum as important occult complication in many respiratory diseases and other conditions: interpretation of clinical literature in light of laboratory experiment. *Medicine*, 1944, *23*, 281–358.
14. Quisling, R., Poznanski, A., Roloff, D., and Borer, R. C. Postmortem gas accumulation in premature infants. *Radiology*, 1974, *113*, 155–159.
15. Rukstinat, C. J., and LeCount, E. R. Air in coronary arteries. *J.A.M.A.*, 1928, *91*, 1776–1779.
16. Schmidt, A. G. Portal vein gas due to administration of fluids *via* umbilical vein. *Radiology*, 1967, *88*, 293–294.
17. Segal, A. J., and Wasserman, M. Arterial air embolism: cause of sudden death in status asthmaticus. *Radiology*, 1971, *99*, 271–273.
18. Taylor, J. D. Postmortem diagnosis of air embolism by radiography. *Brit. M. J.*, 1952, *1*, 890–893.
19. Van Allen, C. M., Hrdina, L. S., and Clark, J. Air embolism from pulmonary vein. *Arch. Surg.*, 1929, *19*, 567–599.
20. Vinstein, A. L., Giesham, E. L., Lin, M. E., *et al.* Pulmonary venous air embolism in hyaline membrane disease. *Radiology*, 1972, *105*, 627–630

Vol. 125, No. 3

DROWNING: ANOTHER PLUNGE*

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ABSTRACT:

Hypoxia, pulmonary edema, acidosis, and aspiration compose the syndrome of near drowning. A review of 20 cases of near drowning indicates that the initial chest roentgenogram bears little weight in assessing the present or future clinical status. In some cases a 24 to 48 hour delay occurred before roentgenographic evidence for pulmonary edema was noted. The composition of fluid aspirated does not affect the outcome. The results of this report suggest that patients with a history of near drowning should be followed closely for at least 48 hours despite an initial normal chest roentgenogram.

THE most reliable clinical evidence for near drowning (*i.e.*, immersion or submersion in a fluid medium) is the presence of hypoxemia and metabolic acidosis. The chest roentgenogram is essentially noncontributory in determining the type of near drowning incurred or in defining the degree of pulmonary insufficiency. In a recent review of 20 cases of near drowning (six salt water, ten fresh water, four chlorinated water) (Table 1), the laboratory data, sequential chest roentgenograms, and mode of treatment conflict in predicting the outcome of the episode. Blood gases are the only means for determining the degree of pulmonary insufficiency. Furthermore, the onset of pulmonary insufficiency may occur in the ensuing 24 hours following the episode.

REPORT OF CASES

CASE I. D.C. This six year old white male was found in a chlorinated swimming pool, after submersion of approximately ten minutes. He was cyanotic and not breathing. He resumed spontaneous respirations after ten minutes of mouth-to-mouth respiration. Before arriving in the Emergency Room, he was noted to have vomited clear water. Upon arrival he was unresponsive and flaccid. Vital signs were normal except for labored respirations and a respiratory rate of 44. There were scattered diffuse rales and ronchi in both lung fields (Fig. 1). A chest roentgenogram revealed bilateral perihilar infiltrates. Blood gases showed a pH of 7.16, pCO_2 of 49, and pO_2 of 70, with nasal oxygen at four liters per minute. Serum electrolytes were normal except for a bicarbonate of 12.6. Blood gases and the roentgenograms returned to normal three days after admission.

CASE II. M.H. A 56 year old male capsized from his boat into water and was submerged for apparently three minutes. He was brought to

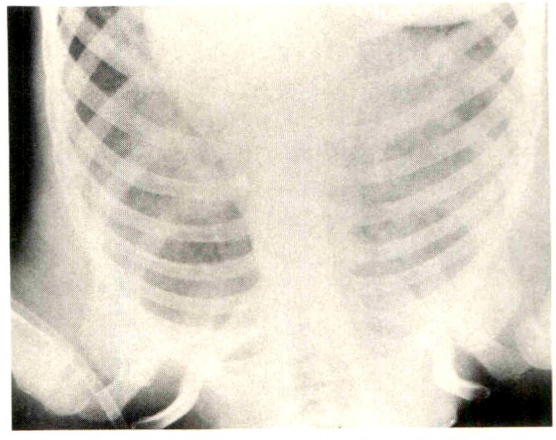

FIG. 1. D.C. Bilateral central haze indicative of alveolar fluid.

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TABLE I

Initials	Type of Immersion	Initial Blood Gases		Roentgen Pattern	Treatment	Complications
DC	Chlorine	pH 7.16 pCO_2 49 pO_2 70	24 hr. 72 hr.	initial—normal normal	Antibiotics, steroids	o
MH	Salt	pH 7.37 pCO_2 41 pO_2 40	24 hr. 72 hr.	initial—ill defined nodular densities, both bases; progressing basilar consolidation residual linear density right lower lobe, posterior basal segment	IPPB, steroids	o
SC	Salt	pH 7.20 pCO_2 40 pO_2 28	24 hr. 72 hr.	initial—bilateral alveolar densities consistent with pulmonary edema developed pneumonia, lung abscess, chronic fibrotic changes in both lower lung zones	IPPB, steroids, diuretics, antibiotics	Pneumonia, heart failure
HJ	Fresh	pH 7.31 pCO_2 44 pO_2 58	24 hr. 72 hr.	initial—bilateral diffuse density consistent with pulmonary edema persistent nodular peripheral density	Steroids, antibiotics	o
MS	Fresh	pH 7.12 pCO_2 50 pO_2 32	24 hr. 32 hr. 48 hr. 4 days 6 days	initial—normal bilateral diffuse alveolar densities consistent with pulmonary edema diffuse air-space consolidation residual markings, right lung normal	Steroids, antibiotics, volume respirator	o
GG	Fresh	pH 7.40 pCO_2 30 pO_2 91	24 hr. 72 hr.	initial—normal normal	24 hr. observation	o
GD	Fresh	pH 7.08 pCO_2 67 pO_2 20	24 hr. 72 hr.	initial—bilateral alveolar infiltrates consistent with pulmonary edema alveolar infiltrates for 7–8 days	Steroids, antibiotics, PEEP tracheostomy	Adult respiratory distress syndrome
CJ	Chlorine	pH 7.12 pCO_2 30 pO_2 77	24 hr. 72 hr.	initial—normal normal	Antibiotics, steroids, IPPB	o
AM	Fresh	pH 7.27 pCO_2 36 pO_2 98	24 hr. 72 hr.	normal normal	Oxygen mist	o
TY	Salt	pH 7.28 pCO_2 40 pO_2 54	24 hr. 72 hr.	initial—increased nodular density, right lower lobe, with linear markings in left lower lobe. Densities increased extending to perihilar region slight residual haze in right lower lung, posterior basal segment	Antibiotics	o
SB	Fresh	pH 7.43 pCO_2 25 pO_2 100	24 hr. 72 hr.	normal normal	24 hr. observation	o

PEEP = positive and expiratory pressure,
IPPB = intermittent positive pressure breathing.

Table I (*Continued*)

Initials	Type of Immersion	Initial Blood Gases		Roentgen Pattern	Treatment	Complications
ED	Fresh	pH 7.34 pCO_2 38 pO_2 260	24 hr. 72 hr.	initial—normal normal	48 hr. observation mannitol, decadron, antibiotics	o
BC	Chlorine	pH 7.31 pCO_2 36 pO_2 80	24 hr. 72 hr.	initial—minimal diffuse increased markings, slight prominence of pulmonary vascularity normal	24 hr. observation antibiotics	o
DM	Salt	pH 7.16 pCO_2 42 pO_2 124	24 hr. 72 hr.	initial—bilateral patchy infiltrates both bases none	Steroids, IPPB, antibiotics 48 hr. observation	o
JC	Salt	pH 7.31 pCO_2 41 pO_2 95	24 hr. 72 hr.	initial—hazy densities both bases, slight volume loss, left lower lobe normal	24 hr. observation	o
SC	Fresh	pH 7.31 pCO_2 36 pO_2 67	24 hr. 72 hr.	initial—increased markings, left lower lobe normal	IPPB steroids O_2 inhlaltion	Lung abscess
RS	Fresh	pH 7.39 pCO_2 37 pO_2 116	24 hr. 72 hr.	initial—patchy fluffy densities right and left lung normal	One week in hospital, steroids, antibiotics, IPPB, Thorazine, Nembutal, Dexamethasone, Gantrisin, Hypothermic blanket	o
KC	Salt	pH 7.35 pCO_2 34.5 pO_2 69	24 hr. 72 hr.	initial—density at both bases, bilateral perihilar haze, obliteration of central pulmonary vascularity normal	Steroids, antibiotics, IPPB, 3 day admission	o
MP	Chlorine	pH 7.15 pCO_2 70 pO_2 30	24 hr. 48 hr. 72 hr.	left upper lobe consolidation pulmonary edema pulmonary edema	Antibiotics, tracheostomy, volume ventilation	Anoxia-death
SJ	Fresh	pH 6.91 pCO_2 88 pO_2 26	24 hr. 72 hr.	bilateral diffuse infiltrates progressed to pulmonary edema persistent linear densities both lungs—clearing pulmonary edema pattern	Antibiotics, tracheostomy, volume ventilation	Hypoxic encephalpathy, perforated viscera, peritonitis, death

the Emergency Room slightly cyanotic with dyspnea and tachypnea. Vital signs were normal. Respiratory rales were noted throughout both lungs. He had peripheral cyanosis without clubbing. Neurological examination was within the limits of normal. Initial blood gases revealed a pH of 7.27, a pO_2 of 42, and a pCO_2 of 19. A chest roentgenogram showed basilar alveolar densities with pulmonary edema (Fig. 2*A*). Repeat blood gases obtained following treatment with intermittent positive pressure breathing (IPPB) and oxygen showed a pH of 7.37, pO_2 of 40, and a pCO_2 of 41. During this 24 hours after admission he was noted to have a grossly bloody sputum which was negative on culture. Three days after admission his blood gases were normal as was the chest roentgenogram (Fig. 2*B*).

Case III. S.C. A 65 year old male was found unresponsive in salt water after an indeterminate period of time. He required external cardiac massage and resuscitation and was brought semicomatose to the Emergency Room. He was intubated and a moderate amount of fluid suctioned from the lungs. Initial blood gases

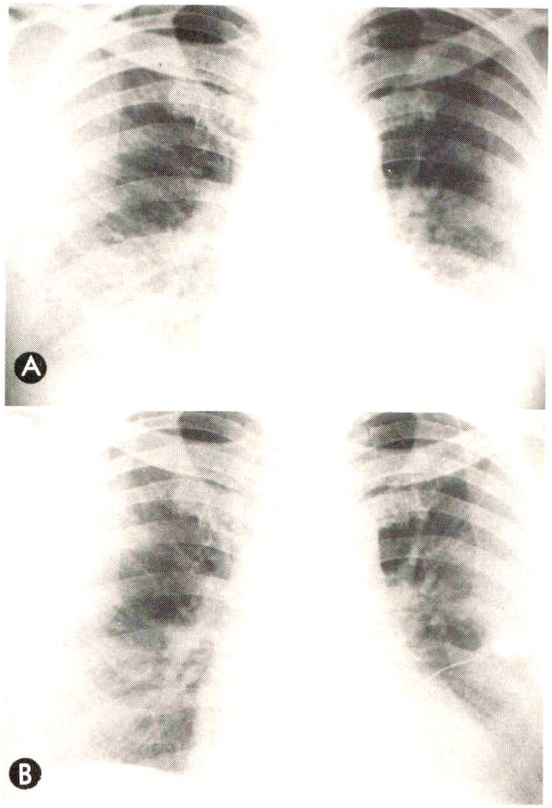

FIG. 2. M.H. (*A*) Bilateral basilar alveolar densities. (*B*) Clearing of infiltrate with return to normal.

showed a pH of 7.2, a pCO_2 of 40, a pO_2 of 28, and a bicarbonate level of 13. An initial chest roentgenogram showed linear densities extending from the hila consistent with interstitial edema (Fig. 3*A*). Despite IPPB treatment, steroids, diuretics, and antibiotics, pulmonary function continued to deteriorate. On the third day he required a tracheostomy. The hospital course was complicated by congestive heart failure, pseudomonas pneumonia and persistent pulmonary insufficiency. The patient was discharged two months after admission with blood gases showing a pO_2 of 64, a pCO_2 of 34, and a pH of 7.4 on room air. A follow-up roentgenogram two weeks later revealed right perihilar consolidation and left upper lung density consistent with pneumonia (Fig. 3*B*).

CASE IV. H.J. A 21 year old Black male was admitted to the Emergency Room after a near drowning episode in fresh water. He was apparently submerged for approximately two minutes. Arriving in the Emergency Room he was tachypneic and slightly lethargic. The vital signs were normal. There were fine crepitant rales over both lung fields. An initial roentgenogram showed a pattern consistent with pulmonary edema (Fig. 4). Blood gases showed a pO_2 of 58, a pCO_2 of 44, a pH of 7.31, and a serum bicarbonate of 20.5. He was treated with prophylactic steroids and antibiotics and was discharged within two days with normal blood gases. The chest roentgenogram showed clearing of the pulmonary edema with residual peripheral pulmonary densities.

CASE V. M.S. A 15 year old Black male was brought to the Emergency Room in a semicomatose state after about four minutes' submersion in a fresh water pond. The patient had vomitus in his mouth when found. His blood pressure was 130/80 mm Hg, pulse 120 per minute, and regular respirations 50 per minute.

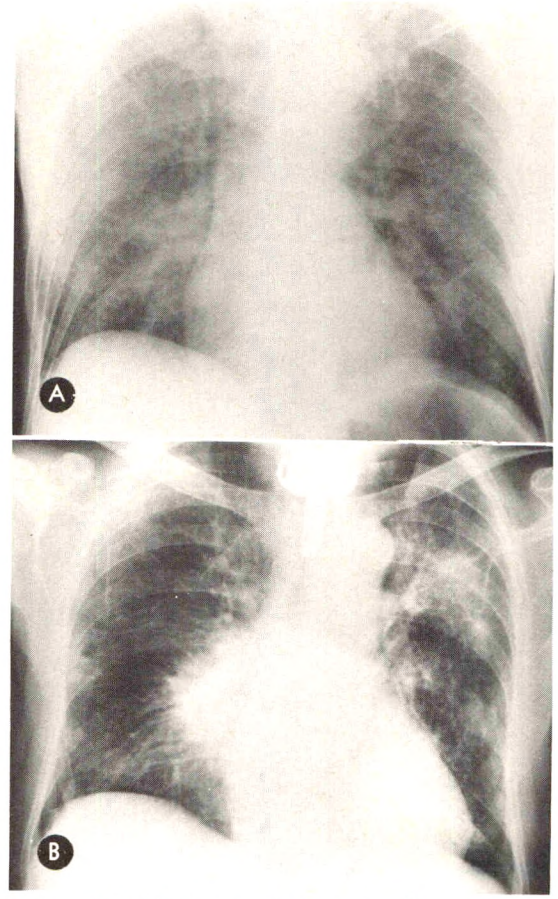

FIG. 3. S.C. (*A*) Scattered linear densities with central congestion. (*B*) Perihilar consolidation on right and left upper lung densities representing pneumonia.

His temperature was 100.6. There were diffuse ronchi and wheezes over both lung fields. No evidence of clubbing or cyanosis was present. An initial chest roentgenogram showed patchy densities throughout the right chest with minimal perihilar density on the left (Fig. 5*A*). Blood gases showed a pO_2 of 32, a pCO_2 of 50, and a pH of 7.12. The bicarbonate level was 17. A chest roentgenogram showed extension of the patchy infiltrates to both lungs consistent with pulmonary edema (Fig. 5*B*) six hours after admission. After utilization of the volume respirator the blood gases returned to a normal range six hours after admission. Antibiotics, steroids, bronchodilators and diuretic therapy were also used. The patient was discharged three days after admission with normal blood gases and a normal chest roentgenogram.

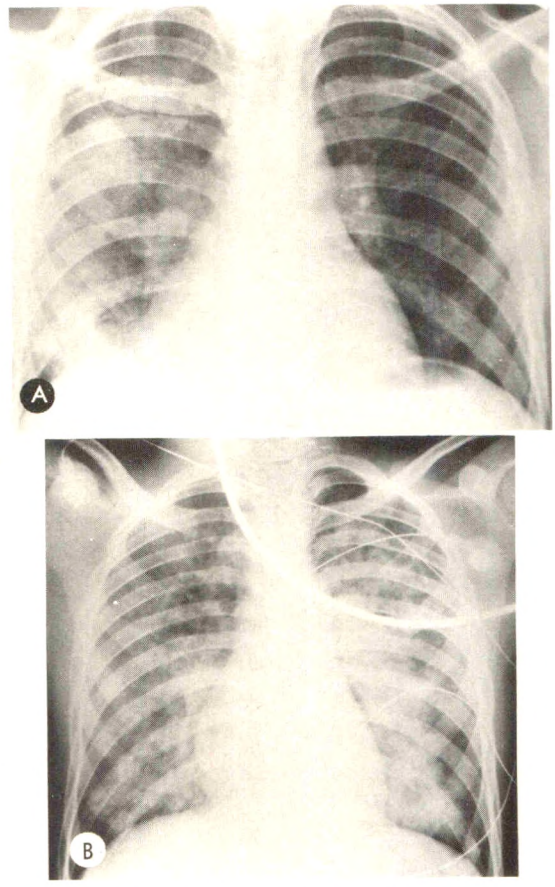

Fig. 5. M.S. (*A*) Patchy alveolar density in right lung. (*B*) Extension of density to left lung—compatible with pulmonary edema. Oxygen line over thorax.

DISCUSSION

The nature of the fluid aspirated appears to have little effect on the ensuing biochemical changes. Massive hemodilution, hemoconcentration, and hemolysis have been described in experimental animals following fresh and salt water near drowning.[6,7,12,13] Our study supports the observation of others that distinctive serum biochemical and hematological values have little relation to the type of fluid medium.[4,14] The following mechanisms may contribute to the development of pulmonary edema: prolonged hypoxia, respiratory obstruction, and fibrin degradation. Prolonged hypoxia causes shunting of unoxygenated blood from the arterial to the venous system accentuating the initial hypoxemia.[2,11] The roentgenographic finding of pulmonary edema related to hypoxemia is linked to the alteration in the permeability of the alveolar capillary membrane. In addition, respiratory obstruction has been shown to induce acute elevation of pulmonary venous pressure with resultant pulmonary edema in animals.[3] Fibrin degradation products have been observed in postmortem blood several hours after drowning. Therefore, it is conceivable that a subclinical consumptive coagulopathy with microembolization may further accentuate the

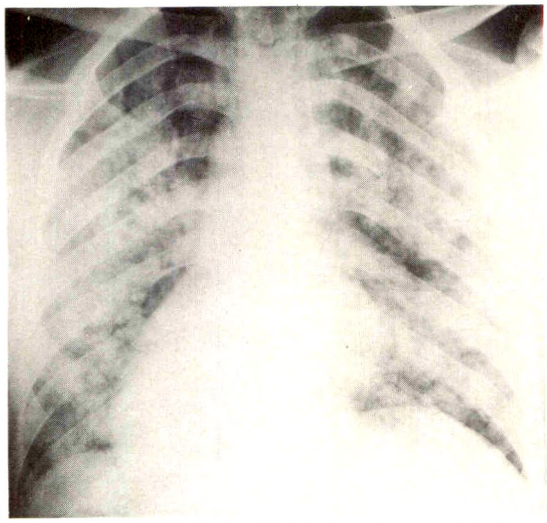

Fig. 4. H.J. Diffuse density in all lung zones consistent with pulmonary edema.

capillary leak.[8] All of the above factors may influence alveolar capillary membrane function by producing prolonged changes in the diffusion pattern, development of intrapulmonary shunts, and rare progression to an adult respiratory distress syndrome.[1] In our experience, the roentgenogram is not the critical indicator of clinical response which previous literature implies.[5,9,10] All near drowning victims may be severely hypoxemic while the initial chest roentgenograms appear normal. Moreover, several of our patients did not show abnormal chest roentgenograms before 24–48 hours. Fifty percent of the salt and fresh water cases showed irregular nodular infiltrates extending from the hila to the periphery. Near drowning in chlorinated water presented the same picture. None of the patients exposed to chlorine developed a delayed hypersensitivity chemical pneumonitis. Several cases demonstrated lobar or segmental densities, others had bilateral basilar infiltrates consistent with aspiration although these infiltrates cleared rapidly. Treatment appeared to have little affect on the roentgenographic appearance. Therapeutic measures included bronchodilators, antibiotics, steroids, and supportive therapy such as volume ventilators.

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REFERENCES

1. Fine, N. L., Myerson, D. A., Myerson, P. J., and Pagliaro, J. J. Near-drowning presenting as adult respiratory distress syndrome. *Chest*, *65*, 347–349.
2. Giammona, S. T. Drowning: pathophysiology and management. *Current Probl. Pediat.*, 1971, *7*, 1–33.
3. Haddy, F. J., Campbell, G. S., and Visscher, M. B. Pulmonary vascular pressure in relation to edema production by airway resistance and plethora in dogs. *Am. J. Physiol.*, 1950, *161*, 336–341.
4. Hasan, S., Avery, W. G., Fabian, C., and Sackner, M. A. Near drowning in humans: report of 36 patients. *Chest*, 1971, *59*, 191–197.
5. Hunter, T. B., and Whitehouse, W. M. Fresh-water-near-drowning: radiological aspects. *Radiology*, 1974, *112*, 51–56.
6. Kott, P. A. Drowning and near-drowning. *Family Physician*, 1974, *10*, 72–76.
7. Modell, J. H., Davis, J. H., Giammona, S. T., Moya, F., and Mann, J. B. Blood gas and electrolyte changes in human near-drowning victims. *J.A.M.A.*, 1968, *203*, 337–343.
8. Nopanitaya, W., Gambill, T. G., and Brinkhous, K. M. Fresh water drowning: pulmonary ultrastructure and systemic fibrinolysis. *A.M.A. Arch. Pathol.*, 1974, *98*, 361–366.
9. Romagosa, J. J., Menville, L. J., and Leckert, J. T. Radiographic changes in lungs during recovery from drowning. *Radiology*, 1950, *55*, 517–521.
10. Rosenbaum, H. T., Thompson, W. L., and Fuller, R. H. Radiographic pulmonary changes in near-drowning. *Radiology*, 1964, *83*, 306–312.
11. Segarra, F., and Redding, R. A. Modern concepts about drowning. *Canad. M.A.J.*, 1974, *110*, 1057–1062.
12. Swann, H. G., and Brucer, M. Cardiorespiratory and biochemical events during rapid anoxic death. *Texas Rep. Biol. & Med.*, 1949, *1*, 509–635.
13. Swann, H. G., and Spafford, N. R. Body salt and water changes during fresh and sea water drowning. *Texas Rep. Biol. & Med.*, 1951, *9*, 356–382.
14. Webster, J. Drowning: 1967. *General Med.*, 1968, *96*, 318–324.

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PULMONARY EDEMA AND CONTINUOUS POSITIVE PRESSURE BREATHING (CPPB)*

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ABSTRACT:

We have successfully treated severe pulmonary edema of various etiologies and the concomitant hypoxia in 17 infants and children with continuous positive pressure spontaneous breathing. The pressure used was eight to 14 cm of water above atmospheric pressure. No significant changes occurred in arterial or central venous blood pressure, and no patient developed clinical evidence of peripheral venous engorgement. Pulmonary edema cleared within 24 hours in all cases. We believe this rapid clearing may differentiate pulmonary edema from pneumonia and pulmonary hemorrhage. The pathophysiology and the roentgenologic findings are presented.

IN recent years, because of technical advances and greater understanding of the pathophysiology involved, continuous positive pressure breathing (CPPB) has been used more and more frequently to assist patients with respiratory difficulties from many causes. We have recently shown that the undesirable effects and complications of CPPB can be avoided if the pressure is kept low, between eight and 14 cm of water.[2,6] Furthermore, acute pulmonary edema can be successfully treated with CPPB when medical management fails.[2]

We present our additional experience with 17 infants and children who were successfully treated with CPPB for severe pulmonary edema of various causes.

MATERIAL AND METHOD

We do not use a respirator for CPPB. The gas is delivered through an endotracheal tube and the patient breathes spontaneously. During inspiration, the desired pressure is maintained at eight to 14 cm of water by regulating the gas flow through the endotracheal tube. During expiration, the positive pressure is created by having the child exhale through a water column eight to 14 cm high.[6] These mechanisms assure fairly constant pressure throughout the respiratory cycle. The pressure is constantly monitored by an oscilloscope.

Seventeen infants and children with severe pulmonary edema and concomitant progressive hypoxia were treated with CPPB. These patients had not responded to conventional measures, including oxygen, digitalis, diuretics, physical therapy, and a satisfactory airway. Eleven had developed severe pulmonary edema following cardiac surgery. The pulmonary edema was caused by upper airway obstruction in two children and accidental overhydration in two others. One had been submerged in salt water, while one had central nervous system ischemia from a large subdural hematoma following a skull fracture.

RESULTS

In each of the 17 infants and children, dramatic clinical and roentgenographic improvement occurred after applying CPPB. Roentgenographically the pulmonary edema cleared in two to 24 hours, with an average of seven hours (Fig. 1, *A* and *B*; and 2, *A* and *B*). The clinical improvement, which was slightly preceded by the roentgeno-

* Presented at the Annual Meeting of the Society for Pediatric Radiology, San Francisco, California, September 22–23, 1974.
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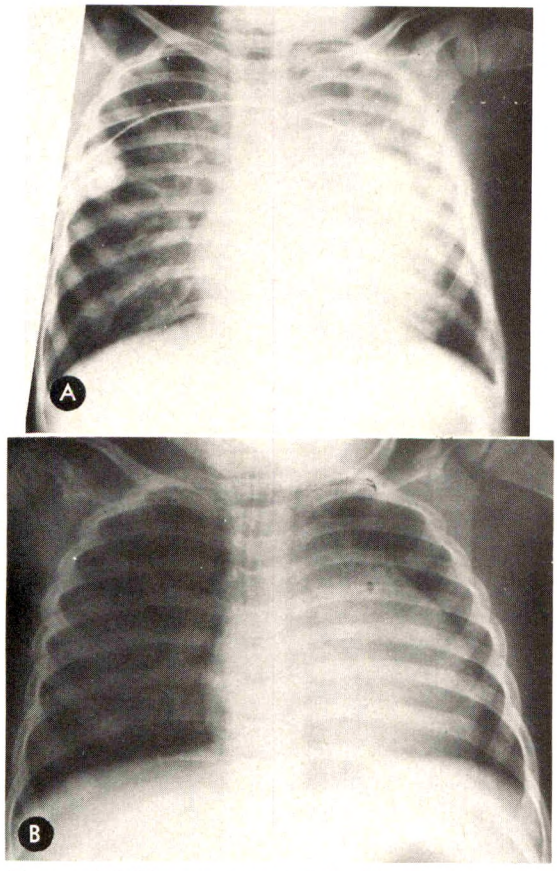

FIG. 1. (*A*) Twenty-two month old girl with hemolytic uremic syndrome was anuric for three days when she developed acute pulmonary edema causing progressive hypoxia. The chest roentgenogram shows acute pulmonary edema. (*B*) Repeat chest roentgenogram seven hours later shows complete clearing of pulmonary edema.

graphic clearing, was accompanied in all cases by a dramatic increase in pO_2 and decrease in pCO_2.

None of the patients developed interstitial emphysema, pneumomediastinum, pneumothorax, a significant change in arterial or central venous blood pressure, or clinical evidence of peripheral venous engorgement.

DISCUSSION

In general, we use CPPB whenever progressive hypoxia occurs despite adequate medical management and a good airway. CPPB appears to clear focal areas of atelectasis, to increase functional residual capacity and to correct regional abnormalities of ventilation and perfusion. With the improved oxygenation, acidosis decreases, production of surfactant is enhanced, and myocardial function improves. In the respiratory distress syndrome, and presumably in certain other conditions, the abnormally increased permeability of pulmonary capillary endothelium is corrected. The continuous positive pressure in the air-

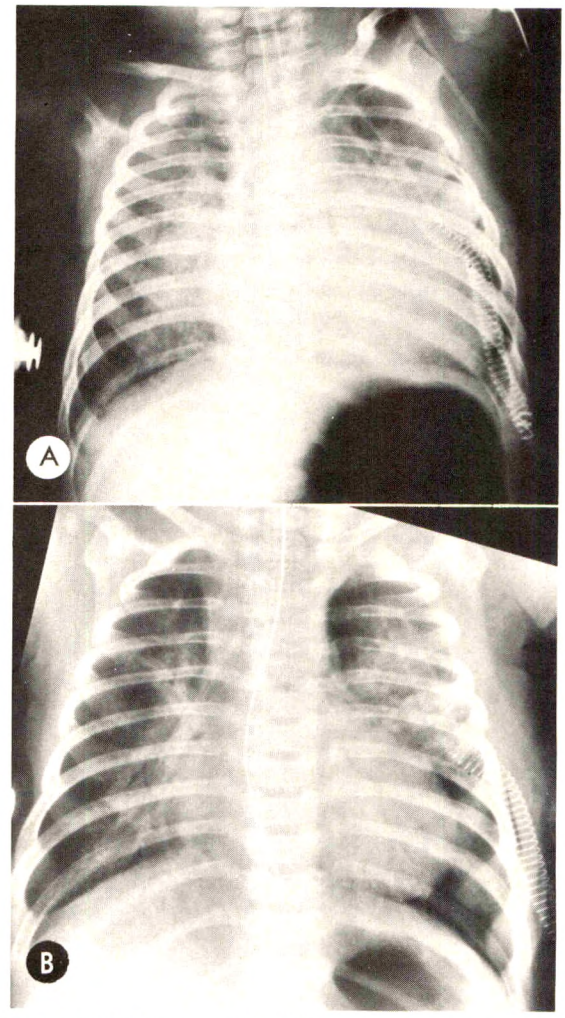

FIG. 2. (*A*) Three month old boy underwent pulmonary artery banding for ventricular septal defect. In spite of vigorous medical management including diuretics and digitalis, severe pulmonary edema persisted and caused progressive hypoxia. The anteroposterior chest roentgenogram taken immediately after he was put on CPPB shows severe pulmonary edema. (*B*) Six hours later there was almost complete clearing.

way apparently displaces fluid from the alveoli into the pulmonary capillaries.[6]

A number of complications has been reported from the use of positive pressure breathing, including: (1) pulmonary consolidation and collapse; (2) pulmonary interstitial emphysema; (3) pneumomediastinum; (4) pneumothorax; and (5) fluid retention.[5,7] These complications are uncommon if pressures are kept below 15 cm H_2O in both inspiration and expiration. With airway pressures of 26 to 28 cm water during expiration and 16 to 22 cm water during inspiration, however, central venous pressure has been shown to increase by a mean of 11 mm Hg. After 20 to 30 minutes, the cardiac index decreased from a mean of 3.7 L/min/sq M to a mean of 2.5 L/min /sq M and central blood volume fell as much as 50 percent.[3] In another study, however, the cardiac index remained normal despite high airway pressure, a mean of 23 cm water, and an end expiratory pressure of 13 cm water.[4]

With prolonged mechanical ventilation, water retention was reported in 19 of 100 patients. These 19 patients experienced a mean weight gain of 2.6 Kg. The level of positive pressure used was not reported.[7] In dogs, CPPB with an end-expiratory pressure of 10 cm H_2O has been shown to decrease both cardiac and urinary output and to increase plasma antidiuretic hormone (ADH).[1] We have not seen any of these changes in our clinical experience, which has been principally with infants and small children.

The positive pressure was set between eight to 14 cm H_2O above atmospheric pressure, a level we believe does not significantly influence cardiac output, urinary output, and antidiuretic hormone secretion. In all 17 infants and children treated, this low CPPB was sufficient to clear pulmonary edema rapidly. CPPB was added because of progressive hypoxia despite treatment with conventional measures. Since these patients were receiving diuretics, we did not anticipate a decrease in urine output, increase in plasma ADH, or water retention with CPPB. Indeed, urine output remained adequate and patients had no evidence of increased venous pressure or other signs of fluid retention. ADH levels were not measured.

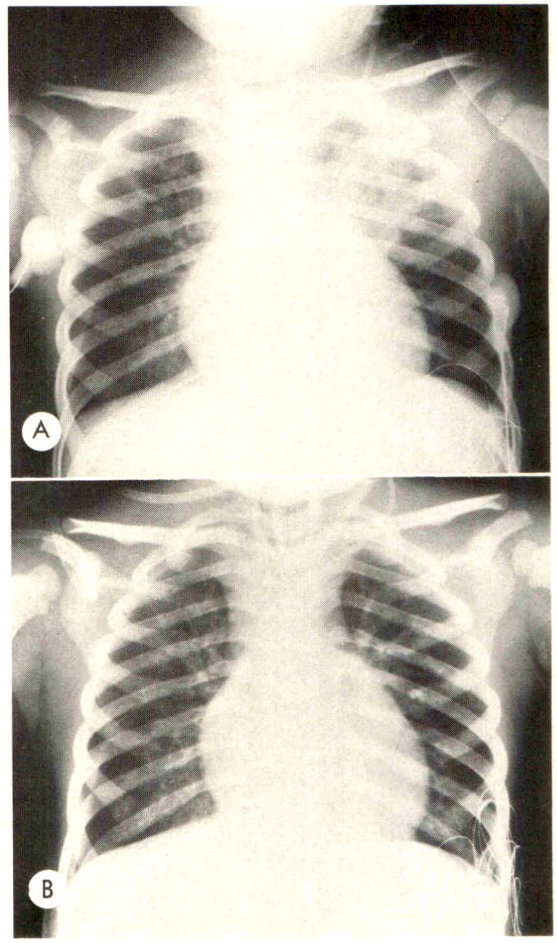

FIG. 3. (*A*) Four year old boy developed fever on the third postoperative day after repair of pulmonic valve stenosis. There are small areas of increased densities in the left upper lobe. The remainder of the lung is normal. (*B*) Fifteen hours after CPPB there was complete clearing. This indicates re-expansion of the small areas of atelectasis.

Because of the dramatic increase in pO_2, once CPPB was started, the oxygen concentration could soon be reduced, and the duration of oxygen administration was significantly shortened, thereby decreasing the possibility of oxygen toxicity.

In our experience, small foci of atelectasis clear as rapidly as pulmonary edema (Fig. 3, *A* and *B*). The rapid resolution of pulmo-

nary edema in these 17 patients led us to believe that pulmonary infiltrates that do not clear within 24 hours of CPPB therapy usually represent pneumonia, pulmonary hemorrhage, or large areas of atelectasis. Of course, further clinical-roentgenographic-pathologic correlations will be required to determine whether rapid clearing of pulmonary infiltrates with CPPB therapy reliably differentiates pulmonary edema from other lesions.

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REFERENCES

1. Baratz, R. A., Philbin, D. M., and Patterson, R. W. Plasma antidiuretic hormone and urinary output during continuous positive pressure breathing in dogs. *Anesthesiology*, 1971, *34*, 510–513.
2. Galvis, A. G., and Benson, D. W. Spontaneous continuous positive airway pressure (CPAP) breathing in management of acute pulmonary edema in infants. *Clin. Pediat.*, 1973, *12*, 265–269.
3. Kilburn, K. H., and Sieker, H. O. Hemodynamic effects of continuous positive and negative pressure breathing in normal man. *Circulation Res.*, 1960, *8*, 660–669.
4. Kumar, A., Falke, K. J., Geffin, B., Aldredge, C. F., Laver, M. D., Lowenstein, E., and Pontoppidan, H. Continuous positive pressure ventilation in acute respiratory failure (effects on hemodynamics and lung function). *New England J. Med.*, 1970, *283*, 1430–1436.
5. Macperson, R. I., Chernick, V., and Reed, M. Complications of respiration therapy in newborn. *J. Canad. A. Radiologists*, 1972, *23*, 91–102.
6. Oh, K. S., Stitik, F. P., Galvis, A. G., Bearman, S. B., Heller, R. M., and Dorst, J. P. Radiological manifestations in patients on continuous positive pressure breathing. *Radiology*, 1974, *110*, 627–630.
7. Sladen, A., Laver, M. B., and Pontoppidan, H. Pulmonary complications and water retention in prolonged mechanical ventilation. *New England J. Med.*, 1968, *279*, 448–453.

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ARTERIOMEGALY*

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ABSTRACT:

Arteriomegaly describes tortuous, ectatic, irregular vessels with prolonged blood flow. Seven cases of this entity are outlined with representative case reports. The increased incidence of aneurysms, thrombosis and embolization is noted in this and other series. The possibility that tortuous vessels may be confused with aneurysms on examination is also discussed. Due to the prolongation of blood flow, adequate arteriographic evaluation may be quite difficult.

ARTERIOMEGALY is the most facile descriptive term for a vascular disease consisting of varying degrees of arterial ectasia, tortuosity, and luminal irregularity with prolongation of blood flow, often in the absence of any actual occlusion. While this condition has long been accepted as a definite entity in Europe, the American literature has paid it little cognizance. The present series of seven cases, save one, represents the result of a survey of 75 aortograms and bilateral femoral arteriograms performed by direct translumbar and retrograde femoral technique over the course of two and one-half years at Newton-Wellesley Hospital. The clinical and roentgenographic findings of these seven cases are respectively recorded in Tables I and II. The following four representative case reports outline the range of findings in this interesting arterial disease.

REPORT OF CASES

CASE I. W.B. This 64 year old white male was admitted for evaluation of hematuria. Two and one-half years before he had had surgery for a bleeding left hypogastric aneurysm which was controlled, but apparently never completely resected. On intravenous pyelography the left kidney was hydronephrotic, and it was assumed the ureter was involved with scar from the same procedure. A translumbar arteriogram was then done which showed the aorta and iliac vessels to be dilated and tortuous with a large right hypogastric aneurysm (Fig. 1*A*). The superficial femoral vessels were dilated with heavy arteriosclerotic beading and aneurysmal dilatation of the right profunda femoris (Fig. 1*B*). The trifurcation vessels on the left side did not fill until 30 seconds after injection with only minimal filling of these structures on the right (Fig. 1*C*). A small popliteal aneurysm was noted on the left side and a thrombosed aneurysm was suspected on the right. An aorto-iliac graft was placed after resection of the distal aorta, common iliac, and bilateral hypogastric aneurysms with extension of the graft to the femoral level on the right because of excision of the profunda femoris aneurysm. Seven months later the patient returned with complaints of numbness and tingling of the right foot, and embolization was surmised from the suspected right popliteal aneurysm. A dumbbell-shaped aneurysm with clot was resected and a venous graft interposed. Despite his extensive surgical procedures, all were well tolerated and the patient's convalescence has been most satisfactory.

CASE II. A.L. This 64 year old white male had noted ischemic pain in his right leg for several months after an initial sudden onset. On physical examination a pulsating popliteal aneurysm was palpated on the left and a popliteal aneurysm with thrombosis and bleeding was suspected on the right side. On retrograde femoral arteriography the aorta, iliac and femoral vessels were irregular and ectatic with prolongation of blood flow. There was a small left hypogastric aneurysm (Fig. 2, *A* and *B*). There were fusiform popliteal aneurysms with distal occlusion on the right and adjacent filling

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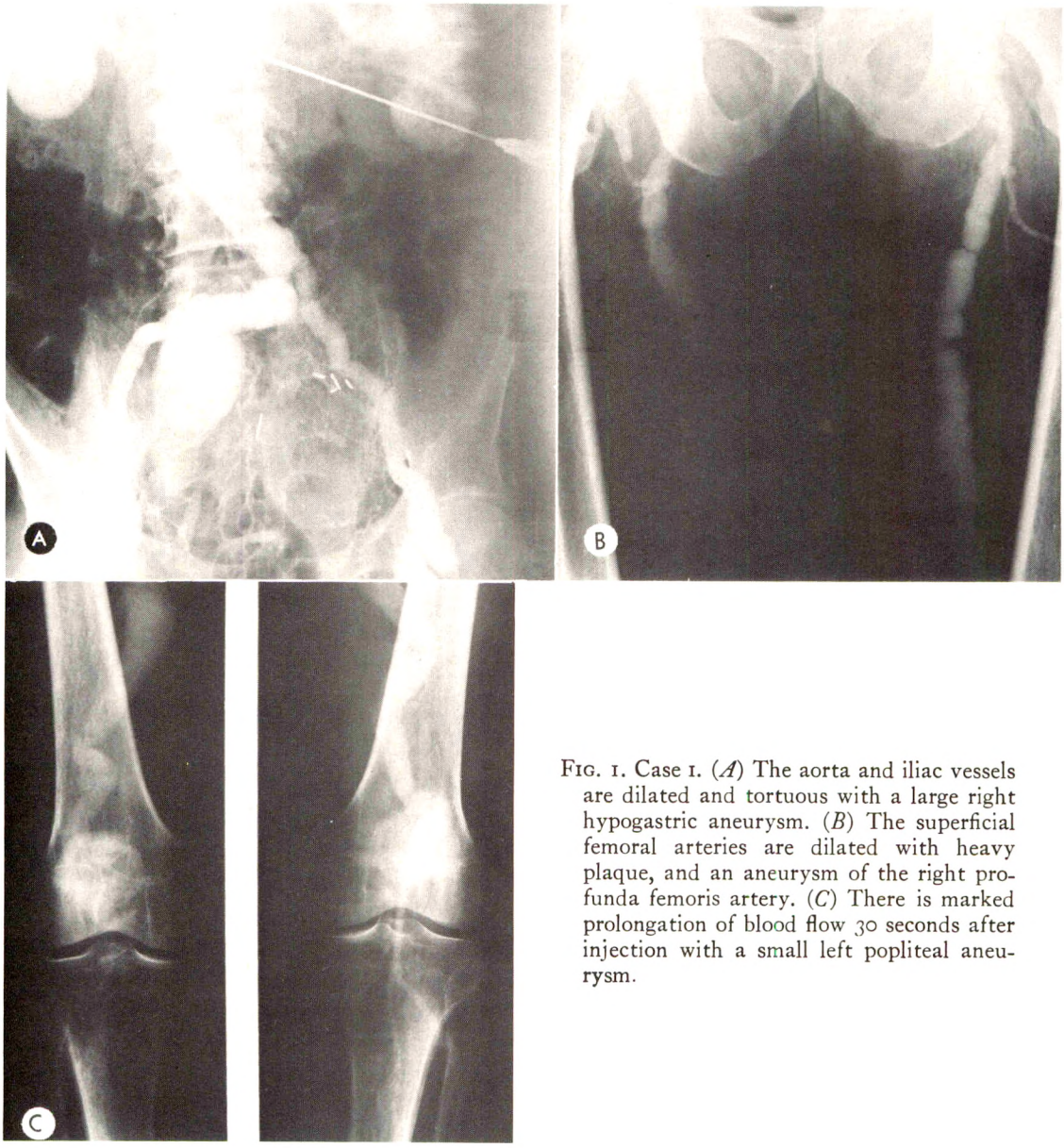

FIG. 1. Case 1. (*A*) The aorta and iliac vessels are dilated and tortuous with a large right hypogastric aneurysm. (*B*) The superficial femoral arteries are dilated with heavy plaque, and an aneurysm of the right profunda femoris artery. (*C*) There is marked prolongation of blood flow 30 seconds after injection with a small left popliteal aneurysm.

defects suggesting fresh blood clot. In addition, an area of faint extravasation of contrast medium was noted lateral to the right popliteal aneurysm and slightly obscured by the bone (Fig. 2*C*). The right popliteal aneurysm was resected and a saphenous vein graft placed. Following the procedure the patient had return of the dorsalis pedis pulse. Recovery was uneventful.

CASE III. L.B. This 69 year old white male was noted to have a large abdominal aortic aneurysm on physical examination. His initial chest roentgenogram showed a strikingly tortuous, ectatic thoracic aneurysm (Fig. 3*A*). Evaluation was desired for possible resection of the abdominal aortic aneurysm, and a retrograde femoral arteriogram was done. A large aortic aneurysm, 6 cm in diameter, was shown in virtual continuity with the thoracic aneurysm and included the right renal artery, the left being unsatisfactorily delineated (Fig. 3*B*). The tortuous aneurysm also extended into large tortuous iliac aneurysms. Distal roentgenograms were not obtained as the information as to unresectability had already been secured

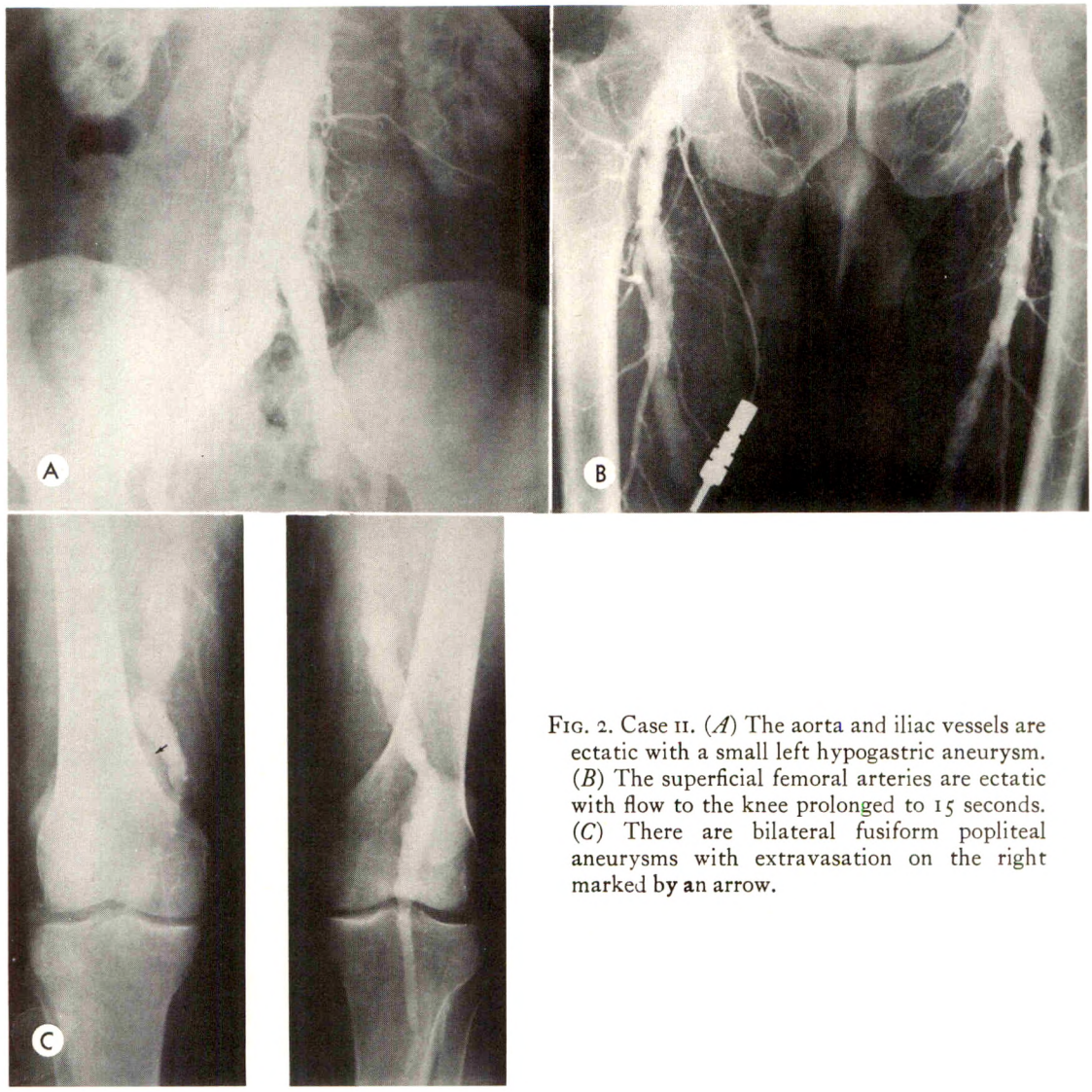

Fig. 2. Case II. (*A*) The aorta and iliac vessels are ectatic with a small left hypogastric aneurysm. (*B*) The superficial femoral arteries are ectatic with flow to the knee prolonged to 15 seconds. (*C*) There are bilateral fusiform popliteal aneurysms with extravasation on the right marked by an arrow.

and the prolongation of a technically difficult procedure was not desired. The patient was discharged without surgical treatment and later succumbed to a pulmonary embolus.

Case IV. A.M. This 81 year old white female was admitted with crampy abdominal pain of ten days' duration. While some improvement was noted after treatment for diverticulitis, there was a persistence of pain in the left lower quadrant. A pulsatile mass was palpated in this same area and arteriography was requested to rule out an iliac aneurysm. On translumbar arteriography strikingly tortuous iliac and aortic vessels were delineated, particularly the left external iliac (Fig. 4). Only mild prolongation of flow and ectasia of the arteries were noted, the distal vessels being patent. The patient's symptoms gradually subsided and she was discharged as a probable acute sigmoid diverticulitis.

DISCUSSION

In 1943 and 1947 Leriche[10,11] described the clinical, roentgenographic, and operative findings in two patients with tortuous, dilated iliac and common femoral arteries that were diagnosed as aneurysms preoperatively. These were described by the term "dolicho et méga-artère." Since then numerous other reports of this entity have

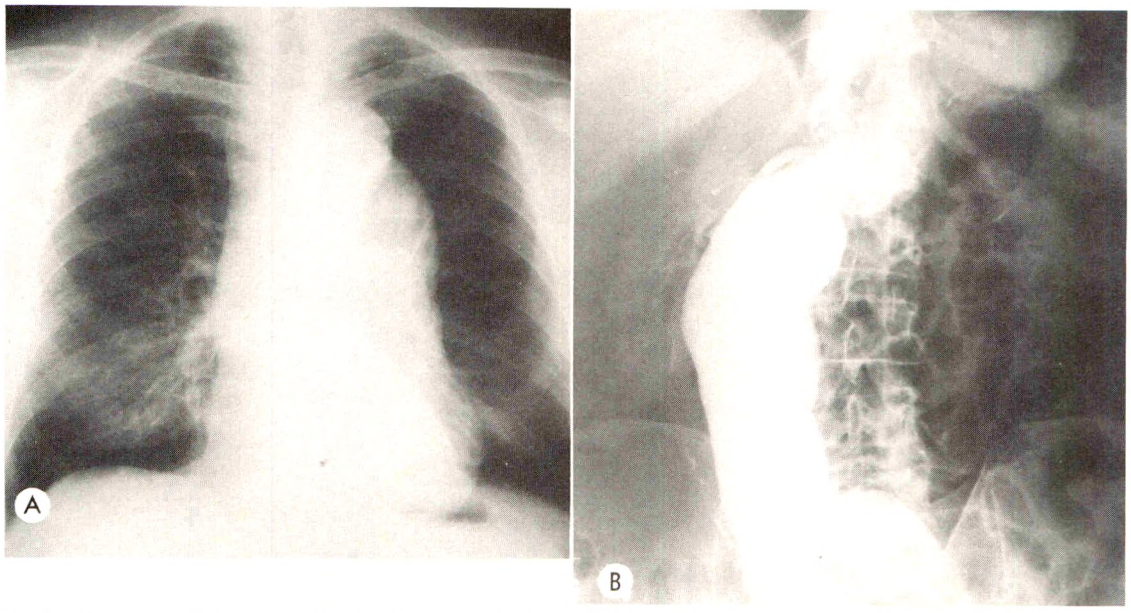

Fig. 3. Case III. (*A*) A posteroanterior chest roentgenogram shows a large, extensive aneurysm of the thoracic aorta. (*B*) The abdominal and thoracic aorta are markedly dilated and irregular with the iliac vessels similarly involved.

appeared, many in the French literature.[5,8,12,13] Arteriomegaly, as has been suggested before, is probably the simplest descriptive term.[8]

Fig. 4. Case IV. The aortic and iliac vessels show marked tortuosity particularly on the left.

Our patients varied in age from 64 to 89 with an average of 71 years. While hypertension has been commonly associated with this condition,[8] its absence in our series would suggest a coincidental occurrence. Three of our patients had embolism or thrombosis, which was probably related to their aneurysms. Both thrombosis and embolism are frequently noted with arteriomegaly, and it is suggested that this may occur in the absence of an aneurysm.[8,12] One would question if Case VI (Table I), who probably had either an embolism or thrombosis with only slow blood flow and no coexistent aneurysm, might not fall into this latter category. All of our patients had prolonged blood flow with ectatic and irregular vessels. The tortuosity alone may be of some significance as in Case IV (Table I) who presented for evaluation of a pulsatile mass which represented only a tortuous vessel. Leriche's[10,11] first two cases of this entity presented in a similar manner. It is to be noted that all of our cases but one had prolonged blood flow without arterial occlusion and in Case VII (Table II) the unoccluded leg also had prolongation of blood flow. This delay often

TABLE I

CLINICAL INFORMATION

Case No.	Age	B.P.	Thrombosis, Embolism or Occlusion	Surgery	Other
I. W.B.	65	140/90	Tingling right foot, probable embolization	(1) Resection of bilateral hypogastric and right profunda aneurysms with aorto-iliac and femoral graft (2) Resection right popliteal aneurysm with vein graft	
II. A.L.	64	140/90	Thrombosed right popliteal aneurysm	(1) Resection right popliteal aneurysm	
III. L.B.	69	140/80	None	None	Died of pulmonary embolus
IV. A.M.	81	130/90	None	None	Presented with pulsatile mass LLQ
V. R.D.	89	120/70	Right superficial femoral occlusion	Right femoral popliteal by-pass graft	
VI. D.R.	66	150/90	Gangrene right foot ? embolization	(1) Resection right 4th toe (2) Right below-knee amputation	Diabetic
VII. C.H.	65	140/80	(1) Emboli left foot (2) Clotted graft	(1) Resection left popliteal aneurysm with graft (2) Left below-knee amputation (3) Resection right popliteal aneurysm with graft	

B.P.= blood pressure.
LLQ= left lower quadrant.

results in poorly timed roentgenograms with poor delineation of distal vessels. The slow flow is probably directly related to the large cross sectional area of the arterial lumen.[8,12] By including the impressive aortic and iliac aneurysms of Case III there was a total of four aneurysms. This frequency of occurrence with arteriomegaly has been previously emphasized.[8,12] While the dividing line between a segmentally ectatic vessel and a fusiform aneurysm may be quite thin, the presence of thrombosis was usually considered evidence of the latter. It is to be noted that the incidence of arteriomegaly in our population (six of 75) is much higher than has formerly been reported.[12]

While it is not the concern of this paper, we have also noted occasional patients with ectatic, tortuous changes involving the great vessels. One of these was felt to have an aneurysm of the innominate artery until the arteriogram showed merely a tortuous vessel. This aspect of arteriomegaly has

TABLE II

ROENTGENOGRAPHIC FINDINGS

Case No.	Prolonged Flow	Tortuosity	Ectasia	Plaque	Aneurysm	Occlusion
I. W.B.	Femoral 7 sec. Knee 30 sec.	4+ Iliac	4+	4+	Aorta, right and left common iliac, right, profunda, right and left popliteal	Poor run-off right
II. A.L.	Femoral 3 sec. Knee 30 sec.	2+	4+	4+	Bilateral popliteal, left hypogastric	Clots right popliteal with extravasation
III. L.B.	Appears prolonged, no distal films	3+	4+	3+	Thoracic and abdominal aorta and iliacs	None
IV. A.M.	Femoral 4 sec. Knee 8 sec.	4+	2+	1+	None	None
V R.D.	Femoral 5 sec. Knee 20 sec.	2+	2+	4+	None	Right superficial femoral
VI D.R.	Femoral 4 sec.	4+ Iliac	3+	3+	None	None
VII. C.H.	Times uncertain but markedly prolonged	4+ Iliac	4+	3+	Bilateral popliteal	Poor distal filling

also been described previously.[3,7]

While none of our patients had venograms, in his original work Leriche[10,11] reported venous ectasia as well, suggesting some type of generalized connective tissue disorder. However, this has not been substantiated in other reports, and only the anticipated deformity from the tortuous arteries has been noted.[9]

On review of the pathological material from our operative cases no characteristic findings were noted that would differentiate arteriomegaly from the changes seen with varying degrees of arteriosclerosis. This has been the experience of previous authors.[1,8,12] In an attempt to make a parallel with other conditions, we have found two pediatric cases, one at 17 months and the other at ten years of age, with bizarre tortuous vessels.[4,6] One of these showed an increase in the number of elastic fibers,[4] while the other showed an actual decrease of the external elastic membrane with fragmentation of the internal elastic membrane.[6] In addition, with the kinky-hair syndrome, a metabolic disease which is probably related to copper deficiency, tortuous vessels are common with intimal thickening and fragmentation of the internal elastic lamina on histological evaluation.[2] While patients with Marfan's disease often have tortuous vessels, one would expect a related condition to have a similarly increased incidence of aortic dissection which has not been noted with any reported case of arteriomegaly. In our cases with an average age of 71, such extensive arteriosclerosis is noted that the fine changes described in the pediatric patients discussed above would be obscured. Hence further investigation should be directed toward the younger patient with arteriomegaly, particularly in relation to uninvolved vessels. Therefore, while arteriomegaly appears to represent only an interesting variant of arteriosclerosis, the possibility of a yet undetected structural abnormality still persists.

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REFERENCES

1. Ackerman, L. V., and Butcher, H. R. Surgical Pathology. Third edition. C. V. Mosby, St. Louis, 1974, pp. 1191–1192.
2. Adams, P. C., Strand, R. D., Bresnan, M. J., and Lucky, A. W. Kinky hair syndrome: serial study of radiological findings with emphasis on similarity to battered child syndrome. *Radiology*, 1974, *112*, 401–407.
3. Bariéty, M., Paulet, J., and Terris, G. Les dolicho-méga-artèries. *Bull. et mem. Soc. méd. hôp.* Paris, 1961, *77*, 394–412.
4. Beuren, A. J., Hort, W., Kalbfleish, H., Müller, H., and Stoermer, J. Dysplasia of systemic and pulmonary system with tortuosity and lengthening of arteries. *Circulation*, 1969, *39*, 109–115.
5. Ecoiffier, J., Laval-Jeantet, M., and Kauffmann, Ciné-arteriographic femorale dans les artheromatoses avec dolicho-méga-artère. *J. de radiol., d'électrol. et de méd. nucléaire*, 1968, *49*, 294–296.
6. Ertugrul, A. Diffuse tortuosity and lengthening of arteries. *Circulation*, 1967, *36*, 400–407.
7. Hultén-Gyllenstein, J. L., Löfstedt, S., and von Reis, G. Observations on generalized arteriectasis. *Acta med. scandinav.*, 1959, *163*, 125–132.
8. Lea Thomas, M. Arteriomegaly. *Brit. J. Surg.*, 1971, *58*, 690–694.
9. Lea Thomas, M., and Andress, M. R. Phlebographic changes in arteriomegaly. *Acta radiol.*, 1970, *10*, 427–432.
10. Leriche, R. Dolicho et méga-artère dolicho et méga-veine. *Presse méd.*, 1943, *51*, 554–555.
11. Leriche, R. Les symptoms douloureux des dolicho-artères. *Presse méd.*, 1947, *55*, 641.
12. Staple, T. W., Friedenberg, M. J., Anderson, M. S., and Butcher, H. R., Jr. Artérie magna et dolicho of Lériche. *Acta radiol.* (Diag.), 1966, *4*, 193–305.
13. Warembourg, H., Niguet, G., Merlen, J. F., Ducloux, G., and Dubeaux, P. A. Les dolichomégaartères *Lille méd.*, 1963, *8*, 661–668.

Vol. 125, No. 3

DISSECTING ANEURYSM OF THORACIC AORTA: REAPPRAISAL OF RADIOLOGIC DIAGNOSIS*

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ABSTRACT:

In a series of 24 cases of acute dissecting aneurysm of the aorta (not including Marfan's disease) the diagnosis was usually suspected on the basis of the clinical picture and plain chest roentgenograms.

The most consistent clinical sign was severe pain. Absent pulses and a neurological deficit were each noted in only five patients. In many cases there was no correlation between the clinical picture and the type or the extent of the dissection.

Widening of the aortic arch and obliteration of the aortic knob with displacement of the trachea to the right are the most common signs in plain chest roentgenograms. A barium swallow examination in these cases reveals an elongated compression and displacement of the esophagus by the aortic arch. Calcification in the area of the aortic arch is the exception rather than the rule in dissecting aneurysms.

Angiography is essential for the definitive diagnosis of dissecting aneurysms. The diagnosis is based on the demonstration of two channels, either by the presence of a linear radiolucency separating the two lumens, or by differences in flow that present as delayed opacification or delayed washout.

If only the true lumen is opacified, widening of the outer extraluminal border of the aorta or narrowing of the lumen indicates the presence of a dissection. Abnormal catheter recoil and position were helpful in only two cases, and are not informative when the false lumen is catheterized. Failure to visualize main aortic branches was not always due to involvement by the dissection. It can also be caused by reduced flow due to severe proximal compression of the main lumen.

The exact location of the intimal tears is usually not demonstrated unless additional injections are made in the area assumed to contain the tear.

If only the false lumen is opacified in the ascending aorta, this can be recognized by the demonstration of a blind end, by failure to visualize the sinuses of Valsalva, from flattening of the medial border of the opacified channel, and from delayed washout in the blind end.

THE mortality from dissecting aneurysm of the aorta has decreased in recent years as a result of improved surgical and medical treatment.[6,11,20,22]

The typical clinical picture and plain chest roentgenogram are usually suggestive of the diagnosis of dissecting aneurysm. Aortography is essential for the demonstration of the site of the primary tear and the extent of the dissection.[1,2,19] This information is indispensable in order to determine which case is suitable for initial medical management, and in which case emergency surgery is indicated.[20–22]

The present report deals with clinical observations and analyses of the roentgenological features of 24 cases of dissecting aneurysm of the thoracic aorta, in an attempt to define the contribution of the plain chest roentgenogram and to evaluate the reliability, specificity, and interpretation of various angiographic features.

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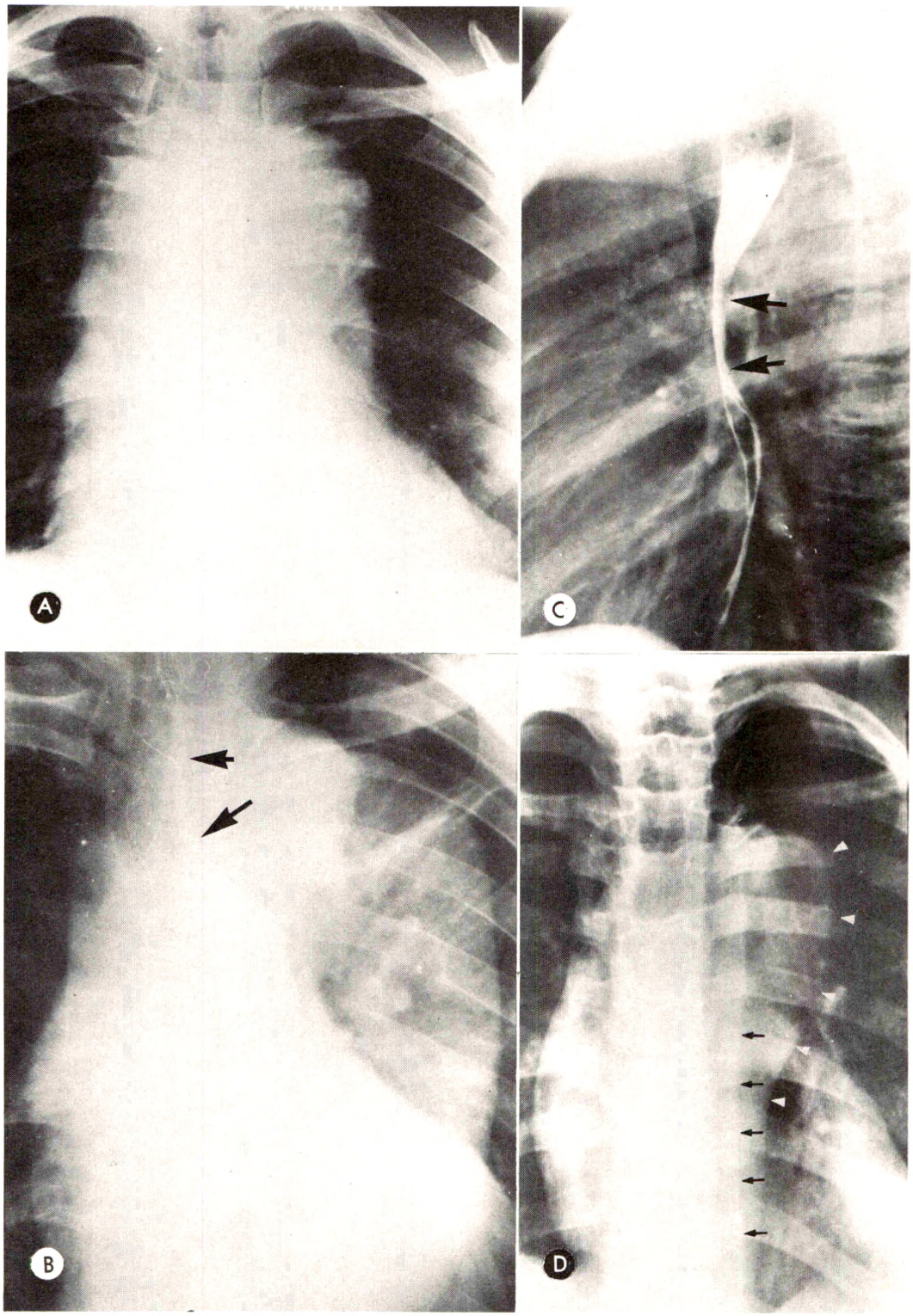

FIG. 1. Dissecting aneurysms of thoracic aorta of different patients. Plain chest roentgenograms and esophagograms. (*A*) Plain chest roentgenogram, frontal view. Widening of the aortic arch and disappearance of the aortic knob. (*B*) Esophagogram, frontal view. Elongated displacement of the esophagus (arrows) and trachea at the aortic arch level. (*C*) Esophagogram, lateral view. Elongated, segmental anterior displacement of the esophagus mainly below the level of the aortic arch (arrows). (*D*) Penetrated plain chest roentgenogram, frontal view. Widening of the descending aortic shadow (white arrows) and a prominent mediastinal paravertebral line (black arrows).

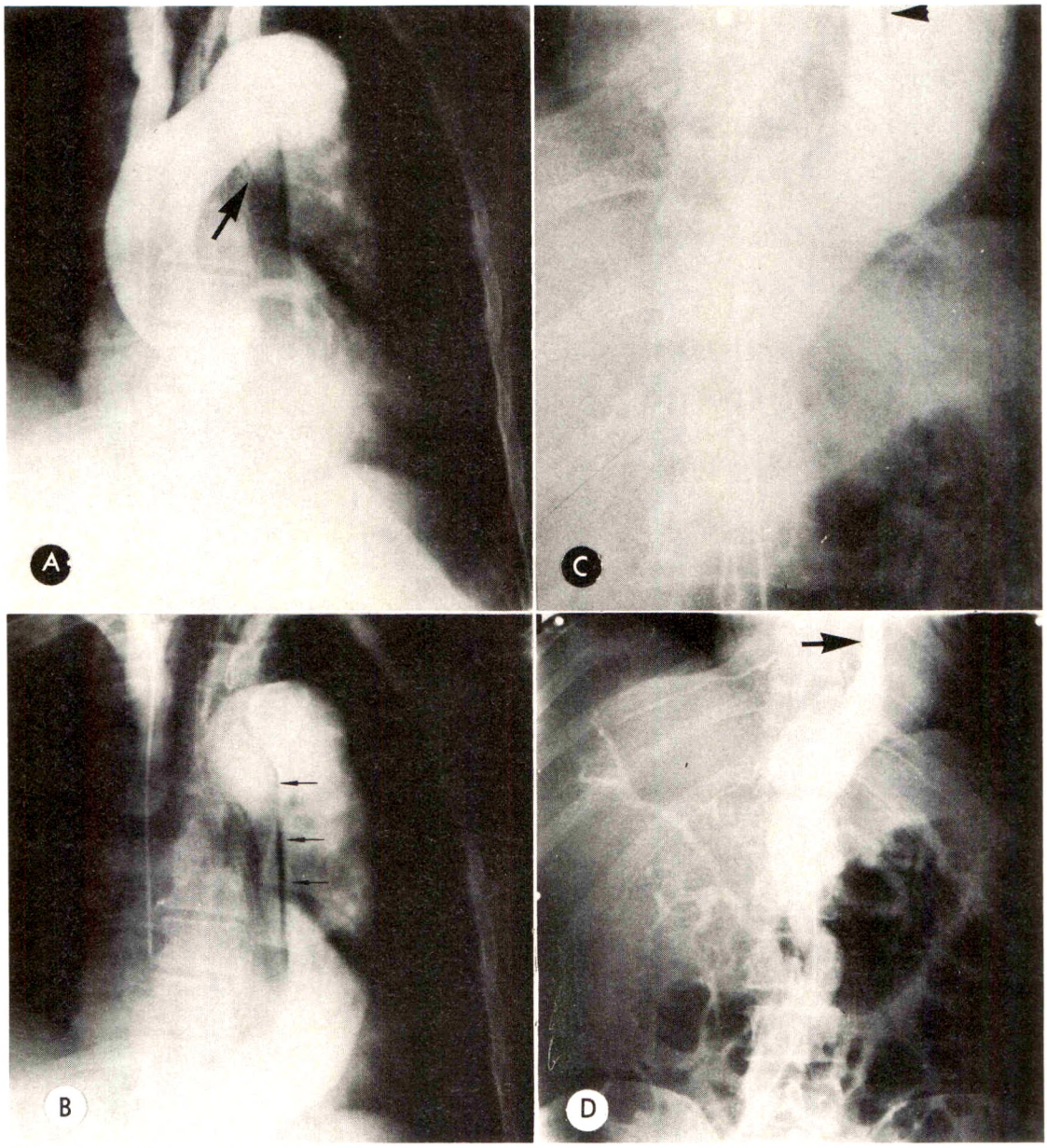

Fig. 2. Dissecting aneurysm Type III demonstrating dominant flow through the large false lumen, a narrowed true lumen and the exact localization of the re-entry. (*A*) Injection into the ascending aorta by the axillary route, early phase. The contrast material advances mainly in the laterally located dilated false lumen. Note the delayed opacification of the true lumen (arrow). (*B*) Later phase. Good visualization of both lumens separated by a linear radiolucency. The medially positioned true lumen is markedly narrowed. The exact site of re-entry cannot be recognized. (*C*) Catheterization of the abdominal aorta *via* the femoral artery demonstrates the exact site of re-entry (arrow). (*D*) Later phase. Disappearance of the dye from the false lumen with persistent opacification of the true lumen due to decreased flow and delayed washout in it.

CLINICAL OBSERVATIONS

During the period 1960–1973, 24 cases of dissecting aneurysm of the thoracic aorta were studied. The diagnosis was confirmed by angiography, surgery, or autopsy.

The cases were classified into types, according to the location of the primary tear and the extent of the dissections, as de-

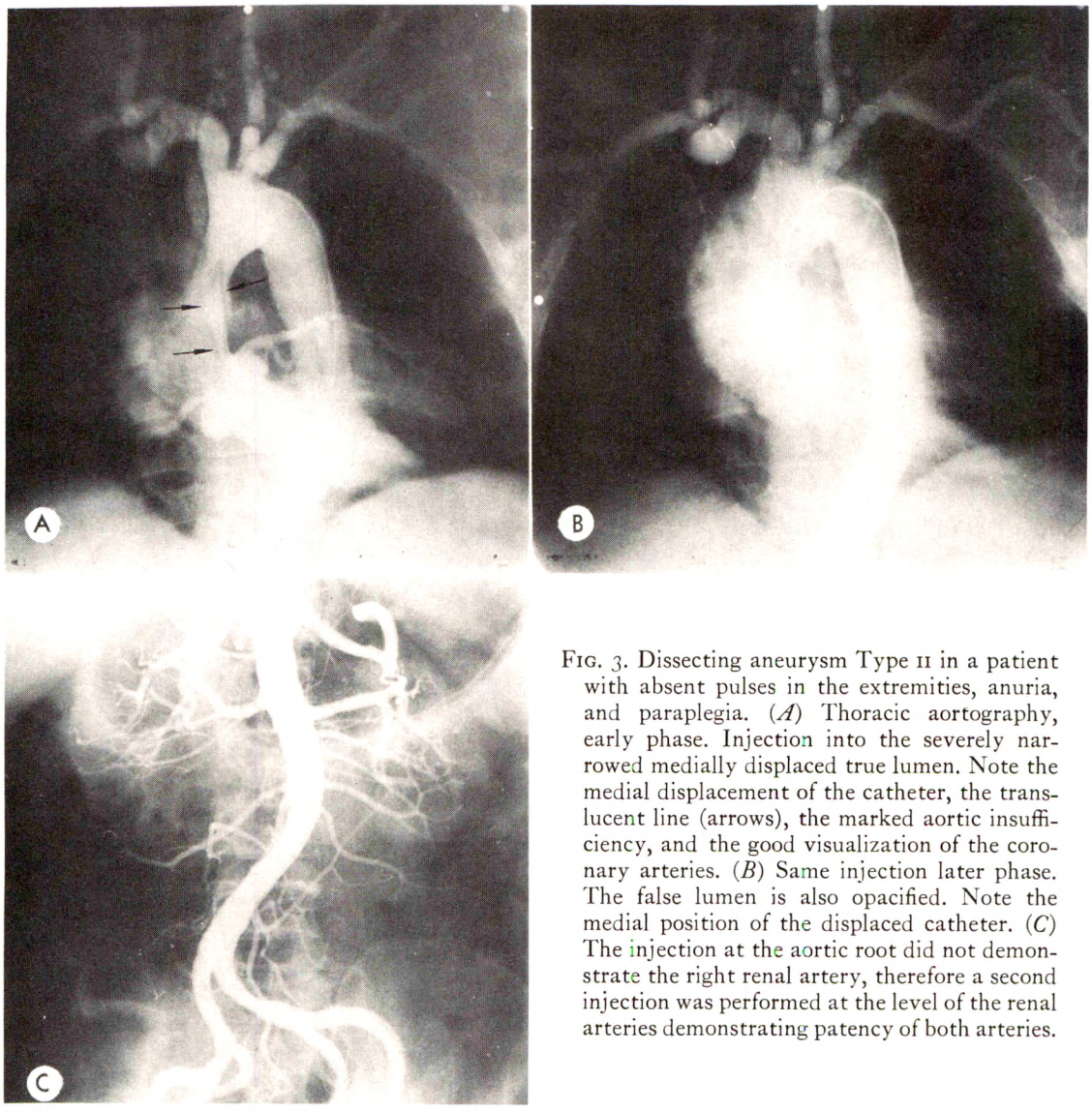

Fig. 3. Dissecting aneurysm Type II in a patient with absent pulses in the extremities, anuria, and paraplegia. (*A*) Thoracic aortography, early phase. Injection into the severely narrowed medially displaced true lumen. Note the medial displacement of the catheter, the translucent line (arrows), the marked aortic insufficiency, and the good visualization of the coronary arteries. (*B*) Same injection later phase. The false lumen is also opacified. Note the medial position of the displaced catheter. (*C*) The injection at the aortic root did not demonstrate the right renal artery, therefore a second injection was performed at the level of the renal arteries demonstrating patency of both arteries.

scribed by DeBakey *et al.*[5] There were five cases of Type I, three cases of Type II and 13 cases of Type III dissection. Three other cases were atypical with a primary tear as in Type III but with extension of the dissection proximal to the arch.

The average age of the patients was 57 years (range twelve–86). Seventeen patients were between 40–65 years old. There were 18 males and six females. Except for two young females, all patients were hypertensive. The youngest female patient had cystic medial degeneration, and in the other the diagnosis is yet unknown. Although six cases of Marfan's disease, with the typical dilatation of the aortic bulb, were excluded from this series, these two young normotensive females were included as they did not conform with the typical angiographic characteristics of Marfan's syndrome, and they presented some interesting angiographic features.

The most common clinical sign was a sudden severe pain in the chest radiating to

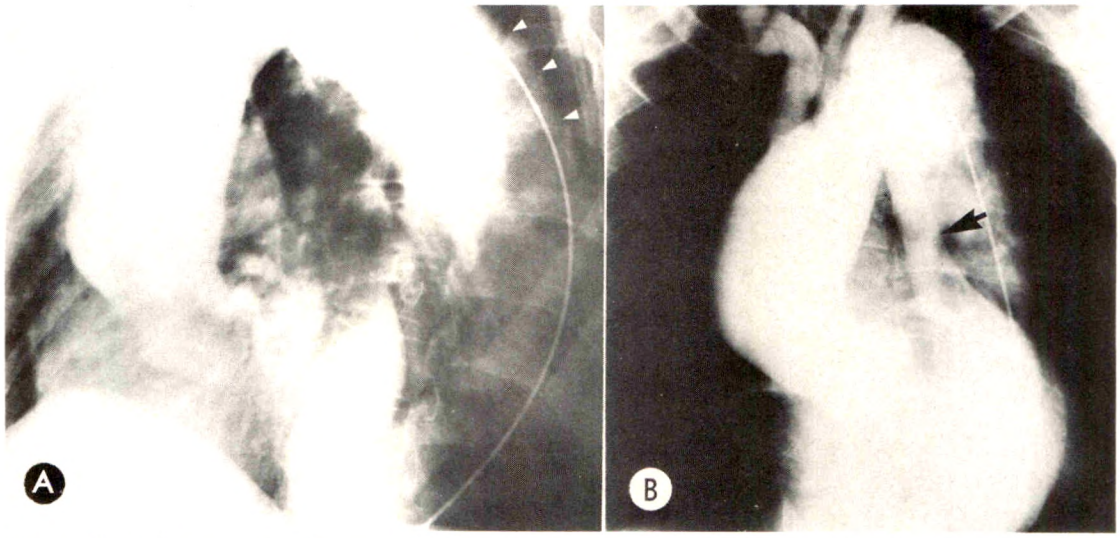

Fig. 4. Dissecting aneurysm Type III. The catheter passed the false lumen of the thoracic aorta and re-entered the true lumen at the arch. (*A*) Injection in the ascending aorta. Note the position of the catheter (left anterior oblique view) in the false lumen at the outer border of the aortic shadow (arrowheads). There is delay in the opacification of the false lumen. (*B*) Frontal view. Segmental narrowing of the true lumen (arrow) and delayed opacification of the laterally positioned false lumen.

the back (eight cases). Severe abdominal pain was also present in seven patients, one of which was operated upon at another hospital for suspected acute appendicitis. In two of our patients the abdominal pain preceded the pain in the chest.

Absence of peripheral pulsations was noted in five patients. A neurological deficit was present in five patients: paraparesis in three; a sensory and motor deficit in one leg in the fourth; and hemiplegia in the fifth patient. These neurological signs are ascribed to ischemia of the spine, peripheral nerves, or the brain.[9]

Burchell[3] attempted to relate the site and extent of the dissection to the location and radiation of the pain. We were not able to correlate the clinical symptoms with the type and the extent of the dissection: patients with Type III dissection had a variety of pain patterns, and the typical chest pain radiating to the back was encountered in all types of dissection. Hirst[13] also found that only 54 percent of patients with involvement of the descending thoracic aorta had pain in the back. Others reported that almost one-fourth of the patients with abdominal pain did not have any extension of the dissection below the diaphragm.[12,13,16] Even signs of major vessel occlusion do not indicate the extent of the dissection with certainty: in one of our cases of Type II dissection, femoral pulses were not palpable and the patient was paraplegic and anuric. Although the dissection did not extend beyond the ascending aorta in this patient, the main aortic lumen was so severely compressed that distal perfusion was extremely low. It appears, therefore, that the clinical symptomatology is inadequate for firm anatomical conclusions, and angiographic information is essential.

RADIOLOGIC OBSERVATIONS

Plain chest roentgenograms were available for evaluations in all patients, and 18 patients underwent angiography.

Our method of examination includes frontal and lateral roentgenograms of the chest followed, if possible, by fluoroscopy with a barium swallow examination, and comparison with previous roentgenograms if available. The angiographic catheter is introduced into the aortic arch percutane-

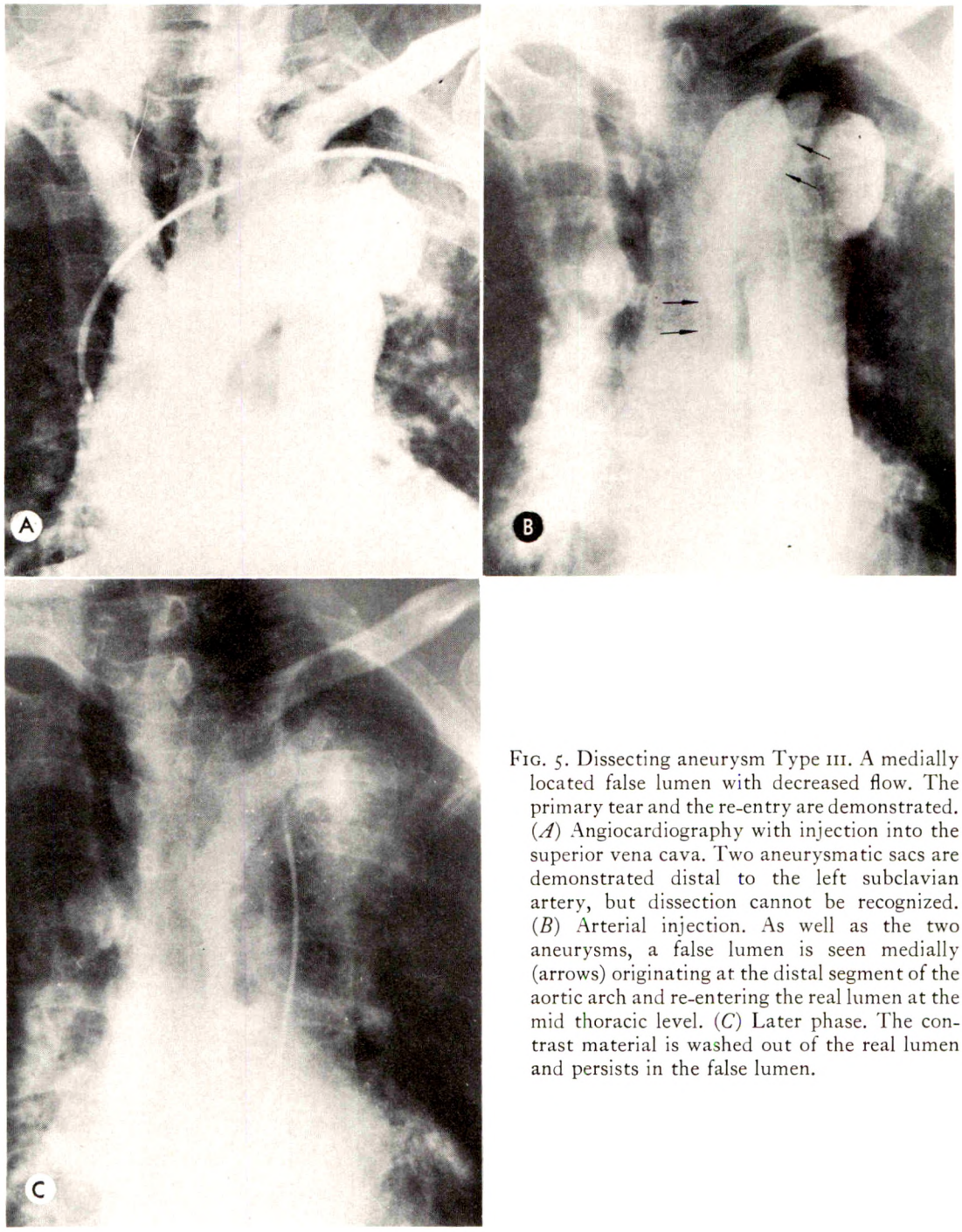

FIG. 5. Dissecting aneurysm Type III. A medially located false lumen with decreased flow. The primary tear and the re-entry are demonstrated. (*A*) Angiocardiography with injection into the superior vena cava. Two aneurysmatic sacs are demonstrated distal to the left subclavian artery, but dissection cannot be recognized. (*B*) Arterial injection. As well as the two aneurysms, a false lumen is seen medially (arrows) originating at the distal segment of the aortic arch and re-entering the real lumen at the mid thoracic level. (*C*) Later phase. The contrast material is washed out of the real lumen and persists in the false lumen.

ously by the Seldinger technique. The procedure followed has been previously described.[1,2,15,19]

PLAIN CHEST ROENTGENOGRAM

Dissecting aneurysm can be suspected from abnormalities in the contour of the aorta seen on the plain chest roentgenogram, but usually the roentgenographic findings are nonspecific and cannot confirm or rule out the diagnosis.[1,14] In other cases when a dissecting aneurysm is not even con-

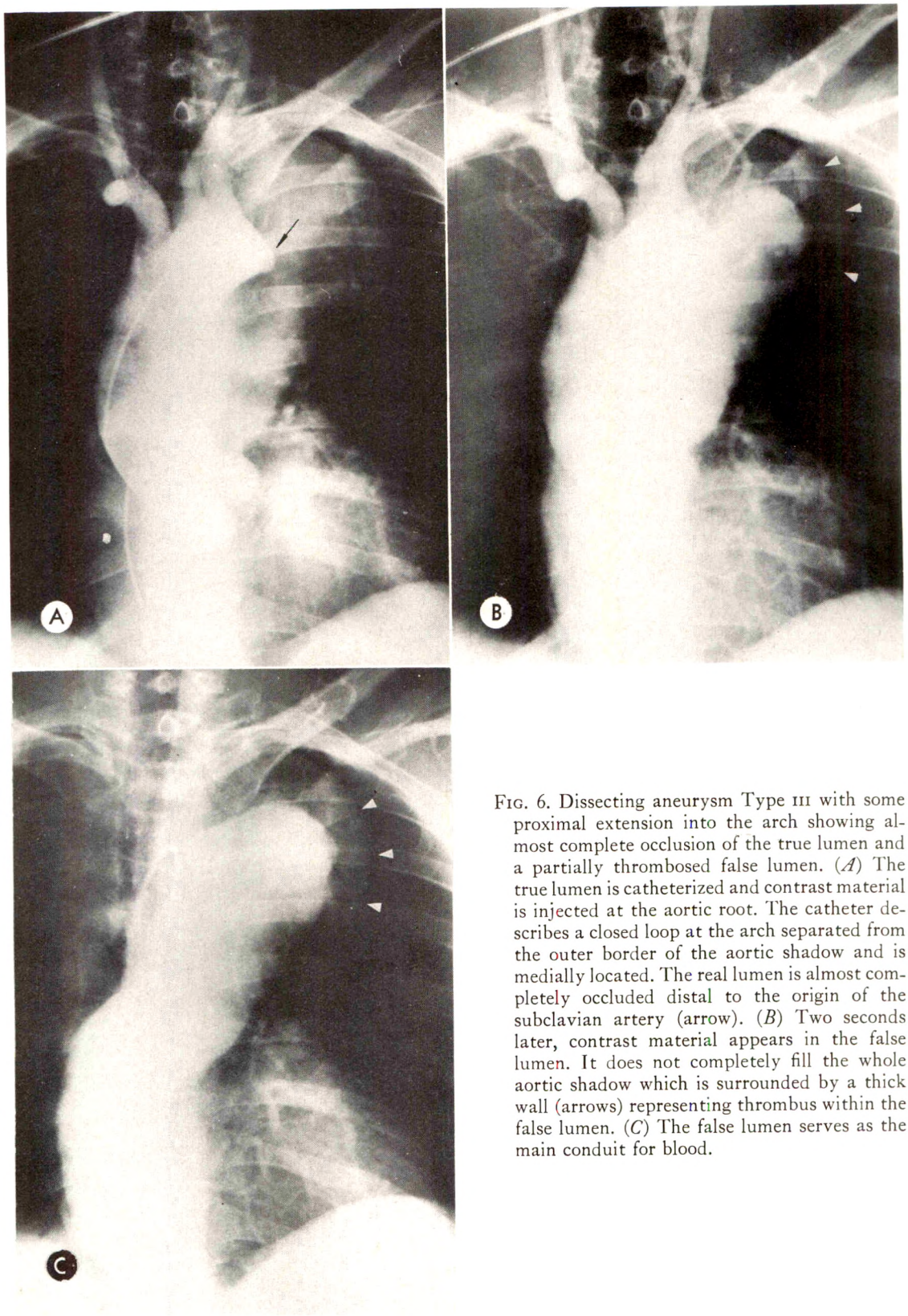

Fig. 6. Dissecting aneurysm Type III with some proximal extension into the arch showing almost complete occlusion of the true lumen and a partially thrombosed false lumen. (*A*) The true lumen is catheterized and contrast material is injected at the aortic root. The catheter describes a closed loop at the arch separated from the outer border of the aortic shadow and is medially located. The real lumen is almost completely occluded distal to the origin of the subclavian artery (arrow). (*B*) Two seconds later, contrast material appears in the false lumen. It does not completely fill the whole aortic shadow which is surrounded by a thick wall (arrows) representing thrombus within the false lumen. (*C*) The false lumen serves as the main conduit for blood.

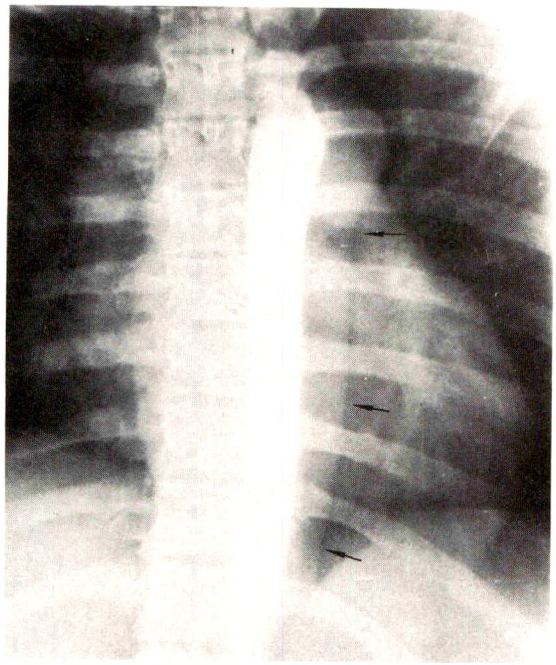

Fig. 7. Dissecting aneurysm Type III. In this young woman the plain roentgenogram of the chest showed a markedly widened descending aorta. Aortography showed the opacified descending aorta and the thrombosed false lumen (arrows).

sidered on clinical grounds, the chest roentgenogram may be very valuable and provide the first clue for a correct diagnosis. This is particularly true in cases of chronic dissection.

A common but not pathognomonic finding on the chest roentgenogram is a widening of the mediastinum and the aortic shadow. This sign was seen in 20 out of our 24 cases (Fig. 1). Comparison of successive roentgenograms is particularly useful in evaluation of changes in the configuration of the aorta. In four of our cases this information contributed to the diagnosis.

Obliteration of the aortic knob was seen in 21 out of 24 cases (Fig. 1*A*). Blurring of the outer border of the aortic knob indicating involvement of the arch was seen in eight of these 21 cases (five cases of Type I and three cases of Type III dissection extending into the arch).

Out of eight cases affecting the ascending aorta (five cases of Type I and three cases of Type II) in three the ascending aorta appeared widened. Unusual widening of the descending aorta contributed to the diagnosis in three cases (Fig. 7).

Left pleural effusion was observed in four patients, and in two a widened paraspinal shadow was seen (Fig. 1*D*). This latter feature in the presence of a dissection indicates probable leakage.[10]

On barium swallow examination the esophagus may be displaced anteriorly and to the right. This displacement appears as an elongated exaggeration of the normal aortic indentation on the esophagus, and was noted in six out of nine cases examined (Fig. 1, *B* and *C*). A shift of the trachea to the right may also be present and was seen in 14 cases (Fig. 1*C*).

Displacement of the intimal calcification from the outer border of the aorta has been

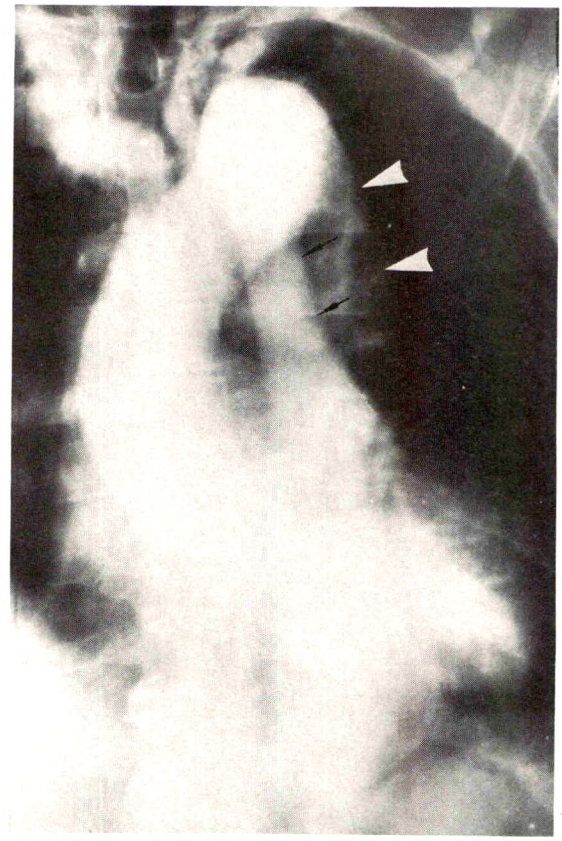

Fig. 8. Dissecting aneurysm Type III. A segmental narrowing of the lumen of the descending aorta (black arrows) with a corresponding widening of the extraluminal shadow (white arrows).

described. However, in the present series only one out of 24 cases had such a calcification, and the interpretation of this sign is not always completely reliable.[4,8] Furthermore, calcification of the aortic knob is rather rare in dissecting aneurysms, probably because arteriosclerosis is not a major feature in these cases.[1,11,13]

In the differential diagnosis of patients suspected clinically of having a dissecting aneurysm, and displaying a widening of the mediastinal shadow, other vascular pathology of the aorta must be considered. An elongated and tortuous aorta secondary to arteriosclerosis and hypertension may cause widening of the aortic shadow or a double contour in the anteroposterior projection. Lateral roentgenograms with a barium swallow may help to differentiate this condition from dissecting aneurysm. The double contour in these cases is caused by a summation of shadows of the afferent and the efferent portions of the tortuosity.

True aneurysms of the thoracic aorta (arteriosclerotic or luetic) will also cause a widening of the aortic shadow. However, in these cases the outline of the aortic border remains distinct, and if the aortic wall is calcified, a common feature of arteriosclerotic aneurysms, the calcification will be seen at the outer border.[1,8,13] In any case of doubt angiography will clarify the real pathology.

ANGIOGRAPHIC FINDINGS

The angiographic diagnosis of dissecting aneurysm of the aorta depends on the demonstration of a double lumen within the aorta, one lumen representing the true and the other the false channel.[1,7,18] Angiography may indicate the extent of the deformation of the true channel, the flow or absence of flow in the false channel, and the presence or absence of aortic insufficiency. It may also indicate involvement of main branches;[17,18] however, such involvement may be present without angiographic evidence.

The angiographic demonstration of two separate channels depends on the site of injection of the contrast material and on the presence and rate of flow in both channels. When the injection is proximal to the primary tear and when there is flow in both lumens, a linear lucency may be seen within the opacified aorta delineating the outer border of the true lumen and the inner border of the false lumen (Fig. 2*B*; and 3*A*). The demonstration of this linear lucency is possible only when its direction is parallel to the roentgen-ray beam.

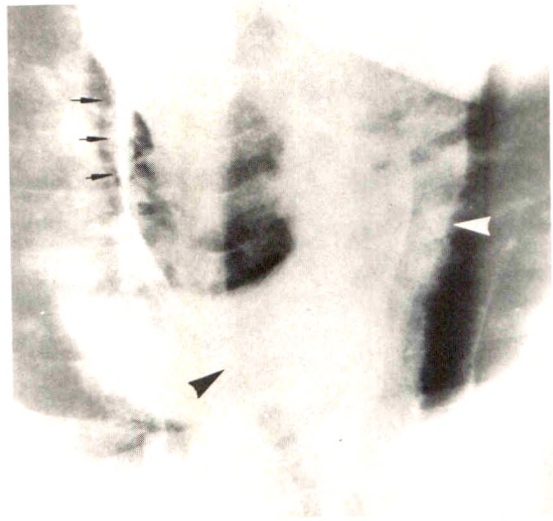

Fig. 9. Dissecting aneurysm Type III. The catheter is in the true lumen and displaced medially from the outer border (black arrowhead). The false lumen is markedly dilated and the true lumen is almost completely occluded (black arrows). Note the widened soft tissue shadow (white arrowhead) accompanying the ascending aorta in this left anterior oblique view, which is caused by superimposition of the superior vena cava and the right atrium.

When there is a difference in the rate of flow in the two channels, late appearance and delayed washout of the dye may be noted in the channel in which flow is decreased (Fig. 2, *A* and *B*; and 4*B*). It is generally assumed that the flow is decreased in the false lumen.[1,18] However, this is not always the case, as the false luman may act as the main conduit for blood (Fig. 2, *A* and *C*). The false lumen is the outer of the two channels (Fig. 2*A*; 4*B*; and 6*B*) except on very rare occasions (Fig. 5, *B* and *C*).

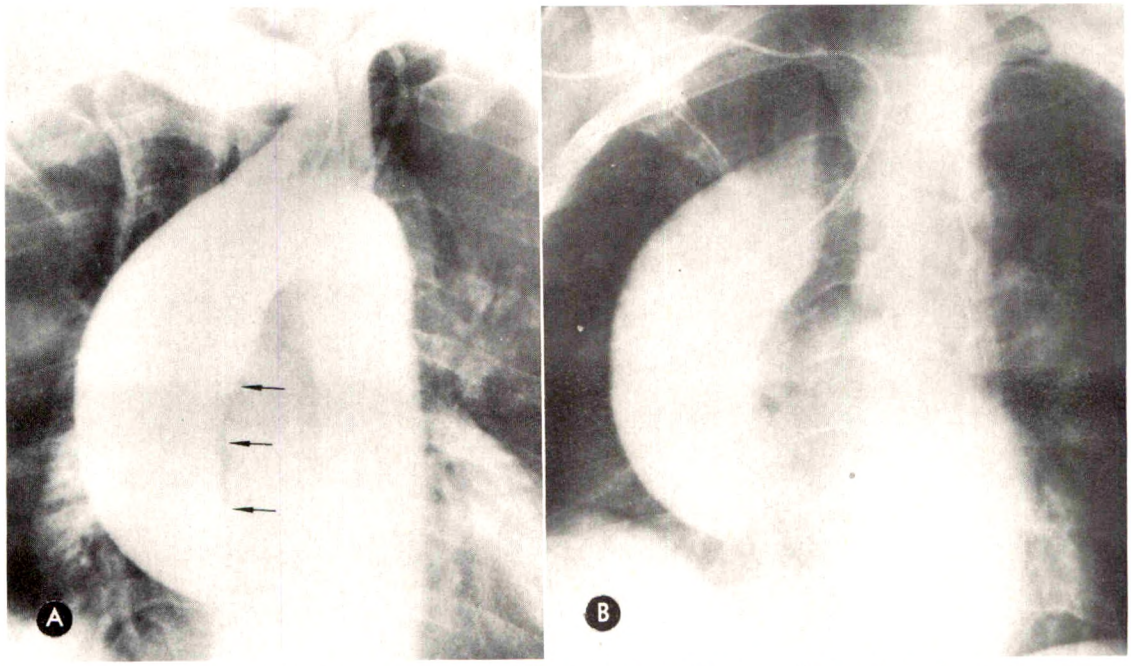

Fig. 10. Dissecting aneurysm Type II entering at the distal part of the ascending aorta and extending toward the aortic root. The catheter passed in the ascending aorta from the true lumen into the false lumen. (*A*) Frontal view. Injection into the false lumen demonstrates straightening of the medial border of the ascending aorta (arrows), non visualization of sinuses of Valsalva, and no washout. (*B*) Left anterior oblique position, late phase. Stagnation of the dye in the false lumen is apparent.

When only one channel is opacified, because of absence of flow in the false channel or because of injection of the dye distal to the primary tear, then the width of the outer extraluminal shadow may indicate the presence of a dissection. Normally the extraluminal shadow of the aorta is 3–5 mm wide. If larger (Fig. 7; and 8) it must be considered as an intramural hematoma.[1,4,18] It must be noted, however, that in the left anterior oblique projection a widened extraluminal shadow may be seen at the outer border of the ascending aorta that is due not to dissection but to summation of the soft tissue shadows of the right atrium and the superior vena cava (Fig. 9). Segmental narrowing of the opacified lumen is an additional indication of a dissection (Fig. 8).

The width of the extraluminal shadow may also be indicated by the position of the catheter during the pressure injection of the contrast material.[4] When the catheter lies in the true lumen at the aortic arch, it tends to straighten out during the pressure injection of contrast material. The catheter then assumes the broadest possible curve and is sprung against the superior wall of the aortic arch. If the catheter does not reach the superior margin of the opacified lumen it can be assumed that a dissection is present and the motion of the catheter is restricted by the displaced intima (Fig. 3*B*; and 9). This situation occurs only when the true lumen is catheterized. If the false channel is catheterized and only it is opacified, then the width of the outer extraluminal shadow will be normal and the position of the catheter during the pressure injection will not be helpful (Fig. 4, *A* and *B*). In some of these cases, especially if the false lumen extends in the ascending aorta proximal to the site of the primary tear, the demonstration of a blind end with delayed washout will indicate that this single channel is indeed the false lumen (Fig. 10, *A* and *B*).

Another important indication of an injection into the false lumen at the aortic root is a peculiar straightening of the medial border of the opacified lumen because of compression by the true lumen (Fig. 10*A*).

Failure to opacify the sinuses of Valsalva also indicates injection into the false lumen.[1,7,14] However, in the presence of a low primary tear the dye may escape from the false channel and the sinuses may be opacified even when the catheter is in the false lumen.

Although the type of dissection can often be judged on angiographic evidence, the exact location of the primary tear, or the site of re-entry when present, is only rarely located precisely. The location is sometimes facilitated by an additional injection of contrast material in the area that is assumed to contain the tear (Fig. 2, *B* and *C*; and 5*B*).

Failure to visualize main branches of the aorta may be due to their involvement by the dissecting process, or to an extreme reduction in pressure distal to a severe compression of the main lumen. In the latter case an additional injection near the origin of the non-visualized branch will opacify the main branches (Fig. 3*C*).

Only two cases in this series had aortic insufficiency (Fig. 3*A*). This relatively small proportion is probably due to the exclusion of cases with Marfan's disease, in which usually the sinuses of Valsalva and the aortic leaflets are involved in the dissecting process.

In the present series, angiography was performed in 18 cases. In ten the diagnosis was made by demonstrating two channels: a linear lucency separating the two lumens was seen in six cases; and differences in the rate of flow were present in seven cases.

In the other eight patients only one channel was opacified. In seven cases the diagnosis of dissection was based upon the presence of widening of the extraluminal shadow. In two of these cases this was determined from the position of the catheter during pressure injection. In one case the only indication for the presence of a dissection was compression of the main lumen. This sign was also present in five other cases.

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REFERENCES

1. Beachley, M. C., Ranniger, K., and Roth, F. J. Roentgenographic evaluation of dissecting aneurysms of aorta. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *121*, 617–625.
2. Björk, L. Angiographic technique in aortic aneurysms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 353–354.
3. Burchell, H. B. Aortic dissection (dissecting hematoma, dissecting aneurysm of aorta). *Circulation*, 1955, *12*, 1068–1079.
4. Cramer, G. G., and Amplatz, K. Catheter position: aid in diagnosis of dissecting aneurysm of thoracic aorta. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 836–839.
5. DeBakey, M E., Henly, W. S., Cooley, D. A., Morris, G. C., Jr., Crawford, E. S., and Beall, A. C., Jr. Surgical management of dissecting aneurysm involving ascending aorta. *J. Cardiovasc. Surg.*, 1964, *5*, 200–211.
6. DeBakey, M. E., Henley, W. S., Cooley, D. A., Morris, G. C., Jr., Crawford, E. S., and Beall, A. C., Jr. Surgical management of dissecting aneurysm of aorta. *J. Thoracic & Cardiovasc. Surg.*, 1965, *49*, 130–149.
7. Dinsmore, R. E., Willerson, J. T., and Buckley, M. J. Dissecting aneurysm of aorta: aortographic features affecting prognosis. *Radiology*, 1972, *105*, 567–572.
8. Eyler, W. R., and Clark, M. D. Dissecting aneurysm of aorta: roentgen manifestations including comparison with other types of aneurysms. *Radiology*, 1965, *85*, 1047–1057.
9. Erb, B. D., and Tullis, I. F. Dissecting aneurysm of aorta: clinical features of thirty autopsied cases. *Circulation*, 1960, *22*, 315–325.
10. Figiel, S. J., Figiel, L. S., and Rush, D. K. Changes in linear thoracic paraspinal shadow due to para-aortic hemorrhage: new sign of dissecting aneurysm. *Dis. Chest*, 1960, *49*, 379–383.
11. Grondin, C., David, P. R., Goor, D. A., Edwards, J. E., and Lillehei, C. W. Dissecting aneurysms of thoracic aorta: review of 52 cases with consideration of factors influenc-

ing prognosis. *Ann. Thoracic Surg.*, 1967, *4*, 29–37.

12. Halliday, J. H., and Robertson, J. S. Dissecting aneurysm of aorta. *Proc. Roy. Australian Coll. Phys.*, 1964, *1*, 3–15.
13. Hirst, A. E., Jr., Johns, V. J., Jr., and Kime, S. W., Jr. Dissecting aneurysm of aorta: review of 505 cases. *Medicine*, 1958, *37*, 217–279.
14. Kanick, V., Henley, S. D., Kittredge, R. D., and Finby, N. Some problems in angiographic diagnosis of dissecting aneurysm of thoracic aorta. Am. J. Roentenol., Rad. Therapy & Nuclear Med., 1964, *91*, 1283–1287.
15. Kirschner, L. P., Twigg, H. L., Conard, P. W., and Hufnagel, C. Retrograde catheter aortography in dissecting aortic aneurysms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *102*, 349–353.
16. Levine, S. A. Pitfalls in care of cardiacs. *Ann. Int. Med.*, 1955, *42*, 1270–1279.
17. Siegelman, S. S., Sprayregen, S., Strasberg, Z., Lari, A. A., and Robinson, G. Aortic dissection and left renal artery. *Radiology*, 1970, *95*, 73–78.
18. Soto, B., Harman, M. A., Ceballos, R., and Barcia, A. Angiographic diagnosis of dissecting aneurysm of aorta. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 146–154.
19. Stein, H. L., and Steinberg, I. Selective aortography: definitive technique for diagnosis of dissecting aneurysm of aorta. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *102*, 333–348.
20. Wheat, M. W., Jr., and Palmer, R. F. Drug therapy for dissecting aneurysms. *Dis. Chest*, 1968, *54*, 372–377.
21. Wheat, M. W., Jr., and Palmer, R. F. Dissecting aneurysms of aorta: present status of drug versus surgical therapy. *Prog. Cardiovasc. Dis.*, 1968, *11*, 198–210.
22. Wheat, M. W., Jr., Harris, P. D., Malm, J. R., Kaiser, G., Bowman, F. O., Jr., and Palmer, R. F. Acute dissecting aneurysms of aorta: treatment and results in 64 patients. *J. Thoracic & Cardiovasc. Surg.*, 1969, *58*, 344–351.

Vol. 125, No. 3

ROENTGENOLOGY OF SINUS OF VALSALVA ANEURYSMS*

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ABSTRACT:

Aneurysms of the sinuses of Valsalva are neither rare nor always fatal. They are of three types: congenital, acquired, and associated with cystic medial necrosis. Each can be recognized by its characteristic appearance on plain roentgenograms and arteriography. With surgical repair, the congenital type and that which is the result of cystic medial necrosis carry a good prognosis for the patient. With the acquired type, usually syphilitic, the prognosis with conservative management is good.

SINUS of Valsalva aneurysms were considered to be rare lesions as recently as 15 years ago. However, with the widespread use of angiocardiography, sinus of Valsalva aneurysms are now being diagnosed frequently. In recent surgical reviews, three institutions have accumulated 45, 35, and 26 cases of surgically corrected sinus of Valsalva aneurysms.[6,13,25] Because many of these once fatal lesions are now surgically correctable and because they are, in fact not rare, it is vital that the radiologist be thoroughly familiar with the plain roentgenographic and angiographic findings of these aneurysms. Careful analysis of the plain roentgenograms may enable the radiologist to make the initial diagnosis and proceed to angiographic confirmation without delay.

Aneurysms of the sinus of Valsalva have usually been divided into congenital and acquired types. Actually there are three groups of aneurysms: congenital; acquired; and associated with Marfan's disease, due to cystic medial necrosis. Each type has a distinctive pathophysiology and a different roentgenographic appearance. The purpose of this paper is to review briefly the clinical findings and to describe in some detail the plain roentgenographic and angiographic findings of each type (Table 1).

ANATOMY

A proper understanding of the different types of aneurysm requires a knowledge of the anatomy of the sinuses. The sinuses are the hollow spaces bounded medially by the aortic valve cusps and laterally by the wall of the aorta which bulges at its base[5] (Fig. 1). The coronary ostia arise from the aortic wall just above the sinuses. The right and left coronary arteries originate above the right and left coronary sinuses, respectively. The third sinus, located posteriorly, is called the non-coronary sinus.

The sinuses are intracardiac in location (Fig. 2*A*) and are intimately related to the four cardiac chambers and the pulmonary outflow tract (Fig. 2, *B* and *C*). The complexities of the sinus-cardiac chamber interrelationships are superbly detailed by Sakakibara and Konno[23] and will not be repeated, but the following interrelation-

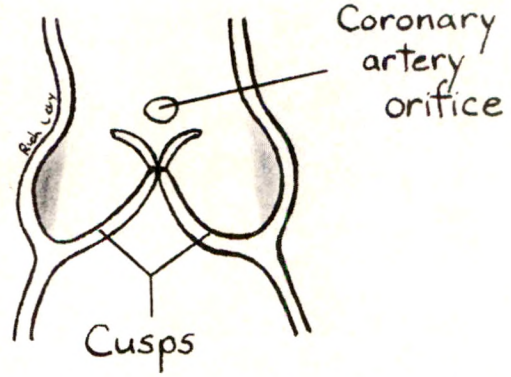

FIG. 1. Schematic cross section of the aortic root. The shaded areas represent the sinuses of Valsalva.

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TABLE I

CHARACTERISTICS OF SINUS OF VALSALVA ANEURYSMS

	Congenital		Marfans'—Cystic Medial Necrosis	Acquired
	Unruptured	Ruptured		
Pathology	Localized developmental defect in a sinus		Generalized cystic medial necrosis	Unequal distribution and degree of involvement
Symptoms	Asymptomatic	Dyspnea, fatigability, sudden chest pain	Angina, dyspnea	Angina, dyspnea, sudden chest pain after rupture
Clinical Findings	Aortic insufficiency or VSD murmur may be present	To-and-fro murmur left sternal border	Diastolic murmur, wide pulse pressure, may have Marfan's phenotype	Diastolic murmur. Wide pulse pressure
Plain Film Roentgenogram	Normal	Increased size right heart and pulmonary vasculature	Prominent ascending aorta. Left ventricular enlargement	Calcified masses adjacent to heart. Calcification ascending aorta. Left ventricular enlargement
Aortography	Small aneurysm. Windsock shape	Contrast medium into right heart and pulmonary artery	Uniform dilatation of all sinuses. Small linear dissections. Aortic insufficiency	Large saccular aneurysms. Irregular inner wall contour. Aortic insufficiency
Surgery	Resection of aneurysm and patching of defect		Replacement of valve and ascending aorta	Surgical correction very difficult
Prognosis	Good with surgery		Good with surgery	May go long period without rupture. Usually die of congestive failure secondary to aortic insufficiency

ships must be stressed. The right coronary sinus is adjacent to the pulmonary outflow tract, the crista supraventricularis of the right ventricle, and the anterior portion of the right atrium. The non-coronary sinus is adjacent to the right atrium and near the atrioventricular node and bundle of His. The left coronary sinus is covered only by pericardium in its mid and posterior portions, and the base of the left sinus is adherent to the anterior wall of the left ventricle.[5] The importance of these anatomic relationships will become clear when congenital aneurysms and pathways of rupture are discussed.

CONGENITAL ANEURYSMS

Congenital aneurysms of the sinus of Valsalva are usually small, measuring less than 4 cm in diameter and involving only one sinus, although cases with multiple sinus involvement have been reported.[3,10,29] Most aneurysms arise from the right sinus or the non-coronary sinus and rupture into the right atrium or right ventricle. Aneurysms arising from the left sinus were considered to be extremely rare[9,25] but three cases were reported by DeBakey *et al.*[6] Although there are many possible anatomic variations, four types comprise 90 percent of congenital aneurysms.[25] Aneurysms arising from the left half of the right sinus and eroding into the outflow tract of the right ventricle comprise 50 percent of congenital aneurysms. Most of these aneurysms are associated with a membranous ventricular septal defect. Another 40 percent are equally divided among the following types: (1) aneurysms arising from the right half of the right sinus eroding into the membranous interventricular septum; (2) from the right half of the right sinus to the atrioventricular portion of the right atrium; and (3) from the anterior half of the non-coronary sinus to the atrioventricular portion of the right atrium. Associated cardiovascular anomalies in addition to the ventricular septal defect include atrial septal

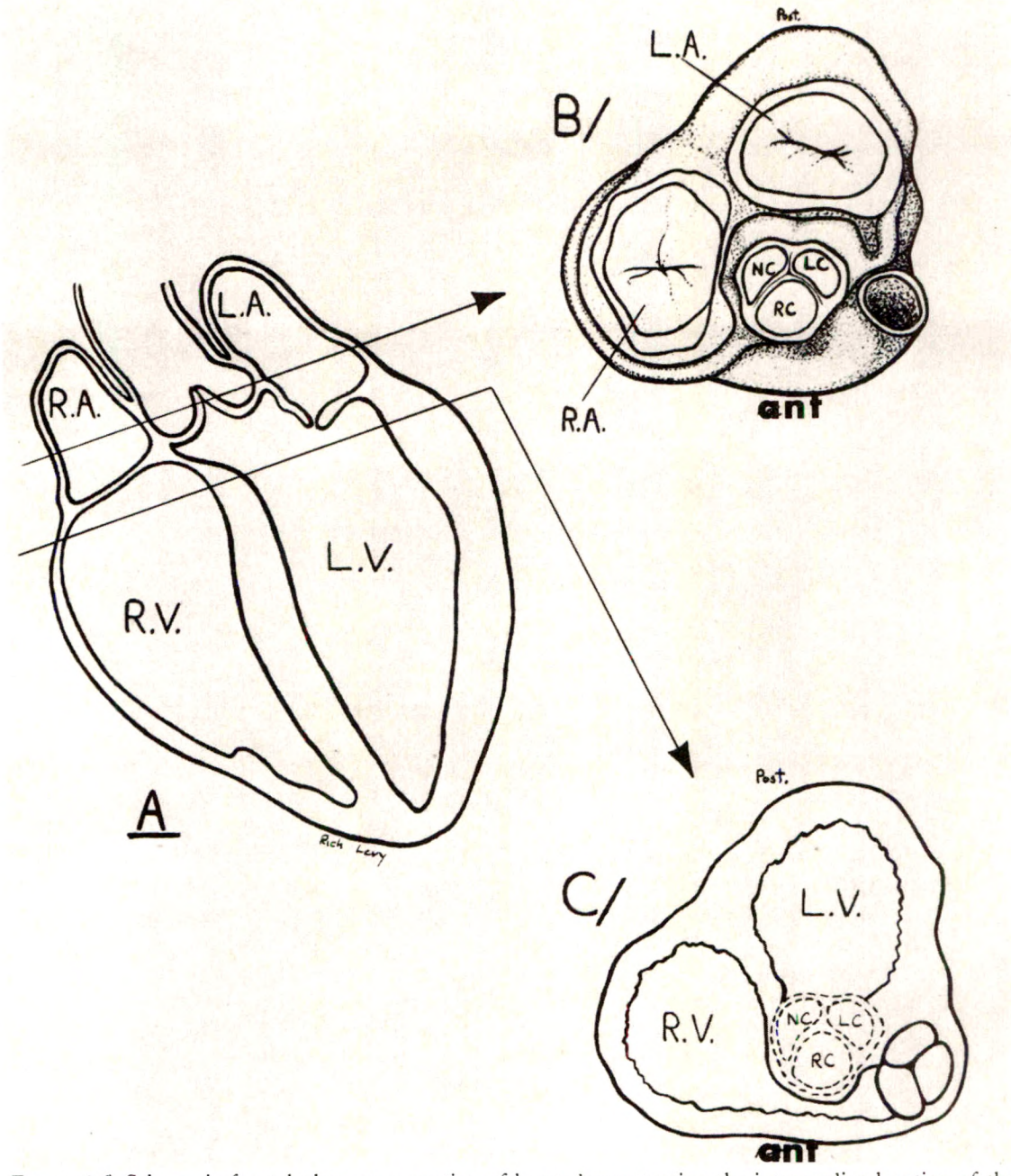

Fig. 2. (*A*) Schematic frontal plane cross section of heart demonstrating the intracardiac location of the aortic sinuses. (*B* and *C*) Cross sections at the atrial and ventricular levels corresponding to the planes of the arrows in *A* and demonstrating the relationship of the sinuses to the cardiac chambers.

RC = right coronary sinus, NC = non-coronary sinus, LC = left coronary sinus, R.A. = right atrium, L.A. = left atrium, L.V. = left ventricle, R.V. = right ventricle.

defect of the secundum type, coarctation of the aorta, bicuspid aortic valve, patent ductus arteriosus, tetralogy, and persistent left superior vena cava.

The etiology of the congenital lesion is thought to be the result of incomplete fusion of the two halves of the distal bulbar septum.[14] This is the structure that sepa-

rates the systemic and pulmonary halves of the bulbis cordis, the primitive exit tube of the fetal heart. Weakness secondary to an incompletely fused septum would predispose the high pressure area of the sinuses to aneurysm formation. Similar deficiencies in fusion in the proximal bulbar septum and in the aortopulmonary septum can cause ventricular septal defects and aortic-pulmonary windows. The left aortic cusp does not arise from the bulbar septum as do the right and non-coronary cusps and this theory could explain the paucity of congenital aneurysms from the left sinus.[14]

CLINICAL FINDINGS

Prior to rupture, patients are asymptomatic, although clinical findings may be present. Murmurs of an associated ventricular septal defect or aortic insufficiency due to prolapsed or fibrotic valve cusps may be heard. Conduction defects may also be present if aneurysms impinge on the atrioventricular node or bundle of His. When rupture occurs there may be sudden chest pain, but more commonly exertional dyspnea and easy fatigability are the presenting symptoms.[13,25] Some patients have ruptures without symptoms. Harsh to-and- fro murmurs are heard with maximum intensity at the left sternal border from the second to fourth interspace. Rupture usually occurs in young adults with a male predominance, but ruptures in the first decade have been reported.[15]

Surgical correction involves resection of the aneurysmal sac at its base with repair of the aneurysmal opening. It may be necessary to repair prolapsed cusps or replace fibrotic cusps with a prosthetic valve. Prognosis following corrective surgery is very good.

ROENTGENOGRAPHIC FINDINGS

Plain Roentgenogram. Prior to rupture, patients have a normal chest roentgenogram. This is understandable since the intracardiac positioned sinus of Valsalva is usually normal in size or only slightly enlarged and the aneurysms are directed into the right atrium or right ventricle. A reported case of unruptured aneurysm protruding into the right ventricular outflow tract produced marked decrease of the pulmonary vascularity on the plain chest roentgenogram.[17] Only two cases of calcification in congenital aneurysm have been reported. In one there was a fine curvilinear calcification just to the right of the spine on the posteroanterior projection.[22] A second case of calcification in a sinus of Valsalva aneurysm projecting into the right atrium was thought to be a calcified atrial tumor on the preoperative plain chest roentgenogram.[2]

After rupture occurs, there is a variable increase in size of the right heart, main pulmonary artery, and pulmonary vasculature, depending on the size of the shunt. Shunts at the atrial level result in right atrial and right ventricular enlargement. Ventricular shunts may also enlarge both right heart chambers (Fig. 3). Fluroscopy may show increased pulsations of the pulmonary vasculature. Following corrective surgery the heart returns to normal size.

Aortography. The sinuses are best visualized in the left anterior oblique projection. There may be a globular enlargement of the sinus or a localized aneurysmal sac arising from the sinus. The sac frequently has a windsock type of configuration (Fig. 4). Contrast medium enters a heart chamber, sometimes with a jet effect, resulting in opacification of the chamber and, on subsequent roentgenograms, opacification of the main pulmonary artery. Occasionally the sinus may appear normal and only opacification of a right heart chamber will occur. In this situation it is important to differentiate a perforation of the sinus from aortic insufficiency associated with membranous ventricular septal defect—a condition which would also opacify the right ventricle.

ANEURYSMS OF THE SINUSES OF VALSALVA WITH CYSTIC MEDIAL NECROSIS WITH OR WITHOUT MARFAN'S SYNDROME

Idiopathic cystic medial necrosis of the

aorta histologically is characterized by fragmentation and rarefaction of elastic fibers and mucoid degeneration in the aortic media. This results anatomically in dilatation of the aortic root and ascending aorta. The brachiocephalic portion of the aorta is normal. Patients with Marfan's syndrome and aortic involvement have identical histologic and anatomic findings. A *forme fruste* of Marfan's syndrome has been described in patients without the typical Marfan's phenotype but with a familial history of Marfan's syndrome and identical anatomic and histologic findings at the aortic root.[16,28] Cystic medial necrosis with aneurysms of the sinuses have also been reported in the hereditary connective tissue disorders, Ehlers-Danlos syndrome,[27] and osteogenesis imperfecta.[4,12] All of the aneurysms in this group are indistinguishable pathologically and roentgenologically, although Marfan's syndrome may also be associated with abnormalities of the mitral valve, pulmonary trunk, and pulmonary vasculature.

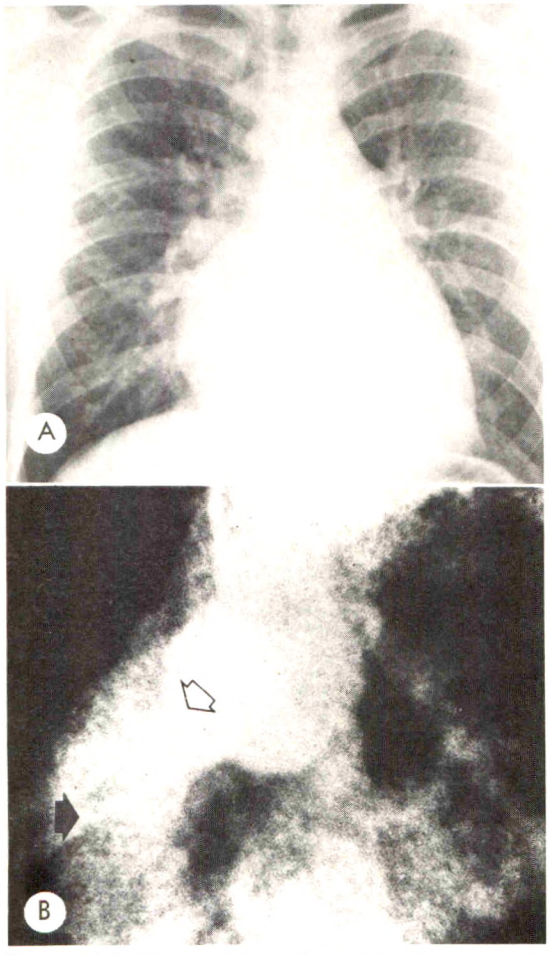

Fig. 3. (*A*) Twenty year old male with two week history of shortness of breath and pain over the precordium. Chest roentgenogram one year previously was normal. Note the right atrial enlargement and increased vasculature. (*B*) Aortography in left anterior oblique projection demonstrates a rupture of the right sinus (open arrow) into the right ventricle (black arrow).

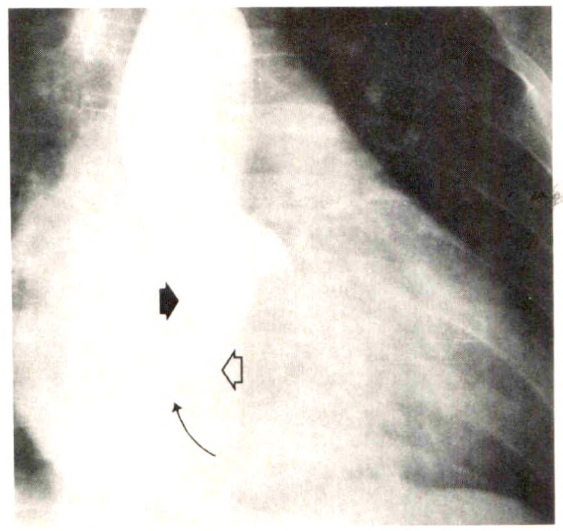

Fig. 4. Thirty-nine year old male with a four month history of progressive exertional dyspnea. Aortogram shows slight dilatation of the right sinus (black arrow) with a small aneurysm (open arrow). A jet of contrast material (curved arrow) flows into a dilated right atrium.

CLINICAL FINDINGS

Clinically these patients are young adults with exertional dyspnea, left ventricular failure, and precordial pain suggesting angina pectoris.[16] Occasionally an enlarged heart discovered by a chest roentgenogram, or murmurs heard on a routine physical examination, will lead to further evaluation in asymptomatic patients.

On physical examination the patients have a wide pulse pressure and a diastolic murmur due to aortic valve incompetence. Physical characteristics of Marfan's syndrome may or may not be present.

The surgical treatment should consist of

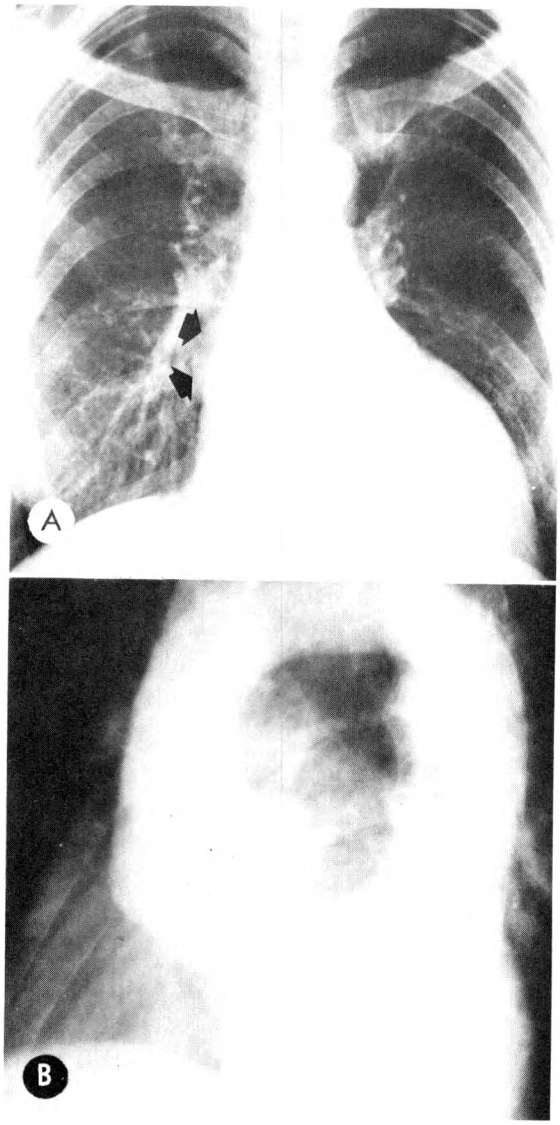

FIG. 5. (*A*) Thirty-three year old male with Marfan's syndrome. There are left ventricular enlargement and a mass overlying the superior right heart border (arrows). (*B*) Aortogram in left anterior oblique projection shows uniform dilatation of the sinuses and regurgitation into a dilated left ventricle.

resection of the aneurysm and replacement with a tubular prosthetic graft, and aortic valve replacement with a rigid prosthetic valve. Free implantation of the right coronary artery into the graft may be necessary if the artery arises from the aneurysm.[21] Patients have a good prognosis with surgical correction.

ROENTGENOGRAPHIC FINDINGS

Plain Roentgenograms. The two characteristic findings in medial necrosis of the aorta are left ventricular enlargement and dilatation of the ascending aorta. If the aortic abnormality is confined to the sinuses, which are intracardiac, then the aorta will usually show no abnormality on the plain roentgenogram, although left ventricular enlargement will still occur because of aortic insufficiency. Marked enlargement of the sinuses without aortic dilatation will produce a localized mass at the upper right heart border (Fig. 5). The extent of prominence of the ascending aorta depends on the extent of the aneurysmal aortic dilatation. In cases of extreme dilatation, the right heart silhouette forms an uninterrupted curve with the ascending aorta (Fig. 6*A*), and this configuration is usually associated with aorta dissection.[8] In some cases prominence of the ascending aorta is detected only on left anterior oblique and lateral views, especially in patients with Marfan's disease with kyphoscoliosis and levoposition of the heart. Calcification of the sinuses has not been reported but has been observed in the ascending aorta associated with chronic dissection of the aorta.[8]

As Eisen and Elliott[8] have observed, chest roentgenograms of patients with medial necrosis are frequently misinterpreted as representing rheumatic or congenital aortic valve disease. In medial necrosis, however, the ascending aorta is much larger than in rheumatic or congenital aortic valve incompetence. In addition, patients with medial necrosis do not have left atrial enlargement which would be expected in many cases of rheumatic heart disease.

Aortography. The characteristic findings at aortography are dilatation of all three aortic sinuses and aortic valve incompetence. The aorta above the sinuses may be normal or there may be dilatation. This may become so extensive that the aortic sinuses become effaced and no longer discernible (Fig. 6*B*). There is equal dilatation

of each sinus with smooth symmetric dilatation of the ascending aorta. The symmetric dilatation is understandable since the pathologic process of medial necrosis involves the whole circumference of the aorta, unlike the localized defect of congenital aneurysm or the unequally distributed disease of acquired aneurysm.

Although dilatation may be limited roentgenographically to the sinuses in some patients with Marfan's syndrome and cystic medial necrosis, it should not be thought that the disease process is confined to the sinuses. A generalized process of the ascending aorta is present even though the gross morphological changes have occurred only at the sinuses.

Dissection is a frequent angiographic finding and usually has an atypical appearance.[8] Thin radiolucent lines arising from the aortic walls most frequently originating just above the aortic valve cusps represent thin flaps of tissue fragmented from the aortic wall. Even though a dissection is present, the smooth contour and symmetric dilatation of the aortic wall are maintained. Less commonly, dissection is diffuse and indistinguishable from dissection associated with hypertension or trauma. This diffuse type of dissection may extend beyond the aortic arch to involve the descending thoracic and abdominal aorta, and may also calcify.

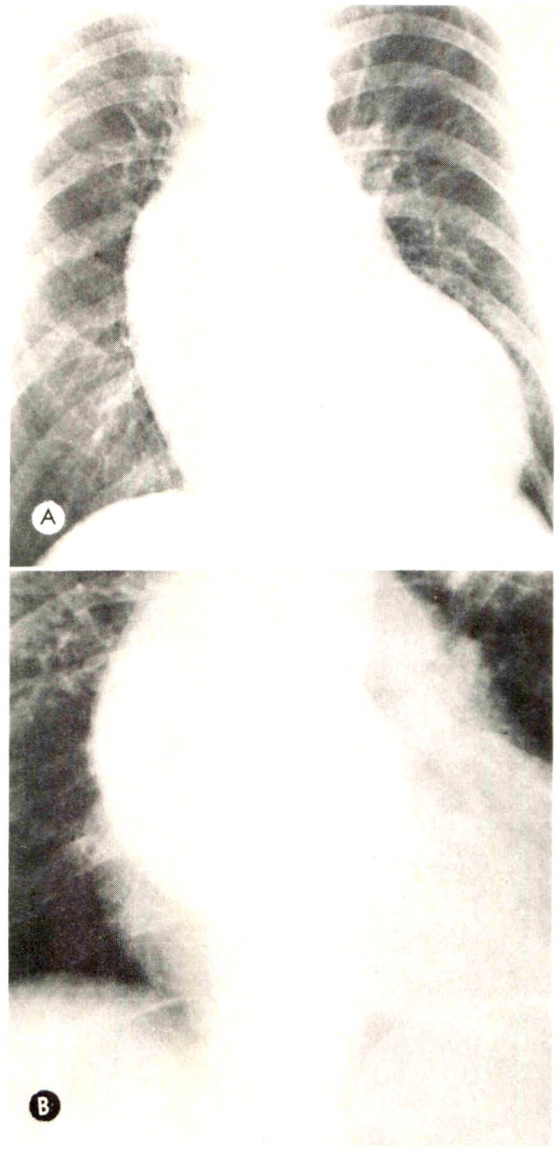

Fig. 6. (*A*) Forty-two year old male with Marfan's syndrome. Posteroanterior chest roentgenogram demonstrates left ventricular enlargement and marked prominence of the ascending aorta forming a continuous curve with the right heart border. (*B*) Anteroposterior aortogram shows marked symmetric dilatation of all sinuses and ascending aorta with effacement of the sinuses. Note the smooth inner walls of the aorta.

ACQUIRED ANEURYSMS

CLINICAL FINDINGS

Acquired aneurysms may be due to atheromatous degeneration of the aortic root, syphilitic aortitis, or nonsyphilitic infectious aortitis. The aneurysm *per se* usually produces no symptoms unless rupture occurs. Since syphilitic aneurysms attain a large size, extracardiac rupture and sudden death can occur. Rupture can also occur into the right atrium, right ventricle, and pulmonary artery, resulting in a left-to-right shunt which may not be immediately fatal. Aortic valve insufficiency which almost always accompanies syphilitic aneurysms, and which is less frequently associated with atheromatous aneurysms, eventually results in symptoms of congestive failure. In syphilitic patients the cause of death is usually congestive failure due to valve disease rather than rupture of an aneurysm. Patients may survive for long

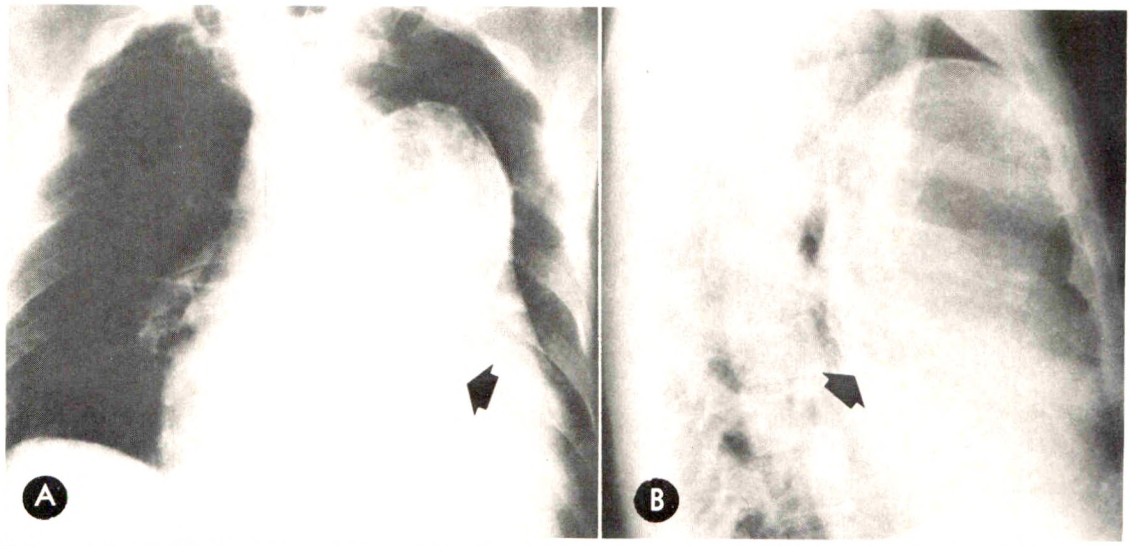

FIG. 7. (*A* and *B*) Seventy-two year old male with a history of syphilis. There is a large calcified mass extending superiorly and to the left. There are left ventricular enlargement and calcification of a dilated left sinus (arrows). At aortography there was a large aneurysm filled with thrombus extending from the left sinus.

periods of time with large syphilitic aneurysms if their failure can be controlled medically.[18] Atheromatous aneurysms may also remain unchanged for many years.

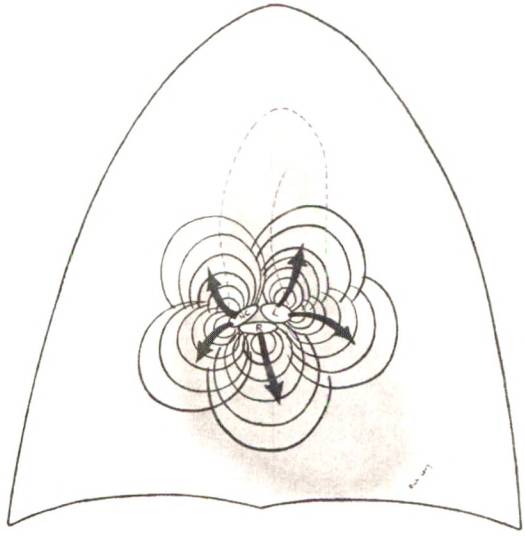

FIG. 8. Diagram of a posteroanterior chest roentgenogram, demonstrating potential location for extracardiac acquired aneurysms. The base of each arrow shows the specific site of origin in the sinuses for the different aneurysm masses.

R = right coronary sinus, L = left coronary sinus, NC = non-coronary sinus.

Occasionally precordial pain is a presenting symptom in patients with acquired aneurysms, presumably due to occlusion of coronary ostia by large aneurysms. Clinically, diastolic aortic murmurs and wide pulse pressures occur, and machinery murmurs are heard after rupture.

Since surgical correction of syphilitic aneurysms is difficult and since rupture may not occur for many years, conservative medical management is recommended.

ROENTGENOGRAPHIC FINDINGS

Plain Roentgenogram. Syphilitic aneurysms may be present for many years, so it is not surprising that they can attain a very large size and produce masses that are extracardiac in location (Fig. 7, *A* and *B*). The site of origin of the aneurysm can usually be predicted by the location of the mass[19] (Fig. 8). Aneurysms arising from the right sinus project anteriorly and can be best seen on the lateral roentgenogram. They may also cause erosion of the sternum.[19] Aneurysms from the anterior portion of the non-coronary sinus project anterolaterally to overlie the right atrium. Aneurysms from the posterior portion of the non-coronary sinus extend toward the

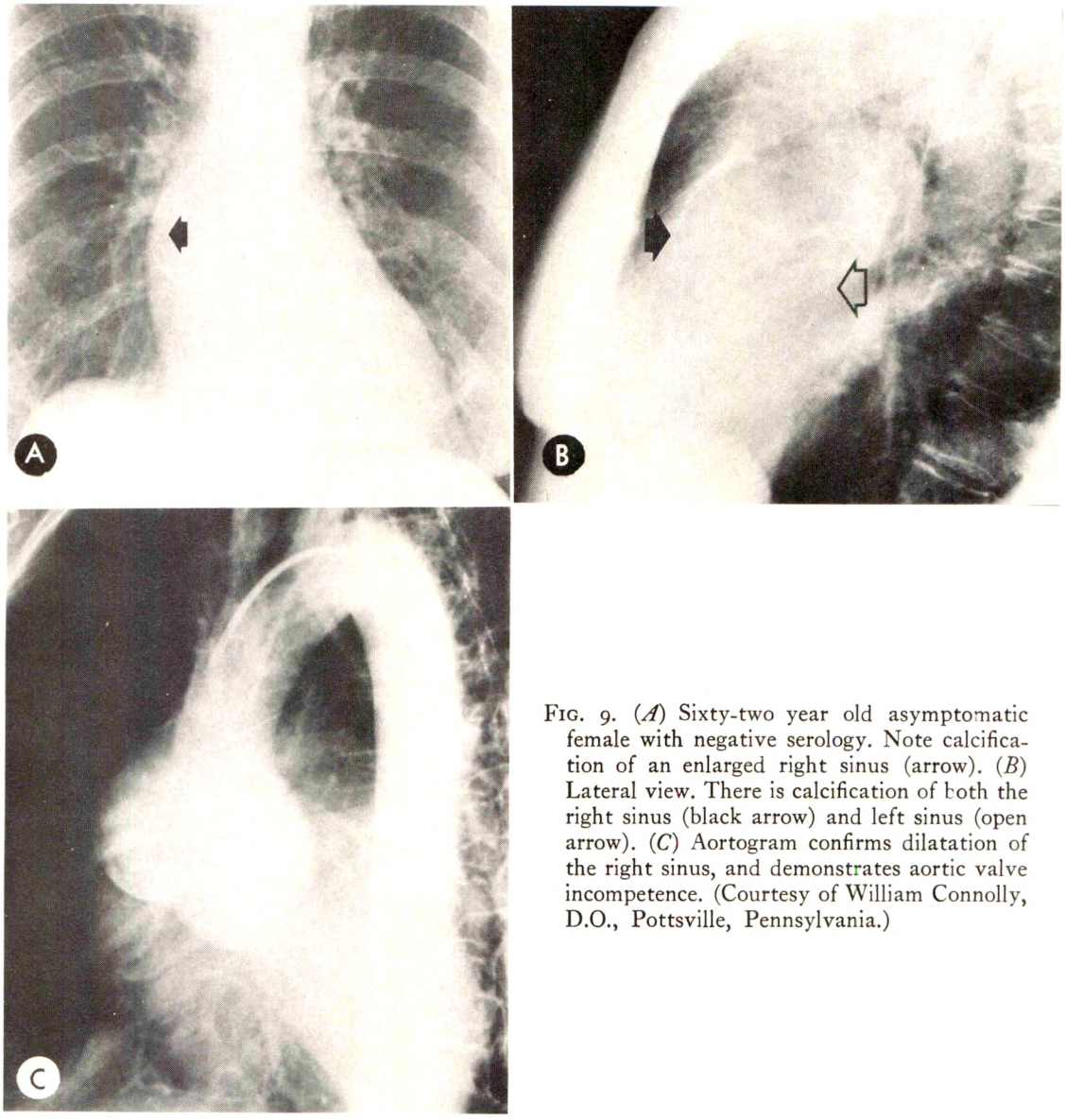

Fig. 9. (*A*) Sixty-two year old asymptomatic female with negative serology. Note calcification of an enlarged right sinus (arrow). (*B*) Lateral view. There is calcification of both the right sinus (black arrow) and left sinus (open arrow). (*C*) Aortogram confirms dilatation of the right sinus, and demonstrates aortic valve incompetence. (Courtesy of William Connolly, D.O., Pottsville, Pennsylvania.)

left atrium. Aneurysms from the anterior left sinus extend beyond the pulmonary outflow tract. Aneurysms from the posterior left sinus extend superiorly and to the left in the posteroanterior projection. Unlike congenital aneurysms, syphilitic aneurysms occur with equal frequency in each sinus. Atheromatous aneurysms occur more frequently in the right sinus.

In addition to a large mass, calcification is a hallmark of acquired aneurysms. Syphilitic aneurysms have extensive calcification of the ascending aorta and aneurysmal mass. Aneurysms may be misdiagnosed as calcified dermoids, if the ascending aorta is not carefully evaluated.[11] A calcified subannular aneurysm of the left ventricle can also simulate a syphilitic aneurysm.[26] If the aneurysm is small and no extracardiac mass is present, the calcification may extend into the heart and outline a dilated sinus or sinuses. Atheromatous aneurysms calcify,[20] but the calcification is usually less extensive than syphilitic aneurysms (Fig. 9, *A* and *B*). Left ventricular enlargement is almost always present with syphilitic aneurysms,

but is less likely to occur with atheromatous aneurysms. Although not yet reported, rupture into the lesser circulation should result in enlargement of the right heart and pulmonary vasculature.

Aortography. Dilatation of the ascending aorta and saccular aneurysms of the sinus are seen in the syphilitic type. Since there is usually unequal extent of involvement pathologically with syphilitic aortitis, the symmetric dilatation of cystic medial necrosis is not present. Instead, there are marked irregularities of contour, and thrombus formation may prevent much of the aneurysm from filling with contrast material. In some cases the extent of the aneurysm may be estimated only by the location of its calcified wall. Atheromatous aneurysms may be quite smooth (Fig. 9*C*), and are usually smaller than syphilitic aneurysms. Angiographic visualization of syphilitic aneurysm rupture into the lesser circulation has not yet been reported. Aortic regurgitation is usually present in acquired aneurysms.

SUMMARY

The different types of aneurysms of the sinus of Valsalva are discussed along with a brief description of the anatomy of the sinuses. The clinical, plain roentgenographic, and angiographic findings which differentiate the different types of aneurysms are stressed.

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REFERENCES

1. Barrett, J. S., Helwig, J., Jr., Kay, C. F., and Johnson, J. Cine-aortographic evaluation of aortic insufficiency. *Ann. Int. Med.*, 1964, *61*, 1071–1083.
2. Björk, V. O., and Edhag, O. Aortic sinus aneurysm. *Scand. J. Thoracic & Cardiovasc. Surg.*, 1972, *6*, 136–139.
3. Cleveland, R. J., Nelson, R. J., Nedelman, A. L., Criley, M., and Lippmann, M. Surgical management of multiple aneurysms of sinuses of Valsalva. *A.M.A. Arch. Surg.*, 1971, *103*, 44–47.
4. Criscitello, M. G., Ronan, J. A., Jr., Besterman, E. M. M., and Schoenwetter, W. Cardiovascular abnormalities in osteogenesis imperfecta. *Circulation*, 1965, *31*, 255–262.
5. Davidsen, G., Petersen, O., and Thomsen, G. Roentgenologic findings in five cases of congenital aneurysm of aortic sinuses (sinuses of Valsalva). *Acta radiol.*, 1958, *49*, 205–217.
6. DeBakey, M. E., Diethrich, E. B., Liddicoat, J. E., Kinard, S. A., and Garrett, H. E. Abnormalities of sinuses of Valsalva. *J. Thoracic & Cardiovasc. Surg.*, 1967, *54*, 312–332.
7. Edwards, J. E., and Burchell, H. B. Pathological anatomy of deficiencies between aortic root and heart, including arotic sinus aneurysms. *Thorax*, 1957, *12*, 125–139.
8. Eisen, S., and Elliott, L. P. Roentgenology of cystic medial necrosis of ascending aorta. *Radiol. Clin. North America*, 1968, *6*, 437–449.
9. Eliot, R. S., Wolbrink, A., and Edwards, J. E. Congenital aneurysm of left aortic sinus: rare lesion and rare cause of coronary insufficiency. *Circulation*, 1963, *28*, 951–957.
10. Flowers, N. C., Horan, L. G., and Copeland, G. D. Congenital aneurysms of all three sinuses of Valsalva with rupture in asymptomatic patient. *Am. J. Med.*, 1966, *40*, 642–650.
11. Harris, E. J. Aneurysms of sinus of Valsalva. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1956, *76*, 767–772.
12. Heppner, R. L. Babbit, H. I., and Bianchine, J. W. Aortic regurgitation and aneurysm of sinus of Valsalva associated with osteogenesis imperfecta. *Am. J. Cardiol.*, 1973, *31*, 654–657.
13. Howard, R. J. Moller, J. C., Castaneda, A., Varco, R. L., and Nicoloff, D. M. Surgical correction of sinus of Valsalva aneurysm. *J. Thoracic & Cardiovasc. Surg.*, 1973, *66*, 420–427.
14. Jones, A. M., and Langley, F. A. Aortic sinus aneurysms. *Brit. Heart J.*, 1949, *11*, 325–341.
15. Kakos, G. S., Kilman, J. W., Williams, T. E., and Hosier, D. M. Diagnosis and management of sinus of Valsalva aneurysm in children. *Ann. Thoracic Surg.*, 1974, *17*, 474–478.
16. Keene, R. J., Steiner, R. E., Olsen, E. J. G., and Oakley, C. Aortic root aneurysm—radiographic and pathologic features. *Clin. Radiol.*, 1971, *22*, 330–340.
17. McLaren, J. R., and Baylin, G. J. Two cases of aneurysms of sinus of Valsalva with interesting roentgen changes. *Radiology*, 1958, *70*, 362–367.
18. Merten, C. W. Finby, N., and Steinberg, I. Antemortem diagnosis of syphilitic aneurysm

of aortic sinuses. *Am. J. Med.*, 1956, *20*, 345–360.

19. Ostrum, H. W. Robinson, B. D., Nichols, C. F., and Widmann, B. P. Aneurysms of aortic sinuses or sinus of Valsalva. Am. J. Roentgenol. & Rad. Therapy, 1938, *40*, 828–837.
20. Reinke, R. T., Coel, M.N., and Higgens, C. B. Calcified nonsyphilitic aneurysms of sinuses Valsalva. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *122*, 783–787.
21. Ross, D. N., Frazier, T. G., and Gonzalez-Lavin, L. Surgery of Marfan's syndrome and related conditions of aortic root (annulo-aortic ectasia). *Thorax*, 1972, *27*, 52–57.
22. Rowley, J. C., and Holmes, R. B. Sinus of Valsalva aneurysms. *J. Canad. A. Radiologists*, 1965, *16*, 254–261.
23. Sakakibara, S., and Konno, S. Congenital aneurysm of sinus of Valsalva: anatomy and classification. *Am. Heart J.*, 1962, *63*, 405–424.
24. Steinberg, I., and Geller, W. Aneurysmal dilatation of aortic sinuses in arachnoidactyly: diagnosis during life in three cases. *Ann. Int. Med.*, 1955, *43*, 120–132.
25. Taguchi, K., Sasaki, N., Matsuura, Y., and Uemura, R. Surgical correction of aneurysm of sinus of Valsalva. *Am. J. Cardiol.*, 1969, *23*, 180–191.
26. Thoms, N. W., Wilson, R. F., Puri, P., and Arbulu, A. Cricular calcification in mediastinum. *Chest*, 1973, *63*, 425–426.
27. Tucker, D. H., Miller, D. E., and Jacoby, W. J. Ehlers-Danlos syndrome with sinus of Valsalva aneurysm and aortic insufficiency simulating rheumatic heart disease. *Am. J. Med.*, 1963, *35*, 715–720.
28. Weaver, W. F., Edwards, J. E., and Brandenberg, R. O. Idiopathic dilatation of aorta with valvular insufficiency: possible *forme fruste* of Marfan's syndrome. *Proc. Staff Meet. Mayo Clin.*, 1959, *34*, 518–522.
29. Yacoub, M. H., Lise, M., and Muir, J. Aneurysms of two sinuses of Valsalva with ventricular septal defect and aortic regurgitation. *Brit. Heart J.*, 1969, *31*, 661–663.

ANGIOGRAPHIC FEATURES OF CIMINO-BRESCIA FISTULAS*

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ABSTRACT:

Forty-four patients with operatively created arteriovenous fistulas have been examined angiographically. Widening of the main arteries of the forearm was evident, the most pronounced widening occurring in the artery feeding the fistula. Stenosis of the arteries or veins seldom influenced the flow significantly. Blood from the fistula was often partly or totally directed into the hand. In nine patients filling of deep veins was evident, and in most of these cases big fistulas were present. A rough estimate of the blood supply to the hand indicated poor supply in eight patients of which only one had symptoms of ischemia.

Brachial angiography is of value in estimation of the blood flow in fistulas, when cannulation of veins is difficult, when thrombosis is suspected, or when a new fistula is planned after decreased function of an old one.

ANGIOGRAPHY is a readily available examination for evaluation of operatively constructed arteriovenous shunts and fistulas. Several reports have appeared regarding Quinton-Scribner shunts, but the angiographic features of Cimino-Brescia fistulas have been described more extensively in only one report, coming from our hospital.[3] As the number of angiographic examinations is now more than doubled, a more quantitative analysis is possible and is considered worth reporting.

MATERIAL AND METHOD

In our hospital arteriovenous fistulas have been constructed for hemodialysis or intravenous therapy in about 400 patients during an eight year period. We use the surgical technique described by Brescia *et al.*[1] with some modifications.[4] Forty-four of these patients, 29 males and 15 females with an age range of 24–85 years (mean 53 years), have been examined by angiography 49 times. The interval between operation and angiography was one week to two years. Of the examined anastomoses 32 were side-to-side, eight end-to-side (artery to vein), one side-to-end (artery to vein), one directly end-to-end, and three end-to-end with interposed vein grafts. The vessels involved are presented in Table 1.

The usual reason for the angiographic examination was difficulty in cannulation of the arterialized veins, but in some cases it was that local or general circulatory symptoms indicated large fistula flow. In the latter cases the angiography was combined with flow studies using a dye-dilution technique, the results of which will be reported elsewhere.

The examination technique has varied somewhat. In most cases retrograde percutaneous catheterization of the brachial artery was performed just above or at the elbow level. After application of local anesthesia, a polyethylene catheter (PE 160, ID/OD 1.14/1.57 mm) with a length of about 20 cm was introduced. Ten to twenty ml meglumine metrizoate (Isopague Cerebral, Nyegaard, Norway) were injected by hand or by a high pressure syringe. The usual series was ten films for five seconds using an Elema-Schönander cut film changer. In some cases one series in anteroposterior projection was obtained with compression of the veins of the upper arm using a blood pressure cuff inflated to a pressure of 30 mm Hg and manual com-

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Table I

Vessels Anastomosed	No. of Patients
Radial artery—cephalic vein	33
Radial artery—subcutaneous vein	4 (one with a graft)
Radial artery—basilic vein	1 (with graft)
Ulnar artery —basilic vein	3
Ulnar artery —subcutaneous vein	1 (with graft)

pression of the axillary artery. One series in lateral projection without compression was made.

The measurements of vessel diameters and fistula widths have been made on films obtained without compression, the geometric magnification being about 1.1.

RESULTS

The mean diameter of the radial artery at its origin was 6.2 mm (range 3.0–11.0, S.D. 1.98 mm), decreasing to a mean of 5.2 mm (2.0–9.0, S.D. 1.69) 2 cm proximal to the fistula. In 18 roentgenograms there was flow into the radial artery distal to the fistula, the mean diameter being 3.8 mm (2.0–7.0, S.D. 1.21). The mean diameter of the ulnar artery at its origin was 4.3 mm (2.0–10.5, S.D. 1.76) and the mean of the interosseous artery 3.3 mm (1.5–8.0, S.D. 1.39). There were few normal brachial angiograms available in the files of this hospital and examination of the contralateral arm was not performed; thus no normal values can be presented.

The mean diameter of the anastomosed vein (or the main channel when more than one vein was dilated) 2 cm proximal to the fistula was 7.1 mm (3.0–10.5, S.D. 2.28), distal to the fistula 6.0 mm (2.3–11.0, S.D. 2.41). The width of the fistula was difficult to measure in some cases, but in the 19 measured patients the mean was 2.5 mm (0.5–10.0, S.D. 3.42). There was stenosis of the feeding artery (two ulnar arteries) proximal to the fistula (Fig. 3) in 15 patients and distal to the fistula in three (Fig. 5). Stenosis of the main vein proximal to the fistula (Fig. 8) was evident in seven patients, distal to the fistula in ten (Fig. 6). Occlusion of the feeding artery (Fig. 13)

Fig. 1. Bridge graft of the saphenous vein interposed between the radial artery and the basilic vein. Despite the irregularities in the fistula and the stenosis of the artery close to it there is a good flow in the arterialized vein.

Table II

Function of Arteries to the Hand

Arteries Filled	No. of Patients
Radial+ulnar+interosseous	16
Radial+interosseous	3
Radial+ulnar	1
Radial	2
Ulnar+interosseous	9
Ulnar	9
Not possible to determine	9

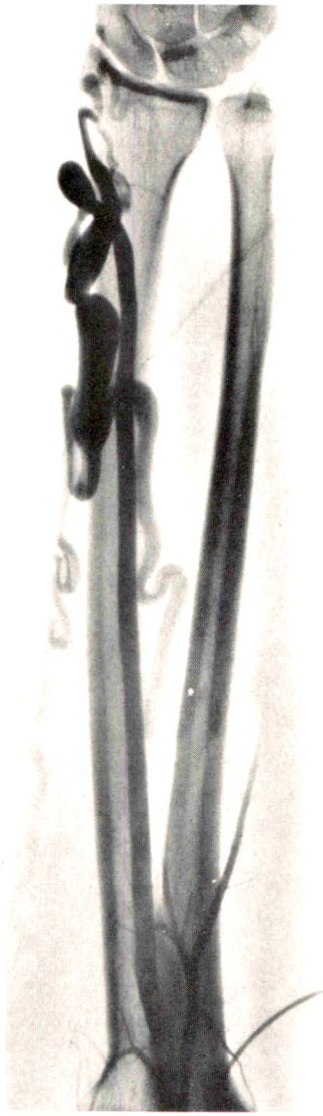

FIG. 2. Side-to-side fistula between the radial artery and a small volar vein. The shunt is functionally an end-to-end one. Twenty weeks after operation there are two dilated, tortuous veins.

was encountered in two patients and occlusion of the main vein in three patients (Fig. 6). The number of dilated venous channels varied from one to more than five, and in most cases the anastomosed vein showed the most pronounced dilatation. In eleven patients there were dilated veins only in the arm, in 28 in both arm and hand (Fig. 4), and in two only in the hand. The arterial supply of the hand, roughly estimated, was good in 25 angiograms, decreased in nine, bad in eight, and was not possible to evaluate in seven angiograms (Table II). Only one of the eight patients classified as bad had clinical evidence of a poor blood supply.

The circulation time in the artery feeding the fistula and the emptying time of the main draining vein are presented in Table

FIG. 3. Radial artery and cephalic vein anastomosed side-to-side. Due to incompetent valves, blood is flowing both distally and proximally. This is the most common flow pattern encountered in our material.

Table III

THE FLOW RATE IN ARTERIES AND VEINS

Artery	Time (sec.)	No. of Patients
Elbow—fistula	<0.5	17
	<1.0	2
	>1.0	2
	>2.5	1
Vein		
Fistula—elbow	<0.5	8
	<1.0	4
	>1.0	3

III. The low arterial flow rate in five patients was due to severe arterial stenosis with a functioning fistula or to a nonfunctioning fistula with impairment of the artery. The decreased flow in the main draining vein in three patients was due to severe stenosis or thrombosis, except for one case where all blood from the fistula was directed into the hand.

Table IV

MORPHOLOGY AND POSSIBLE FUNCTION OF CIMINO-BRESCIA SHUNTS

A. Anatomical side-to-side anastomosis
   1. Functionally end-to-side
      No contrast medium distally in the artery
      Contrast medium in both directions in the vein
   2. Functionally end-to-end
      No contrast medium distally in artery or vein
      No contrast medium distal artery or proximal vein
   3. Functionally side-to-end
      Contrast medium proximally and distally in artery
      No contrast medium distally in vein
      No contrast medium proximally in artery

B. Anatomical end-to-end
   1. Direct anastomosis artery-vein
   2. Graft artery-vein

C. Anatomical end-to-side
   1. Functional end-to-side
      No contrast medium distally in artery
   2. Functional end-to-end
      No contrast medium distally in vein
      No contrast medium proximally in vein

D. Anatomical side-to-end
   1. Functional side-to-end
      Contrast medium distally in artery
   2. Functional end-to-end
      No contrast medium proximally in artery

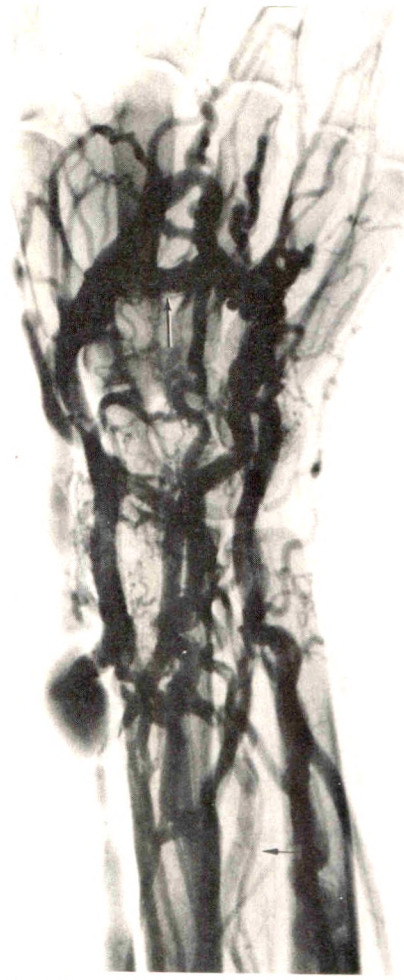

Fig. 4. Anastomosis side-to-side between the radial artery and the cephalic vein with blood flowing mainly distally in the veins. Abundant arterial and venous collaterals and filled deep veins (arrows). Stenosis of the cephalic vein proximal to the anastomosis and dilatation distal to it.

Schematically the shunts can be divided according to the morphology and function as presented in Table IV. The most common type of anastomosis was side-to-side between the radial artery and the cephalic vein, usually appearing to be an end-to-side anastomosis (Fig. 3). The second most commonly occurring was the same anastomosis with all four limbs filled but only

FIG. 5. Radial artery and cephalic vein anastomosed side-to-side. No flow proximally in the cephalic vein. Retrograde flow in dilated veins in the hand. Note the collateral supply from the ulnar artery.

small amounts of blood in the distal part of the artery (Fig. 4).

It was not uncommon for the blood in side-to-side fistulas to empty mainly in the distal direction of the vein (Fig. 5; 6; and 10). Such was the case in almost all the nine patients where deep veins of the hand and the arm were filled (Fig. 4; and 10).

DISCUSSION

The patients studied by angiography are selected, and the material is not representative of ordinary well-functioning fistulas. In spite of this, some general information is given about the angiographic pattern in patients with this type of fistula. Even if no normal material is available, it is obvious that the artery feeding the fistula is widened. The artery has also a fairly uniform diameter from its origin to the fistula, and the normal tapering-off is absent. Dilatation of the artery proximal to the fistula is a well known phenomenon, although the exact mechanism of its development is unknown. Aneurysmal degeneration of the artery, known from other types of arteriovenous fistulas, has been observed in one of our patients.

The artery distal to the fistula was sometimes filled by collaterals from the ulnar

FIG. 6. Side-to-side anastomosis radial artery to cephalic vein with poor function of the fistula. Blood flows only distally in the severely stenosed vein into the hand and through a superficial vein with severe stenosis at its origin. No impairment of the arterial blood flow.

(Fig. 7) or interosseous (Fig. 8) artery. Non-filling of this part of the artery might indicate obstruction of the artery but is more probably a sign of retrograde flow toward the fistula as has been observed in patients studied by other techniques.[5] In patients with small fistulas the artery distal to the fistula was filled in the normal direction.

In several cases even the other main arteries of the arm seem to be dilated, and their form is changed. These signs of increased blood flow indicate that the blood supply to the hand is taken over by these arteries, which can be clearly demonstrated in some cases (Fig. 7; and 8), and also of "steal" by the fistula from collateral arteries.[4]

Fig. 7. Well functioning fistula between radial artery and cephalic vein (side-to-side). There is abundant collateral supply from the dilated ulnar artery. There is a pronounced dilatation of the radial artery.

Fig. 8. Anastomosis side-to-side between the radial artery and the cephalic vein. No blood flow distally in the vein. There is an extensive collateral circulation from the grossly dilated interosseous artery.

Dilatation of the arterialized veins, eventually even venous aneurysms, are also well known and are evidently caused by the turbulence of blood flow. Like other authors[6] we have found the venous aneurysms just opposite the fistula (Fig. 4).

In most cases only one single venous

FIG. 9. Saphenous vein graft between radial artery and basilic vein. Well functioning anastomosis. Irregular narrowing of vein lumen (arrow).

channel was widened, even if several superficial veins were filled. In other cases several veins were dilated and were also used for cannulation.

Kaude and Lindstedt[3] found no filling of deep veins in the arm. In the present material, filling of deep veins has been observed in several cases, mainly in the hand but also in the arm (Fig. 4; and 10). The veins accompanying the interosseous artery are most often filled. Filling of deep veins was found in some cases where the blood flow from the fistula was mainly into the peripheral branch of the fistula vein. Deep veins in the forearm were most apparent in two cases with huge edematous swelling of the whole arm. In these patients several superficial veins in the middle third of the forearm were thrombosed. In these cases the superficial venous pressure was evidently so high that the valves of communicating veins to the deep system were made incompetent.

Stenosis of the artery just proximal to the fistula may be due to improper surgical technique, to perivascular scarring, or to angulation of the artery due to elongation. Such arterial stenosis impaired blood flow significantly in only one case (Fig. 12).

Stenosis of the central vein a few mm proximal to the fistula has been observed by several authors, and it occurs frequently in our material. It can be observed even at the fistula operation. It is probably caused by venous spasm at the site of temporary oc-

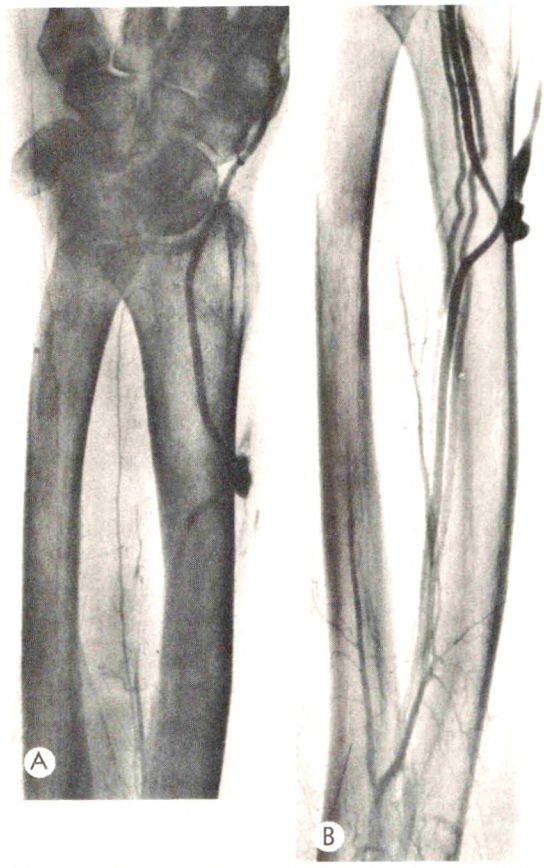

FIG. 10. (*A*) Non functioning shunt due to venous thrombosis. No evident impairment of arterial flow. (*B*) Partial recanalization of the distal part of the veins some weeks later. Note the deep veins along the radial artery.

clusion during operation. Negative pressure in this limb of the fistula[7] may contribute. More careful surgical technique and cutting the adventitia of the vein in this area seem to diminish the tendency to development of this stenosis. Even venous stenosis seems not to have great influence on fistula flow.

Poor function of a fistula was most often due to changes in the fistula itself. Repeated cannulation of the arterialized veins was tolerated well in most cases but could occasionally lead to poor function.

Non-function of a fistula was due to thrombosis of the feeding artery (Fig. 13) or the emptying vein (Fig. 6). A new shunt could be created using the ulnar artery and the basilic or other vein. A graft using a piece of the great saphenous vein (Fig. 9) was sometimes installed.

Fig. 11. (*A*) Side-to-side anastomosis between radial artery and cephalic vein. Three limbs at the anastomosis are filled. Moderate stenosis of the radial artery proximal to the shunt. (*B*) Thrombosis in the upper part of the cephalic vein, demonstrated by selective catheterization and injection.

Fig. 12. Anastomosis side-to-side between radial artery and hypoplastic vein with some blood going distally in veins.

Venous compression and digital occlusion of the axillary artery were performed in an attempt to give more detailed information of the anatomy, especially at the region of the anastomosis. These factors were only occasionally of significant value.

Brachial angiography is a simple method for study of the morphology of Cimino-Brescia fistulas and for obtaining a crude estimate of the fistula flow. More elaborate

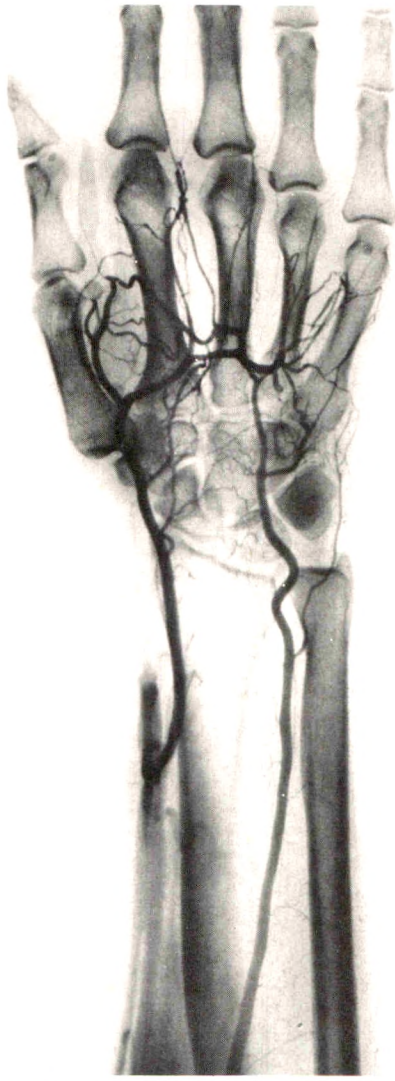

FIG. 13. Side-to-side anastomosis between radial artery and cephalic vein with thrombosis of the radial artery proximal to the fistula. The distal part of radial artery is filled by retrograde flow from the ulnar artery. The fistula is functioning but the patient experienced pain at hemodialysis, indicating ischemia.

flow studies can easily be performed in connection with the angiography using a dye-dilution technique.[2] The angiographic examination is not painful as most of the contrast medium is diverted through the fistula and thus the addition of local anesthetic agent to the contrast medium or anesthesia of the axillary plexus is not necessary.

Preoperative brachial angiography is sometimes desirable to clarify the anatomy. Arterial size, atherosclerotic changes, thrombosis, and collaterals can be evaluated. By using papaverine and local anesthesia before the injection of contrast medium some information regarding the veins may be obtained. If a fistula has ceased to function, angiography reveals eventual possibilities for creating a new fistula. With poorly functioning fistulas, brachial angiography provides the information for deciding which step should be taken.

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REFERENCES

1. BRESCIA, M. J., CIMINO, J. E., APPEL, K., and HURWICH, B. J. Chronic hemodialysis using venipuncture and surgically created arteriovenous fistula. *New England J. Med.*, 1966, 275, 1089–1092.
2. Göthlin, J., *et al.* To be published.
3. KAUDE, J., and LINDSTEDT, E. Angiographic studies in patients with Cimino-Brescia fistulae. *Scand. J. Urol. Nephrol.*, 1972, Suppl. 14, 23–32.
4. LINDSTEDT, E. Studies in therapeutic arteriovenous fistulae. *Scand. J. Urol. Nephrol.*, 1972, Suppl. 14.
5. LINDSTEDT, E., and WESTLING, H. Effects of antebrachial Cimino-Brescia arteriovenous fistula on local circulation in hand. To be published.
6. RÖHL, L., FRANZ, H. E. MÖHRING, K., RITZ, E., SCHULER, H. W., UHSE, H. G., and ZIEGLER, M. Direct arteriovenous fistula for hemodialysis. *Scand. J. Urol. Nephrol.*, 1968, *2*, 191.
7. SCHENK, W. G., MARTIN, J. W., LESLIE, M. B., and PORTIN, B. A. Regional hemodynamics of chronic experimental arteriovenous fistulas. *Surg., Gynec. & Obst.*, 1960, *110*, 44–50.

Vol. 125, No. 3

THE CERVICAL AORTIC ARCH*

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ABSTRACT:

Cervical aortic arch anomaly is a rare congenital entity. The aortic arch extends into the soft tissues of the neck before turning downward on itself to become the descending aorta. Nineteen cases have been previously reported in the world literature with one more presently in publication. We have collected six additional cases, in four of which the cervical arch was located on the left. Formerly the left cervical aortic arch was felt to be much less common than the right. It now appears that they have equal incidence. Four of our cases were also associated with cardiac anomalies, and this coincidence has not been previously reported.

DESPITE the rarity of the cervical aortic arch, and despite a frequently relatively benign or asymptomatic course, the entity gains importance in recognition because of the differential diagnosis it includes. Significantly, a pulsating mass on one or the other side of the neck has been associated with all reported cases, including our own. In at least six cases reported previously[4,7,14,18–20] as well as in two of our own, this mass was the only presenting sign. This could easily be mistakenly diagnosed as an aneurysm of the common carotid artery, another rare entity in childhood; in at least one case, the patient was taken to surgery and the "aneurysm" ligated, resulting in rapid death from congestive heart failure.[1]

When symptoms are encountered in children, they most frequently have been respiratory in nature; previously reported cases have most often mentioned stridor, frequent upper or lower respiratory infections, and dyspnea on exertion (Table I). We encountered one case with tetralogy of Fallot and a cervical arch in which there were respiratory symptoms that were considered mainly due to the arch anomaly (Table II).

Dysphagia is less often encountered as a complaint and is more often seen in those who have survived to adulthood with the cervical aortic arch. This also appears to be the primary symptom in adults with a vascular ring,[8] and perhaps it is not encountered more often in childhood because of the subjectiveness of the complaint, and the inability of children to recognize their own dysphagia as such. Also, possibly the dysphagia leads to frequent aspiration of small amounts of food, with the resulting respiratory symptoms overshadowing the dysphagia which led to the pneumonitis.

Other occasionally mentioned complaints are headaches (in two previously reported cases),[4,6] dizziness and fainting (in three cases previously reported)[4,10,14] and pain between the shoulder blades (in one previously reported case)[6] (Table I). Conceivably, the fainting could have been related to the subclavian steal syndrome in that patient whose left subclavian artery arose anomalously from the descending aorta and was stenotic at its origin. No reference to this possibility was made in the case report, however. In our own two cases, with only the cervical arch abnormality, both had supraclavicular pulsating masses, without other complaints. In our other four cases, the symptomatic cardiac anomalies coexisting with the cervical aortic arch overshadowed complaints arising only from the

* The material from this paper was presented as a Scientific Exhibit at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.

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TABLE I

PRESENTING SYMPTOMS AND SIGNS OF CASES PREVIOUSLY REPORTED

Author	Year	Signs and Symptoms
Reid[16]	1913	Not known
Beavan and Fatti[1]	1947	Dyspnea, dysphagia
Lewis and Rogers[10]	1953	Dyspnea, dysphagia, syncope
Gravier *et al.*[7]	1959	Pulsating mass
Massumi *et al.*[13]	1963	Stridor; frequent respiratory infections
Mahoney and Manning[12]	1964	Recurrent respiratory infections
D'Cruz *et al.*[4]	1966	Pulsating mass
D'Cruz *et al.*[4]	1966	Headaches
Lipchick and Young[11]	1967	Recurrent bronchitis
Deffrenne and Verney[5]	1968	Symptoms of a vascular ring
DeJong and Klinghamer[6]	1969	Headache, dizziness, pain between shoulder blades
Shepherd *et al.*[18]	1969	Pulsating mass
Chang *et al.*[3]	1971	Recurrent pneumonitis; pulmonary hypertension
Yigitabasi and Malbantgil[20]	1971	Pulsating mass
Shuford *et al.*[19]	1972	Pulsating mass
Richie *et al.*[17]	1973	Hoarseness and dysphagia
McCue *et al.*[14]	1973	Pulsating mass
McCue *et al.*[14]	1973	Recurrent bronchitis; dizziness

latter anomaly (Table II).

ROENTGENOGRAPHIC EXAMINATION

Roentgenographic differentiation of cervical arch from carotid artery aneurysms and vascular rings can usually be made on the plain chest film alone. The signs include:

1. Absence of normal aortic knob (Fig. 1).
2. Apparent cut-off of the tracheal air column in the superior mediastinum (Fig. 1).
3. The position of the aorta which sometimes descends along the contralateral side of the superior mediastinal mass, particularly with right-sided cervical arches (Fig. 1).
4. Superior mediastinal widening (Fig. 2).
5. Displacement of the trachea to the opposite side of the superior mediastinal mass, and on the lateral film, *en bloc* displacement of a segment of trachea, rather than a simple notching, differentiating it from tracheal compression due to vascular ring (Fig. 3).

Furthermore, plain films of the soft tissues of the neck may demonstrate a loss of the normal cervical lordosis and an increase in the retropharyngeal space if the arch ascends high enough (Fig. 4).

A barium swallow examination will demonstrate also an *en bloc* esophageal displacement, but toward, rather than away from,

TABLE II

PRESENTING SIGNS AND SYMPTOMS AND ASSOCIATED CARDIAC ANOMALIES OF PRESENT CASES

	Presenting Signs and Symptoms	Age at Presentation	Coexisting Cardiac Anomalies
Case I	Pulsating mass	30 years	None
Case II	Small mass; abnormal chest roentgenogram	10 years	None
Case III	Dyspnea, dysphagia	18 months	Tetralogy of Fallot
Case IV	Cyanosis, clubbing of digits	2 years	Pseudotruncus
Case V	Cyanosis, congestive heart failure	3 months	Double outlet right ventricle
Case VI	Recurrent pneumonitis	4 months	Ventricular septal defect

the abnormally placed arch (Fig. 5). This tendency of the esophagus to follow the path of the descending aorta is also noted in the aortic tortuosity produced by arteriosclerosis in the elderly. It may be secondary to short esophageal branches from the descending aorta. On posteroanterior roentgenograms with barium in the esophagus, sometimes an oblique defect in the barium column may be noted, representing the aorta crossing retroesophageally to descend on the opposite side of the arch (Fig. 6). On lateral roentgenograms the compression of the esophagus from behind may be high or low, depending upon the level at which the aorta crosses the mediastinum (Fig. 7, *A* and *B*). If there is an aberrant subclavian artery, this may be seen as another esophageal filling defect on barium swallow at an acute angle to the defect produced by the descending aorta.

Angiography will demonstrate the redundant aortic arch at any level from

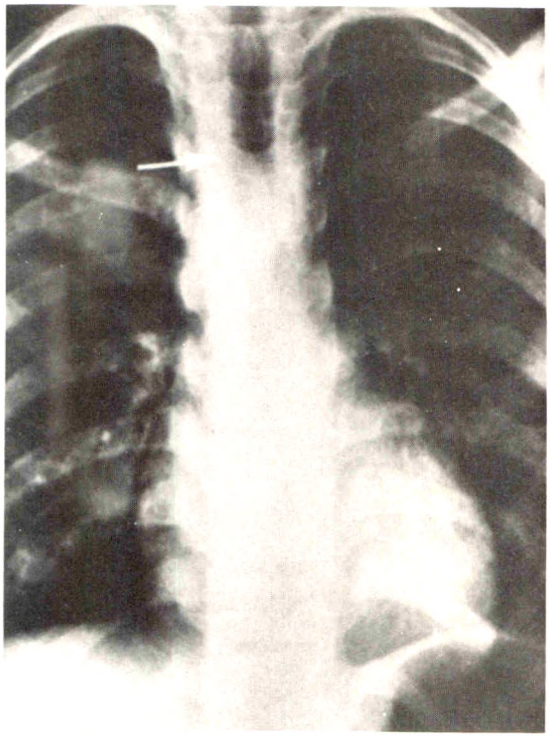


Fig. 1. A case of relatively low-positioned right-sided cervical aortic arch demonstrating absence of the aortic knob and an apparent cut-off of the tracheal air column at the point of crossing of the descending aorta from right to left (arrow).

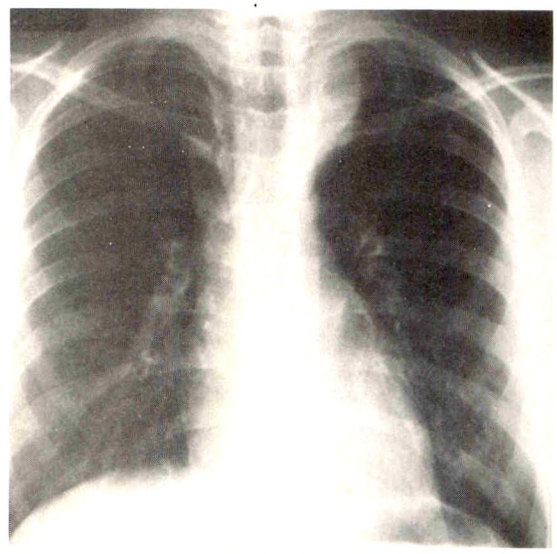


Fig. 2. Superior mediastinal widening in a case of moderately high-positioned left cervical aortic arch.

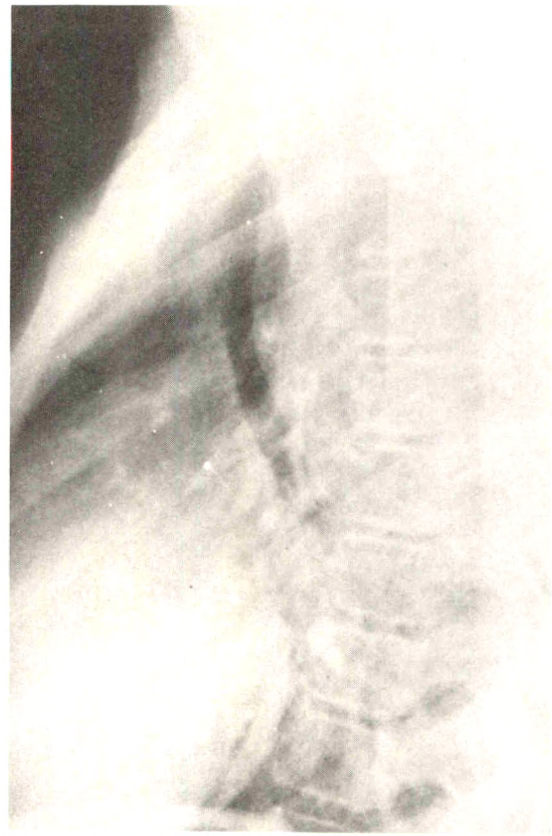


Fig. 3. Lateral film demonstrating *en bloc* displacement of the trachea due to the retrotracheal crossing of the descending aorta from the superior position of the arch on the right, to the left-sided position of the descending aorta.

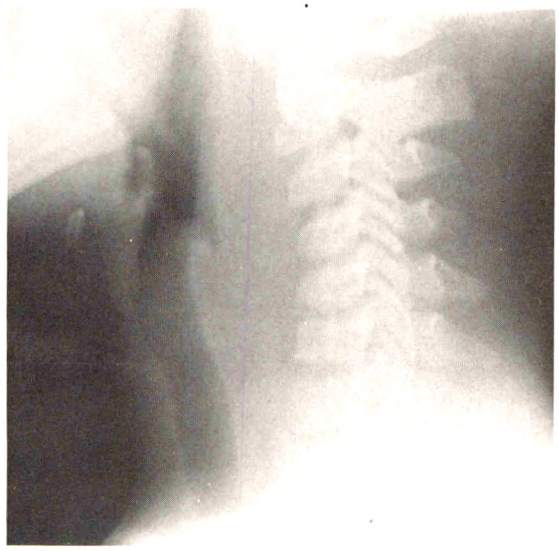


Fig. 4. High position of a right-sided cervical aortic arch, causing an increase in the retrotracheal soft tissues, as well as a slight cervical kyphosis.

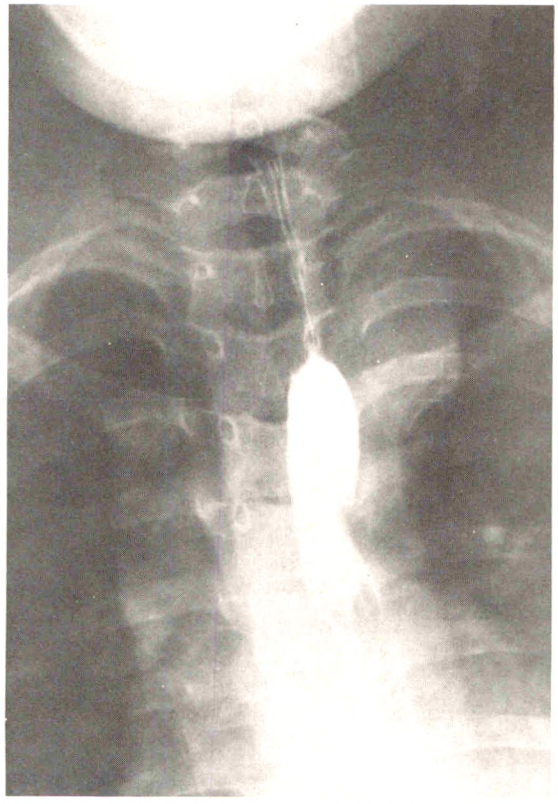


Fig. 5. Leftward displacement of esophagus, adjacent to left cervical aortic arch.

slightly superior to its normal position (Fig. 8, *A* and *B*) to very high into the soft tissues of the neck (Fig. 9, *A* and *B*).

From past reports, and from those of our cases in which the origins were determined, the origin of the great vessels is of three basic variations:

1. Normal (or mirror image, in the case of a right cervical arch).

2. Absent common carotid artery on the same side of the arch, with the internal and external carotids on that side arising directly from the arch.

3. Anomalous, and sometimes stenotic, origin of the subclavian artery contralateral to the side of the arch. In these cases, the subclavian arises as the most distal of the great vessels, from the retroesophageal portion of the descending aorta.

These possible combinations are sum-

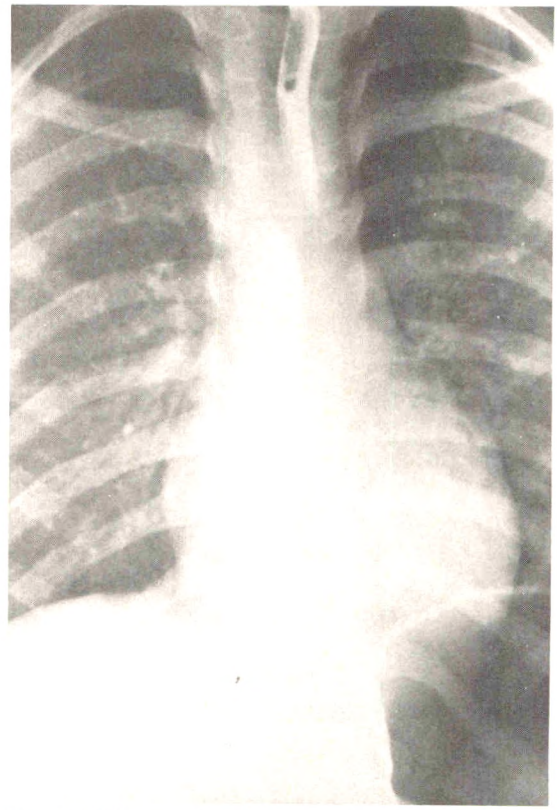


Fig. 6. Oblique defect in barium filled esophagus due to right-sided cervical aortic arch crossing retroesophageally to become the left-sided descending aorta.

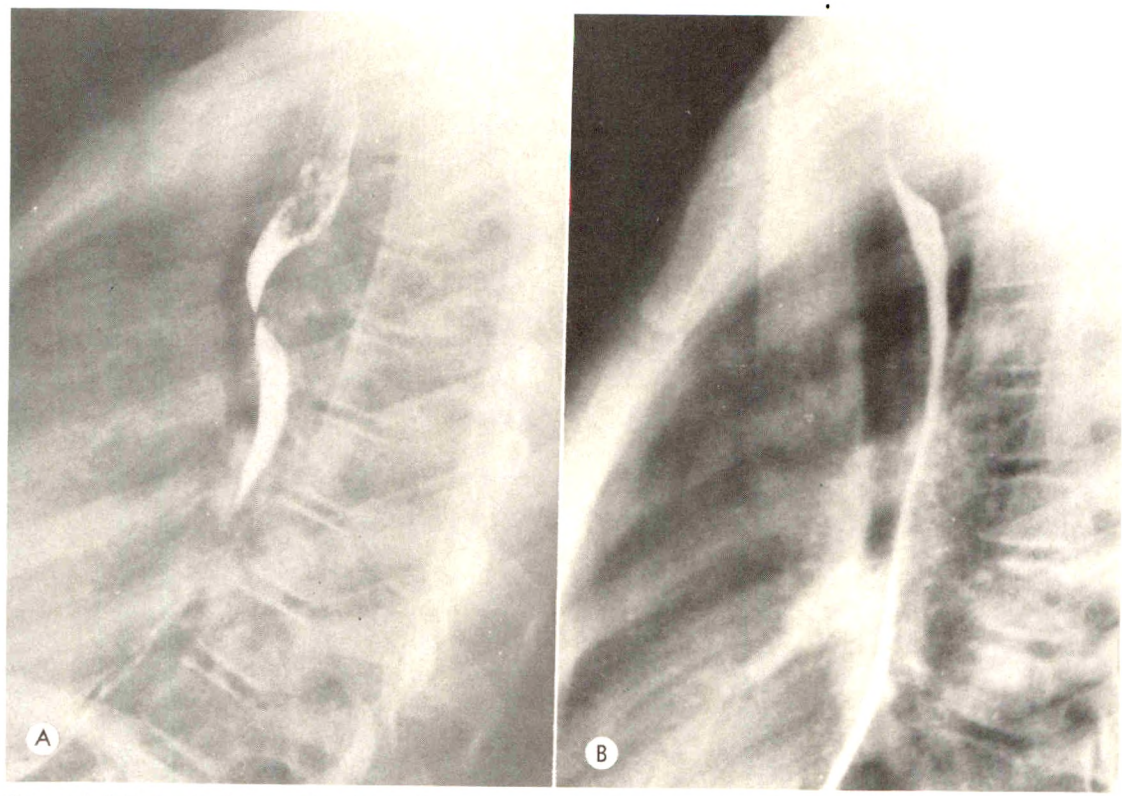


Fig. 7. (*A*) Posterior indentation in barium column in esophagus produced by relatively inferior crossing of descending aorta from right to left at level of T6–7. (*B*) Another case demonstrating the same defect at a more superior level, around T3–4.

marized in Table III.

Not all of the above combinations have been reported, but possibly will be found with future cases, as well as other combinations not listed here. The above arrangements seem to be the most frequent, however, and the development of the separate carotid origins and the anomalous subclavian will be considered later.

It is essential to demonstrate the origins of the great vessels by angiography if surgery is contemplated to relieve symptoms of dyspnea or dysphagia, or for a coexisting cardiac lesion. Our four cases with coexistent cardiac lesions were investigated angiographically as infants, before the cervical position of the aortic arch had become clinically apparent. In two of these (Cases III and IV) the origins of the great vessels from the tortuous, left-sided, cervical arch were normal (Fig. 10; and 11). Cases V and VI, however, did not have angiograms adequate to determine the origins of the great vessels, and only the cervical arch position and cardiac lesion were determined (Fig. 12; and 13, *A* and *B*).

The possibility of subclavian steal syndrome could also be investigated angiographically if there is stenosis at the subclavian origin, with a vertebral artery arising distal to the stenotic region.

Cardiac catheterization has demonstrated small, insignificant blood pressure gradients in two previous reports. One patient had a 5 mm gradient at the apex of the cervical arch, and another a 5 mm gradient at the anomalous subclavian origin.[15] Another had a gradual gradient down the descending aorta.[17] One of our own asymptomatic cases also had a 10 mm gradient at the arch (Case II).

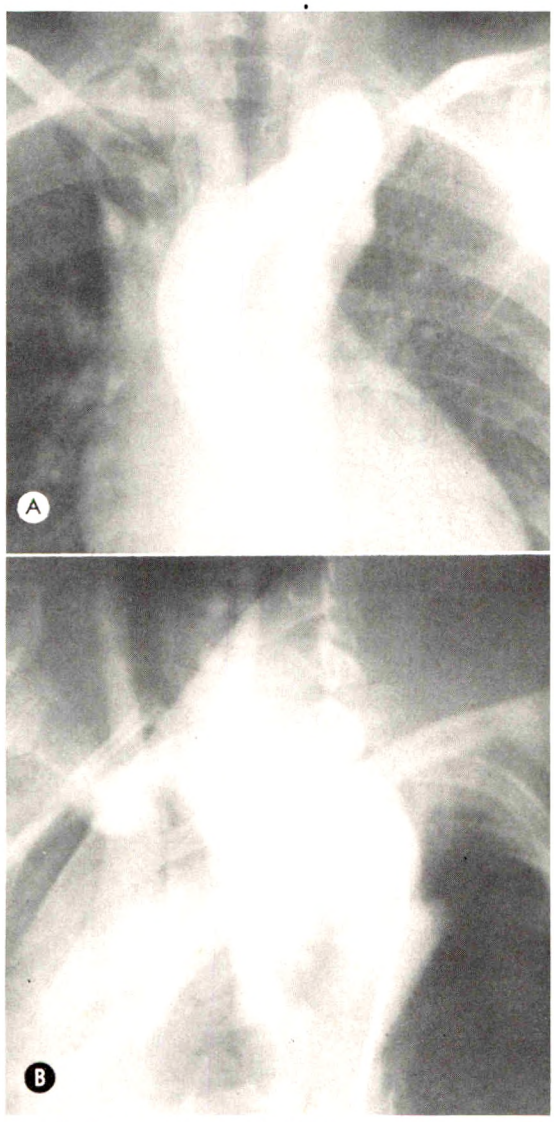


FIG. 8. Case 11. (*A*) Left-sided cervical aortic arch situated at a somewhat higher level than a normal aortic arch. (*B*) Same case, with oblique projection of the opacified arch. There was a normal pattern of the origins of the great vessels. However, it appeared that the left subclavian artery was stenotic at the point just distal to its origin from a diverticulum of the descending aorta (diverticulum of Kommerell).

EMBRYOLOGY

Embryologically, the aortic arch system of human embryos exists as a symmetrical system of blood vessels connected to the primitive cardiac tubes, between the third and seventh weeks of fetal life, during which time the embryo measures from 3 to 16 mm.

Six pairs of branchial arches arise, extending between a single ventral aortic sac; the primitive truncus arteriosus, and paired dorsal aortae, which fuse distally into a single dorsal trunk. The six pairs of arches do not all exist simultaneously, but arise and degenerate in succession from craniad to caudad, in the period between the third and seventh embryonic weeks. The first and second arches lose connection with the ventral aortic sac and form portions of the mandibular and hyoid arteries, respectively. The third arch becomes the proximal portion of the internal carotid artery. The left fourth arch gives rise to the bulk of the normal left thoracic aortic arch. (The remainder of the arch is derived from the proximal portion of the left of the paired dorsal aortae.) The right fourth branchial arch normally becomes the proximal portion of the right subclavian artery. The fifth branchial arches are not known to give rise to any definitive structure in the adult. The proximal portions of both sixth arches become the proximal portions of both pulmonary arteries. The distal left sixth arch becomes the ductus arteriosus. The distal right sixth arch disappears (Fig. 14).

When first formed in the third week, the

TABLE III

ORIGINS OF THE GREAT VESSELS FROM THE CERVICAL ARCH, PROXIMAL TO DISTAL

RBC, LBC—Right, left brachiocephalic artery
RCC, LCC—Right, left common carotid artery
RIC, LIC —Right, left internal carotid artery
REC, LEC—Right, left external carotid artery
RS, LS —Right, left subclavian artery

	Left Cervical Arch	Right Cervical Arch
1.	RBC, LCC, LS (normal)	LBC, RCC, RS (mirror image of normal)
2.	RBC, LEC, LIC, LS	LBC, REC, RIC, RS
3.	RCC, LCC, LS, RS	LCC, RCC, RS, LS
4.	RCC, LEC, LIC, LS, RS	LCC, REC, RIC, RS, LS

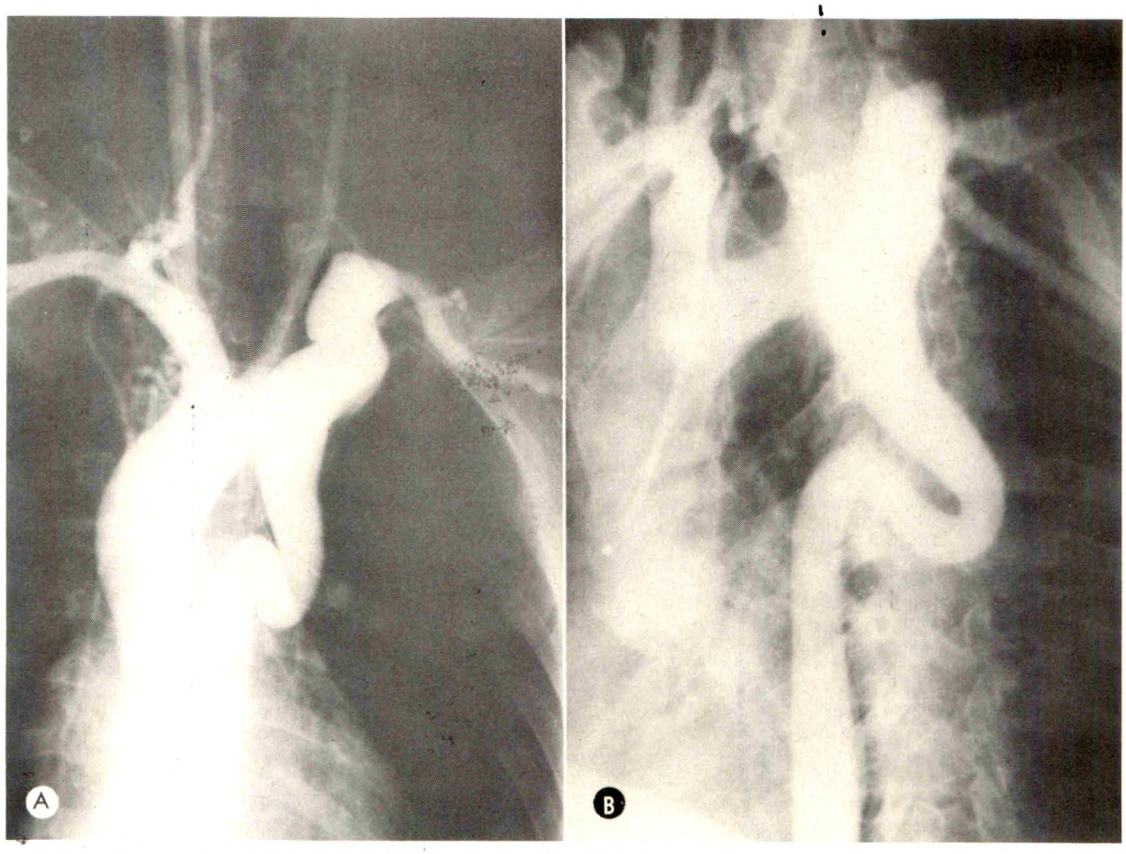


Fig. 9. Case 1. (*A*) Left-sided cervical arch which extends high up into the soft tissues of the neck to the upper border of C7. (*B*) Left anterior oblique view of aortogram.

aortic arch system lies at the occipital level of the embryo, but by the seventh week it has migrated caudad to its final position in the developing thoracic cage.

Because of the original cephalic position of the aortic arch system and the presence of branchial arches at a level higher than the fourth, theories to explain the persistence of a cervical arch have proposed:

1. Failure of descent of the otherwise normal fourth branchial arch.[9]

2. Development of the third[1,9,18,19] or second[12,19] branchial arches into the aortic arch, with consequent persistence of the second or first arch on the side involved as the proximal internal carotid artery (Fig. 15).

3. Fusion of the third and fourth branchial arches on the involved side.[4]

Determination of the true answer may depend on the discovery of a non-vascular structure extending between the third and fourth branchial arches in the embryo with the determination of its position above or below the cervical aortic arch in the postnatal period.

The frequently found abnormal origin of the great vessels has been referred to in Table III as ipsilateral (to the arch) separate origins of the external and internal carotid arteries arising directly from the arch, and the contralateral subclavian artery arising from the descending aorta. Normally, as mentioned, the left thoracic aortic arch is derived from the left fourth branchial arch, along with the proximal portion of the left primitive dorsal aorta. The normal origins of the great vessels, present actually in only about 50–75 percent of the general population, arise

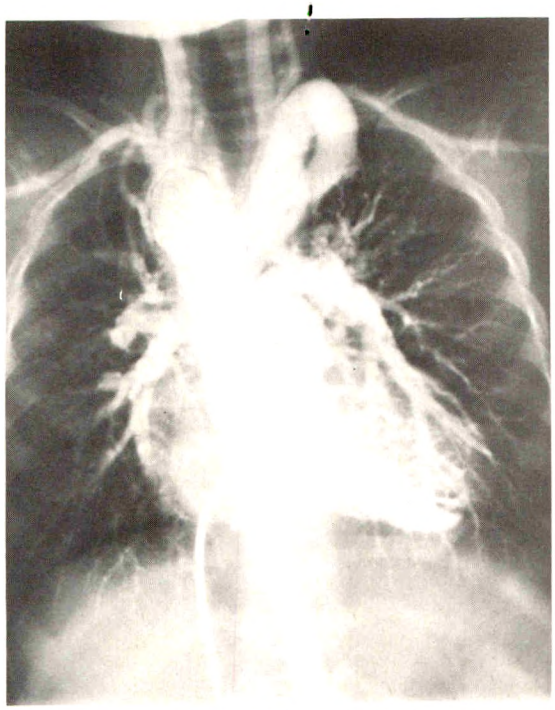
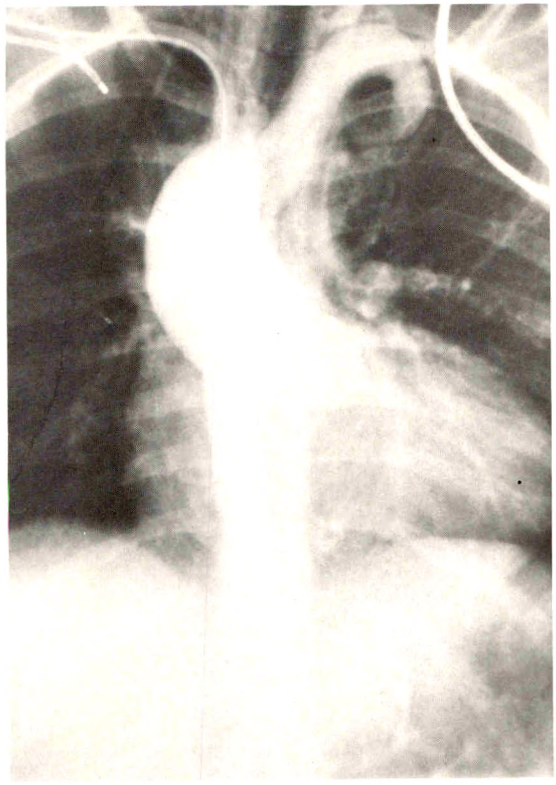


FIG. 10. Case III. Left-sided cervical arch and normal origins of the great vessels, coexisting with a tetralogy of Fallot.



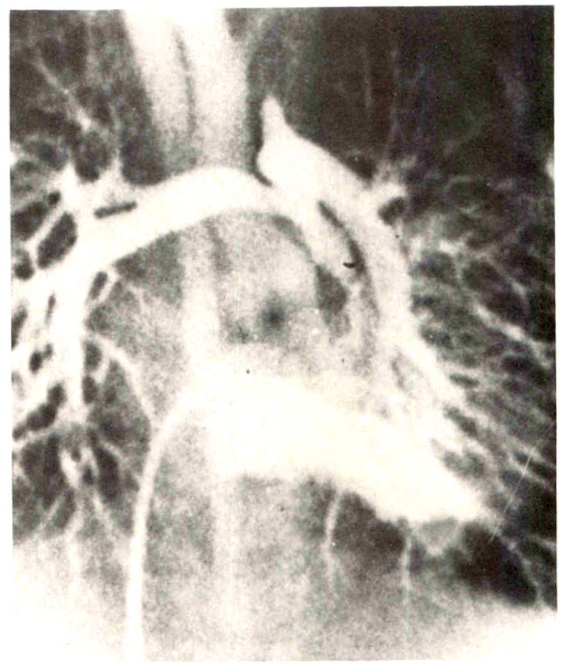


FIG. 12. Case V. Characteristic findings of a double outlet right ventricle with a right-sided cervical aortic arch.

from the disappearance of two other portions of the originally symmetrical aortic arch system. The lack of persistence of the right-sided dorsal aorta distal to the origin of the seventh intersegmental artery on the right permits development of a normal innominate artery, of which the subclavian portion is derived from the right fourth branchial arch, the proximal portion of the primitive right dorsal aorta, and the seventh intersegmental artery of the embryo. Should the right dorsal aorta proximal, rather than distal, to the seventh intersegmental artery drop out, the result would be an anomalous right subclavian artery, arising as the most distal of the great vessels, originating entirely from the seventh intersegmental artery (Fig. 16). A mirror image situation would embryologically explain the anomalous left subclavian arteries, found with frequency in the cervi-

FIG. 11. Case IV. Left-sided cervical arch and normal origins of the great vessels, coexisting with a pseudotruncus deformity. Bronchial circulation is not prominently seen on this view.

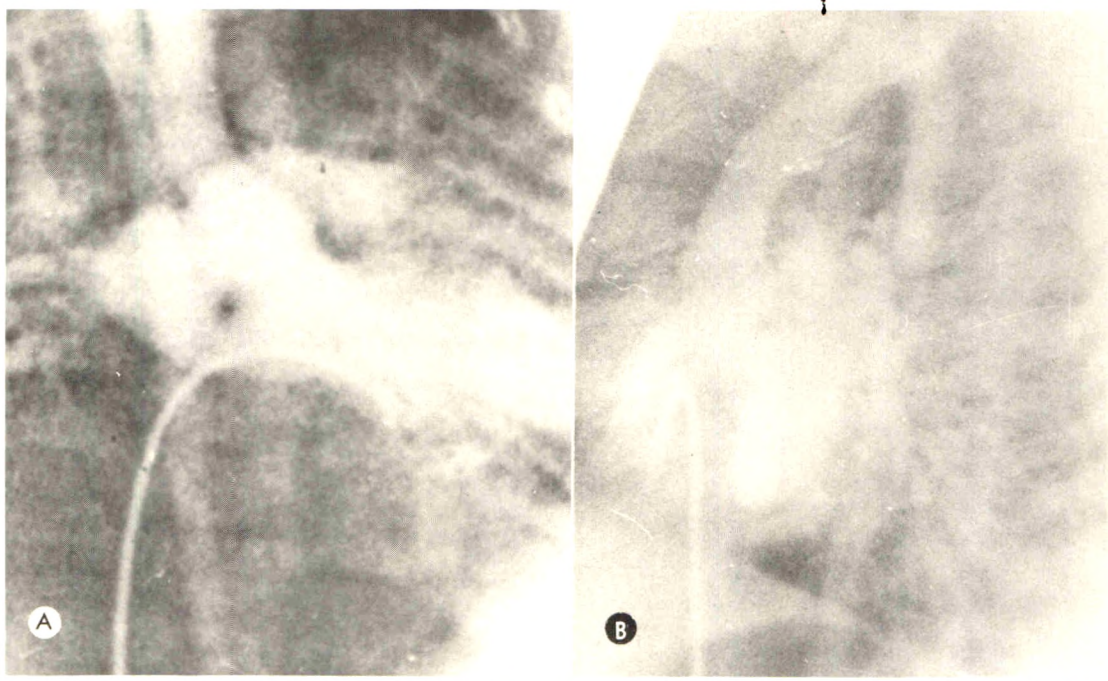


Fig. 13. Case VI. (*A*) Right-sided cervical aortic arch, coexisting with (*B*) a ventricular septal defect.

cal arch cases reported.

The persistence of a normal common carotid artery is dependent on the disappearance of the ductus caroticus—that portion of the paired dorsal aortae in the embryonic system between the dorsal ends of the third and fourth branchial arches. The third arch and the cephalad extension of the dorsal aorta on each side become the internal carotid artery. The external ca-

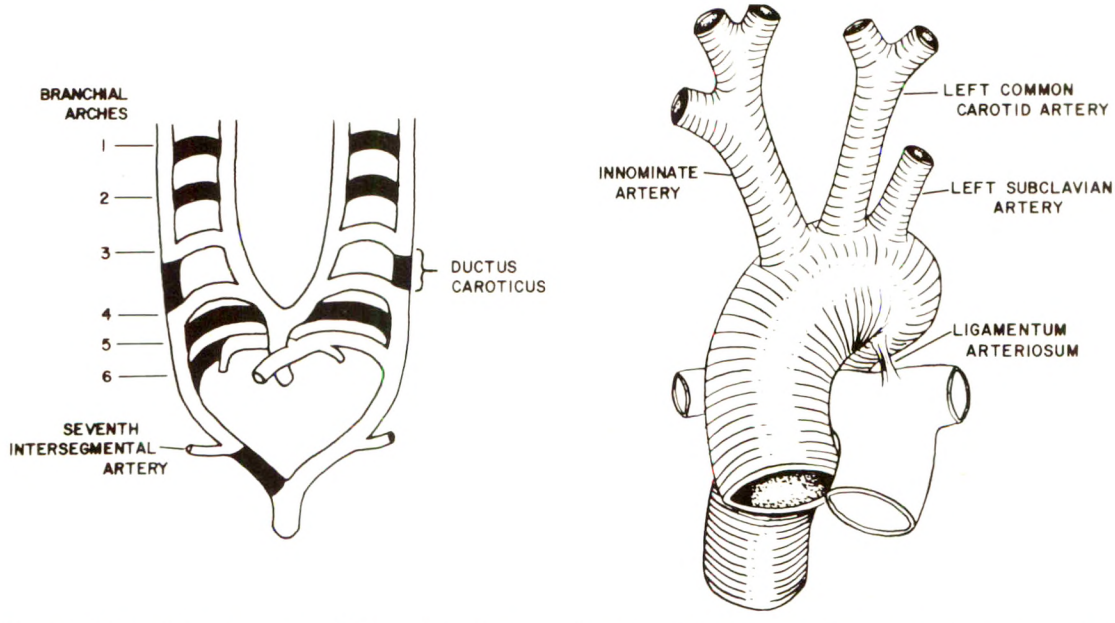


Fig. 14. Normal development of the aortic arch system into the definitive structures of the postnatal period. The six branchial arches do not all exist simultaneously, but arise and degenerate, or become a definitive structure, in order of craniad to caudad (structures in black are those which do not persist in the postnatal period).

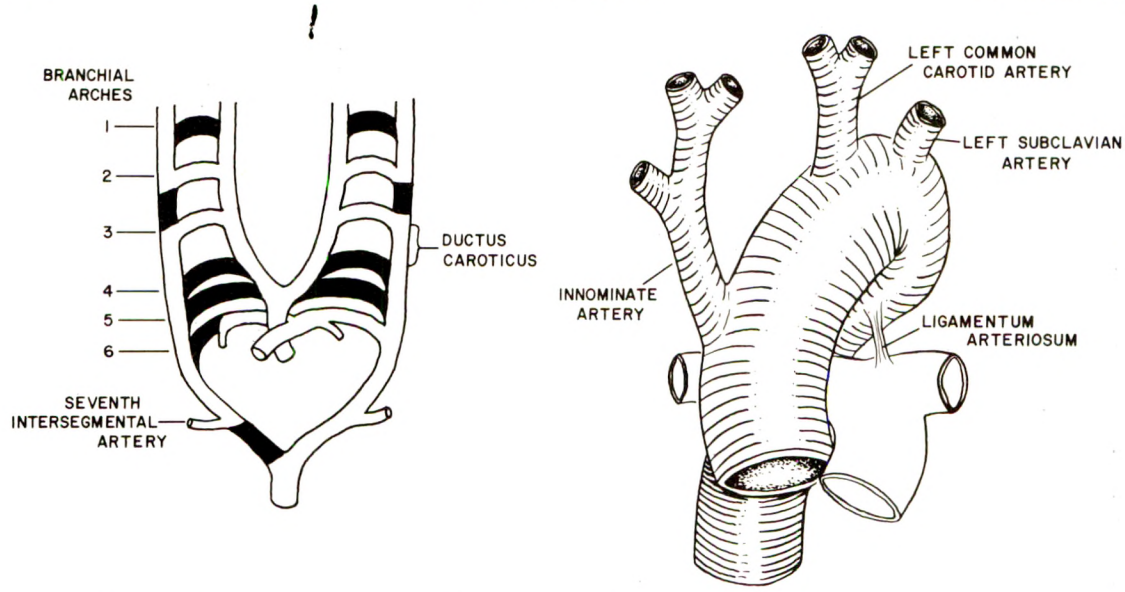


FIG. 15. Hypothetical development of a left cervical aortic arch from the third embryonic branchial arch.

rotid arteries appear to be cephalad outgrowths from the ventral aortic trunk. The common carotid arteries on each side are derived from portions of this ventral sac.

Should the ductus caroticus persist and the third branchial arch disappear, however, the result would be separately originating internal and external carotid arteries on the side involved (Fig. 17). The internal carotid would thus be derived from the ductus caroticus and the cephalad extension of the dorsal aorta. The frequency of this situation in combination with the cervical arch suggests that there is a greater tendency of persistence of the portions of the dorsal aorta above the level of the ductus caroticus, and a greater tendency for lack of persistence of branchial arches cephalad to the third.

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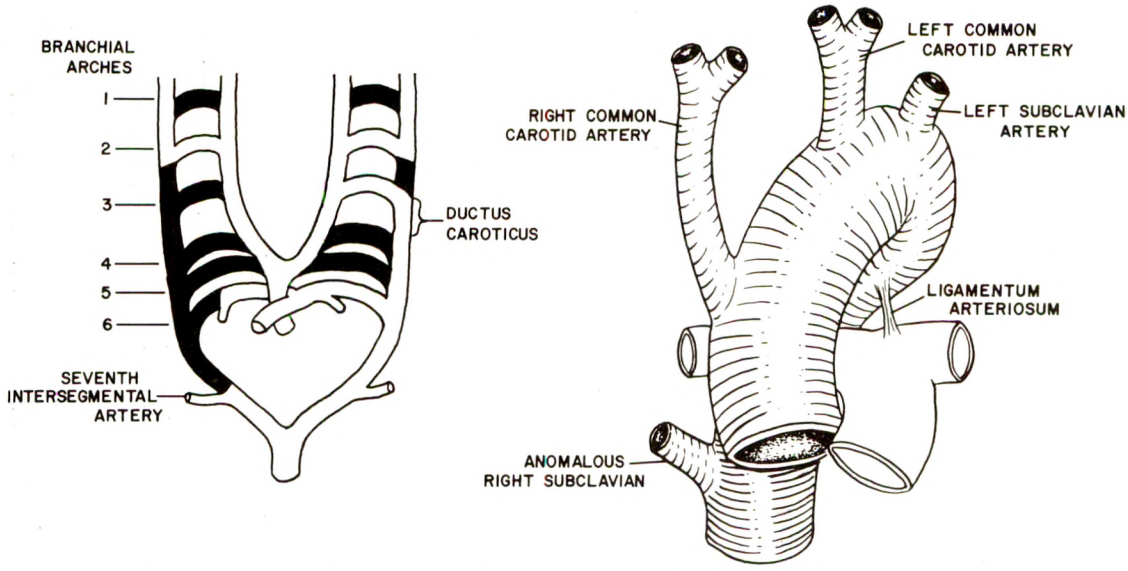

FIG. 16. Hypothetical development of an anomalous right subclavian artery with a left cervical aortic arch.

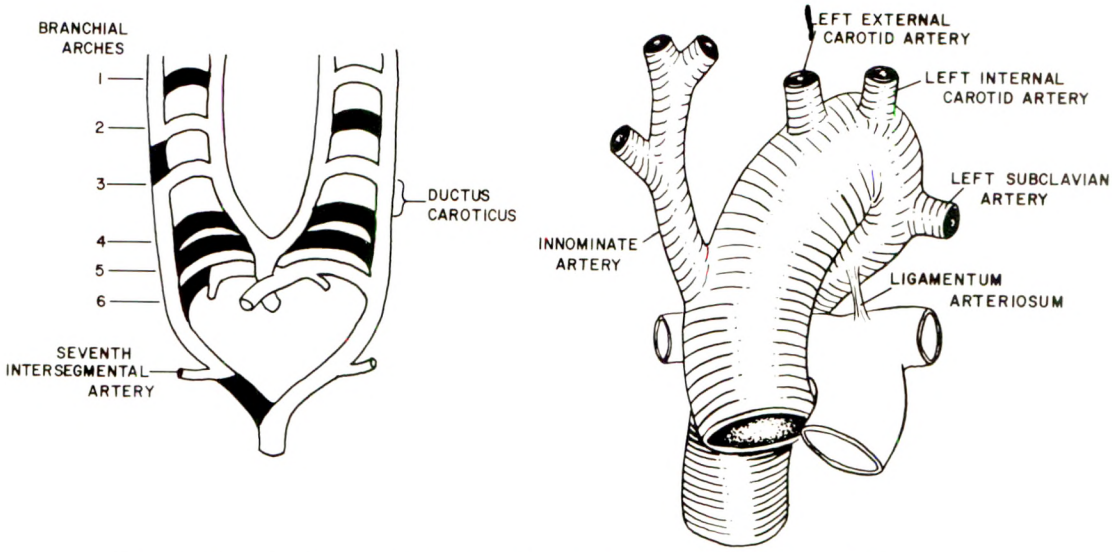

Fig. 17. Hypothetical development of separately originating ipsilateral external and internal carotid arteries with a left cervical aortic arch.

REFERENCES

1. Beavan, T. E., and Fatti, L. Ligature of aortic arch in neck. *Brit. J. Surg.*, 1947, *34*, 414–416.
2. Berman, S. Personal communication.
3. Chang, L. W. M., Kaplan, E. L., Baum, D., and Figley, M. M. Aortic arch in neck: case report. *J. Pediat.*, 1971, *79*, 788–793.
4. D'Cruz, I. A., Cantex, I., Namin, A., Licata, R., and Hastreiter, A. Right sided aorta: part I and II; right aortic arch, right descending aorta and associated anomalies. *Brit. Heart J.*, 1966, *28*, 722–739.
5. Deffrenne, P., and Verney, R. L'Aorte cervicale. *Ann. Radiol.*, 1968, *11*, 525.
6. De Jong, I. H., and Klinghamer, A. C. Left-sided cervical aortic arch. *Am. J. Cardiol.*, 1969, *23*, 285–287.
7. Gravier, J., Vialtel, M., and Pinet, F. À propos d'une tumeur pulsatile du cou: un cas d'aorte cervicale. *Pédiatrie*, 1959, *14*, 437–438.
8. Gray, S. W., and Skandalakis, J. E. Embryology for Surgeons. W. B. Saunders Company, Philadelphia, 1972, p. 827.
9. Harley, H. R. Development and anomalies of aortic arch and its branches with report of case of right cervical aortic arch and intrathoracic vascular ring. *Brit. J. Surg.*, 1959, *46*, 561–573.
10. Lewis, C., and Rogers, L. Cervical aortic knuckle which resembles aneurysm. *Lancet*, 1953, *1*, 825–826.
11. Lipchick, E. O., and Young, L. W. Unusual symptomatic aortic arch anomalies. *Radiology*, 1967, *89*, 85–90.
12. Mahoney, E. B., and Manning, J. A. Congenital abnormalities of aortic arch. *Surgery*, 1964, *55*, 1–14.
13. Massumi, R., Wiener, L., and Charif, P. Syndrome of cervical aorta: report of case and review of previous cases. *Am. J. Cardiol.*, 1963, *11*, 678–685.
14. McCue, C., Mauck, H. P., Tingelstad, J., and Kellett, G. Cervical aortic arch. *A.M.A. Am. J. Dis. Child.*, 1973, *125*, 740–742.
15. Mullins, C., Gillette, P., and McNamara, D. Complex of cervical aortic arch. *A.M.A. Arch. Pediat.*, 1973, *51*, 210–215.
16. Reid, D. G. Three examples of right aortic arch. *J. Anat. Physiol.*, 1913, *48*, 174–181.
17. Richie, R., Delrio, C., Mullins, C., and Hall, R. J. Right-sided cervical aortic arch. *Am. Heart J.*, 1973, *84*, 531–536.
18. Shepherd, R. M., Keith, W. J., and Rosenthal, J. R. Right cervical arch with left descending aorta: case report and review of literature. *A.M.A. Am. J. Dis. Child.*, 1969, *118*, 642–648.
19. Shuford, W. H., Sybers, R. G., Milledge, R. D., and Brinsfield, D. Cervical aortic arch. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 519–527.
20. Yigitabasi, O., and Nalbantgil, I. Murmur over abnormal left apical mass. *Chest*, 1971, *59*, 561–562.

TRANSJUGULAR APPROACH TO THE LIVER, BILIARY SYSTEM, AND PORTAL CIRCULATION*

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ABSTRACT:

More than five years of clinical and animal experience with the use of a transjugular approach to the liver, biliary system, and portal circulation is reviewed. It has been of clinical value in connection with hepatic manometry and venography, liver biopsy, and cholangiography. Satisfactory hepatic manometry and venography were achieved by the transjugular approach in all 47 cases where attempted; diagnostic liver specimens were obtained in 71 of 83 patients (86 percent); and cholangiography was successful in 48 of 52 patients with enlarged intrahepatic ducts (92 percent). No complications occurred with these studies. In animals, transjugular catheterization was used as a means for portal, mesenteric, and pancreatic venography. These procedures are ready for diagnostic clinical use. Therapeutic techniques explored in animals include intravascular tamponade of gastric coronary vein and the nonsurgical creation of intrahepatic portacaval shunts, both of promise in the future management of massive gastrointestinal bleeding from varices.

IN the transjugular (transvenous) approach, a catheter needle system introduced *via* the jugular vein is advanced through the right atrium into a hepatic vein. The catheter or the needle, or both, are then used for various diagnostic or therapeutic procedures relating to the liver, biliary system, and portal circulation (Fig. 1). The transjugular approach was introduced by Hanafee and Weiner[5,19] for cholangiography, and its clinical use was further extended to include liver biopsy,[10] dilatation of a biliary stricture,[6] and hepatic manometry and venography.[13] In animal experiments, it has been used for portal venography,[8] mesenteric venography,[4] pancreatic venography,[11] intravascular tamponade of the gastric coronary vein,[12] and the nonoperative creation of an intrahepatic portacaval shunt.[8,9] This review is based on our over five years of clinical and investigative experience with this approach and its applications.

TECHNIQUE

Procedures are performed in a roentgenologic suite equipped for television monitored fluoroscopy, serial filming, and EKG monitoring. A commercially available transjugular catheter needle set* includes a 45 cm long radiopaque Teflon catheter (3 mm o.d., 2.08 mm i.d.) and a 50 cm long needle (1.83 mm o.d., 1.40 mm i.d.). Both have slightly curved tips.

Clinical procedures are done using local anesthesia. A combination of sedatives (secobarbital, meperidine hydrochloride, or both) and atropine is used for premedication. For cholangiography, prophylactic antibiotics (usually 500 mg ampicillin by mouth four times daily) are also given for a day before and one or two days following the examination. An internal jugular vein, preferably the right, is used for catheter insertion in patients. The skin puncture site

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Supported in part by USPHS Grants HL05828 and HL03275 and the James Picker Foundation on the recommendation of the Committee on Radiology NAS-NRC. Also, the Oregon Heart Association.

is high on the neck, 3–4 cm below the angle of the mandible.

Animal studies are done under general anesthesia and the right external jugular vein, exposed by cut-down, is used for catheter introduction.

The catheter is usually introduced over a guidewire into the right hepatic vein, although any major vein of the right or left lobe can be catheterized. Subsequent steps of the examination depend on the procedure(s) to be done; often several are performed at the same session.

HEPATIC MANOMETRY AND VENOGRAPHY

We consider the transjugular approach for hepatic manometry and venography as complementary to the transfemoral route. We use it in cases where a transfemoral approach is either technically not feasible or contraindicated by femoropelvic thrombosis, inferior vena caval anomaly, or obstruction. We also use it in patients where hepatic studies include simultaneous liver biopsy.

Hepatic manometry and venography are used to evaluate patients possibly having Budd-Chiari syndrome and even more often to assess cirrhotic patients being considered for portal decompressive surgery. Both manometry and venography are done with the catheter in a free and a wedged position. Free hepatic venography helps assess the stage and severity of the cirrhotic process[1,3,14] (Fig. 2*A*); wedged hepatic venography helps clarify hepatoportal hemodynamics and thereby aids in selecting the appropriate type of shunt[16,17] (Fig. 2*B*). The measurement of wedged and free hepatic pressures and those in the inferior vena cava and right atrium permits the diagnosis of portal hypertension as well as an assessment of its type and degree.[16,17]

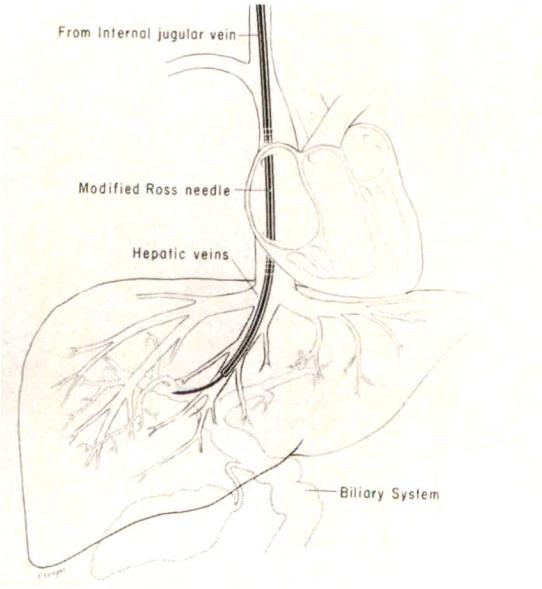

Fig. 1. Schematic drawing of the transjugular approach for cholangiography.

A wedged position of the catheter tip is easily achieved, using the transjugular approach. We occasionally introduce a smaller catheter of 1.8 mm o.d. through the Teflon catheter to extend its reach into small peripheral hepatic veins. Satisfactory hepatic manometry and venography were achieved by the transjugular approach in all 47 cases where attempted.

LIVER BIOPSY

We consider transjugular liver biopsy as complementary to the ordinary transperitoneal technique.[10] It is of particular value in patients with hemocoagulation defects, high grade obstructive jaundice, or in major ascites, where the transperitoneal route is unduly hazardous or contraindicated. Our biopsies are done under fluoroscopic control, employing suction from a syringe attached to the needle. In such cases, the catheter stays in the hepatic vein; only the needle punctures the liver parenchyma. It is usually advanced laterally about 2–4 cm into the parenchyma; several biopsies may be done in various directions from different veins. A crucial point in this technique is limiting suction to the time when the needle is in the liver parenchyma and thus keeping the obtained specimen in the needle. Undesired suction of the specimen into the syringe often results in tissue destruction.

In our series of 83 patients subjected to attempted liver biopsy by the transjugular approach, satisfactory specimens were ob-

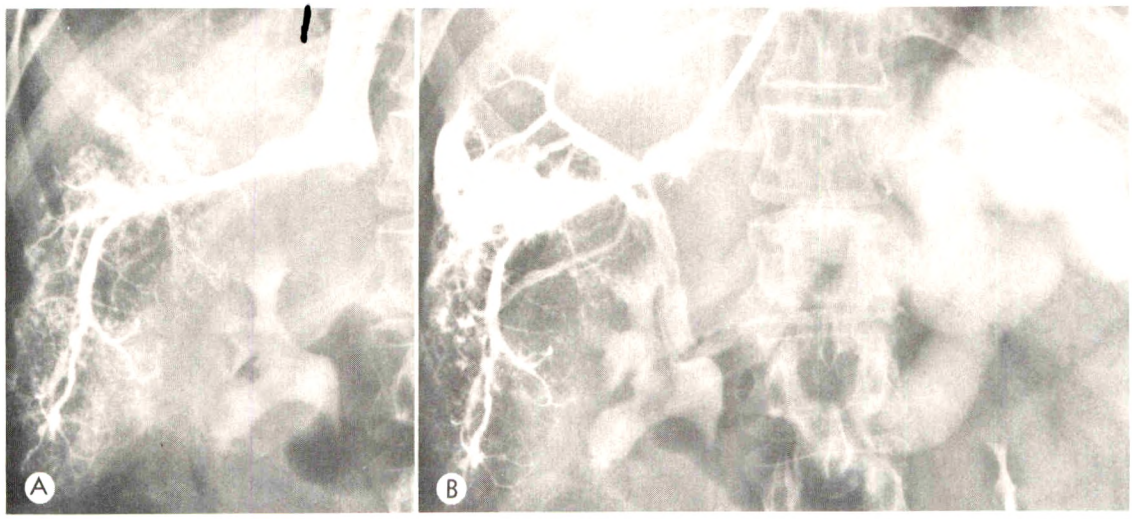

FIG. 2. Hepatic venography performed by the transjugular approach in a 53 year old man with advanced cirrhosis and large gastroesophageal varices. (*A*) Free hepatic venography shows advanced deformity of the hepatic venous system with irregular parenchymal filling. (*B*) Wedged hepatic venography visualizes the portal vein and large gastric varices by retrograde flow.

tained in 71 patients (86 percent). Specimens were usually 3–10 mm long and 1–1.2 mm in diameter (Fig. 3). Biopsy failures in 12 patients were due to failure to enter the internal jugular vein (one patient), to introduce the needle into hepatic veins which joined the inferior vena cava at an unusually sharp angle (one patient), to puncture a hard cirrhotic liver (two patients), and to obtain adequate material (eight patients). We have had no complications associated with liver biopsy.

CHOLANGIOGRAPHY

We consider transjugular cholangiography complementary to endoscopic retrograde cholangiography.[5,10,19] We use it in patients with obstructive jaundice, where the latter approach fails or gives insufficient information. In our hands, transjugular cholangiography has replaced the transperitoneal technique. Safety is one of the important reasons; the transjugular approach can be used even in patients with bleeding diathesis and ascites. Acute suppurative cholangitis is the principal contraindication.

FIG. 3. Liver biopsy performed by the transjugular approach in a 48 year old man with liver metastases from a pancreatic carcinoma.

In the conduct of this procedure, the catheter again stays in the hepatic vein and only the needle traverses the liver to enter the biliary system. Puncture is done under fluoroscopy, usually toward the hilum so as to favor entry into a major intrahepatic bile duct. Knowledge of the size and shape of the liver such as can be gained through prior angiography promotes the safety and success of this technique. Following puncture, the position of the needle is checked by a small test injection of contrast medium. After entry into a biliary duct has been effected, bile is aspirated and sufficient contrast medium injected to fill the entire ductal system. The needle is then withdrawn back into the catheter, which remains in the hepatic vein. Films are exposed during injection of contrast medium and after needle withdrawal.

Satisfactory visualization of the biliary system in our series of 76 patients was re-

lated to the size of intrahepatic ducts. Enlarged ducts were entered and visualized in 48 of 52 patients (92 percent) (Fig. 4). With normal ducts, a satisfactory study was obtained in only two of 24 patients (eight percent). We encountered no complications requiring surgery. At the time of elective surgery in 43 patients, neither blood nor bile was found in the abdominal cavity. In two of these patients, operated on one day after the study, some blood was found in the biliary system. Two patients had minor transient febrile reactions three to five hours after the study.

PORTAL VENOGRAPHY

Portal venography by the transjugular approach has been widely explored in animals and is ready for clinical use. Here, also, the intrahepatic portal circulation is entered by needle puncture from a hepatic vein. In dogs, the usual experimental subjects, the left portal branch provides the best puncture target because of its close relation to the intermediate hepatic vein and its straight course in line with that of the introduced catheter. Following puncture, a guidewire is introduced through the needle into the portal circulation and, thereafter, a Teflon catheter. With the needle and guidewire out, the catheter can be used for venography of any major trunk of the portal circulation. Excellent visualization of the portal vein, its intrahepatic portal branches, and its splenic and mesenteric tributaries is usually possible.[8] For the detailed study of peripheral mesenteric branches, a double-lumen balloon catheter can be introduced. After its inflation in the proximal superior mesenteric vein, the injection of contrast medium through the second lumen beyond the balloon provides detailed visualization of the mesenteric venous bed.[4] Selective filling of individual peripheral portal tributaries is easily ob-

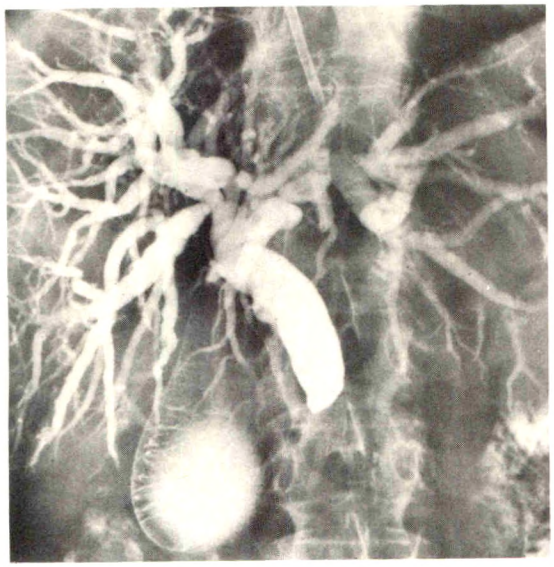

Fig. 4. Transjugular cholangiogram in a 63 year old woman with pancreatic carcinoma causing subtotal obstruction of the distal common bile duct.

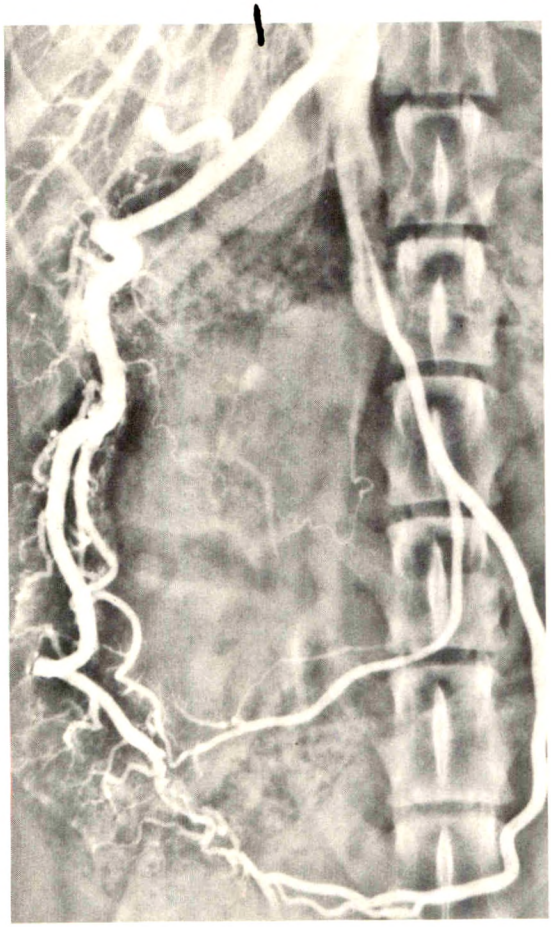

Fig. 5. Selective retrograde pancreaticoduodenal venogram performed in a dog by the transjugular approach with good visualization of duodenal and pancreatic veins.

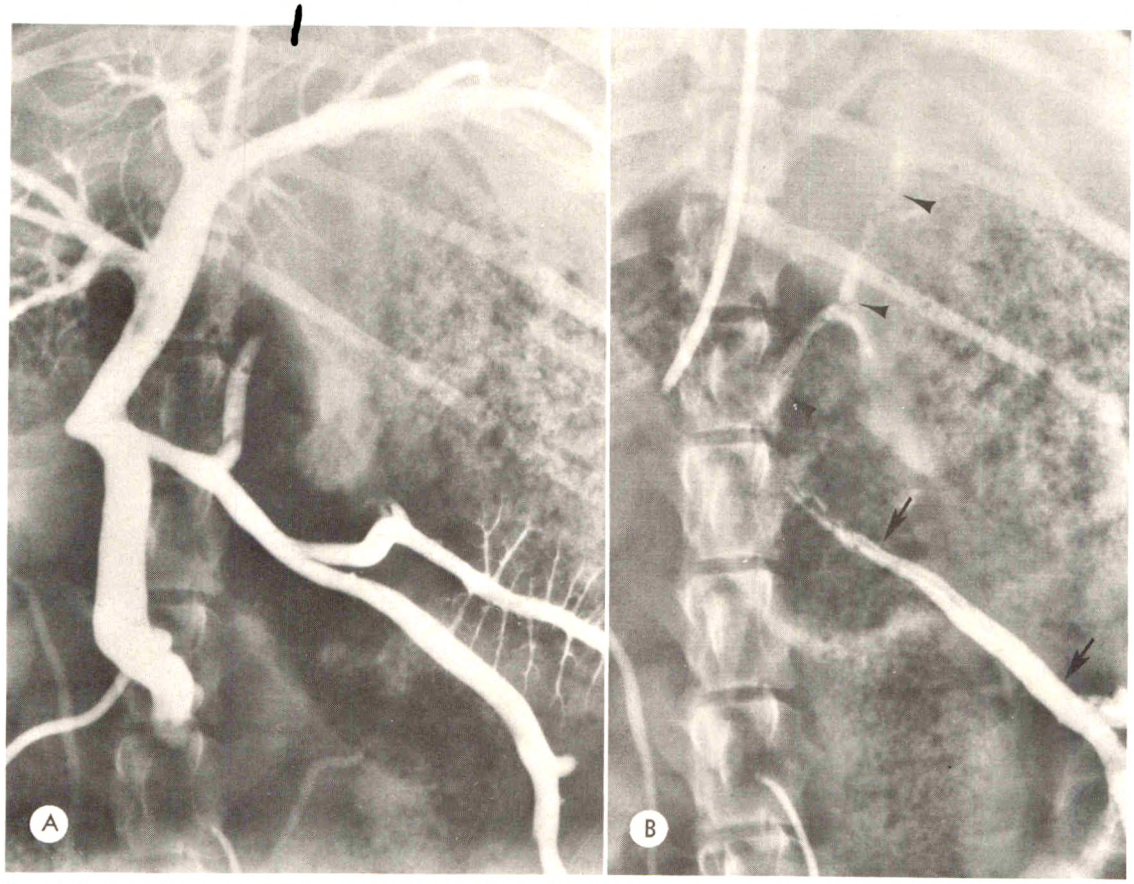

FIG. 6. Isobutyl 2-cyanoacrylate "cast" occlusion of the left gastric and splenic veins in a dog by the transjugular approach. (*A*) Control retrograde portal venogram. (*B*) Plain abdominal roentgenogram after venous occlusion reveals opaque isobutyl 2-cyanoacrylate "cast plugs" in the left gastric (arrowheads) and splenic veins (arrows).

tained with a small catheter coaxially introduced through the Teflon catheter, a technique permitting selective retrograde pancreatic venography[11] (Fig. 5).

INTRAVASCULAR TAMPONADE OF THE GASTRIC CORONARY VEIN

Transportal tamponade of the gastric coronary vein using a transjugular approach has been developed in dogs as a method of promise for the emergency, nonoperative management of bleeding from gastroesophageal varices.[12] Portal entry that was used for venography and a coaxially introduced catheter were used to catheterize the gastric coronary vein. Temporary intravascular tamponade of the gastric coronary vein was achieved, using various types of balloon catheters. The injection of a rapidly polymerizing tissue adhesive, isobutyl 2-cyanoacrylate, enabled its prompt, selective, and definitive obliteration. Polymer casts were used to obliterate long segments of large veins such as the splenic (Fig. 6).

INTRAHEPATIC PORTACAVAL SHUNT

A method for the nonoperative creation of intrahepatic portacaval shunts has been explored in normal dogs.[8,9] Following transjugular entry into the portal circulation, the transhepatic puncture tract in the liver was dilated to a diameter of 6 mm by a system of coaxial dilating catheters, similar to that used for angioplasty of arteriosclerotic obstructions.[4] A short length of tubing was then slipped over the catheters and left in place in the liver, to form a

sleeve graft connecting the portal and inferior caval veins. Such shunts have stayed open for six to twelve days (Fig. 7). After percutaneous retrieval of the shunt which was in place for less than five days, the fistulous tract closed rapidly. In animals in which the shunt was in place for two weeks, a portacaval fistula persisted for 24 to 48 hours.

DISCUSSION

The transjugular approach to liver parenchyma, bile ducts, and the portal circulation involves somewhat elaborate techniques; however, it has considerable diagnostic and therapeutic potential. Skilled professional and technical personnel experienced in angiographic techniques are essential, as are specially equipped roentgenologic facilities. Among its advantages are safety and the opportunity of achieving several diagnostic and therapeutic objectives through a single procedure. Since transperitoneal passage and puncture of the liver capsule are eliminated, cholangiography and liver biopsy can be done even in patients with hemocoagulation defects, ascites, or high grade obstructive jaundice without risk of hemoperitoneum or bile peritonitis. Combined studies such as cholangiography and liver biopsy or manometric and venographic examinations with liver biopsy give complex information about hepatobiliary diseases and their secondary hemodynamic changes, thereby favoring an informed decision as to the need for and type of surgery.

Diagnostic and therapeutic procedures explored in animal experiments are now ready for use in selected patients. Portal venography can help in the preoperative work-up of patients undergoing decompression surgery or in the evaluation of operability in hepatic or pancreatic tumors, where simpler procedures fail to give sufficient information. Pancreatic venography can help in the early diagnosis of pancreatic carcinoma. Portal catheterization also offers an important clinical and research tool for collecting venous blood samples from individual portal tributaries for various laboratory studies.

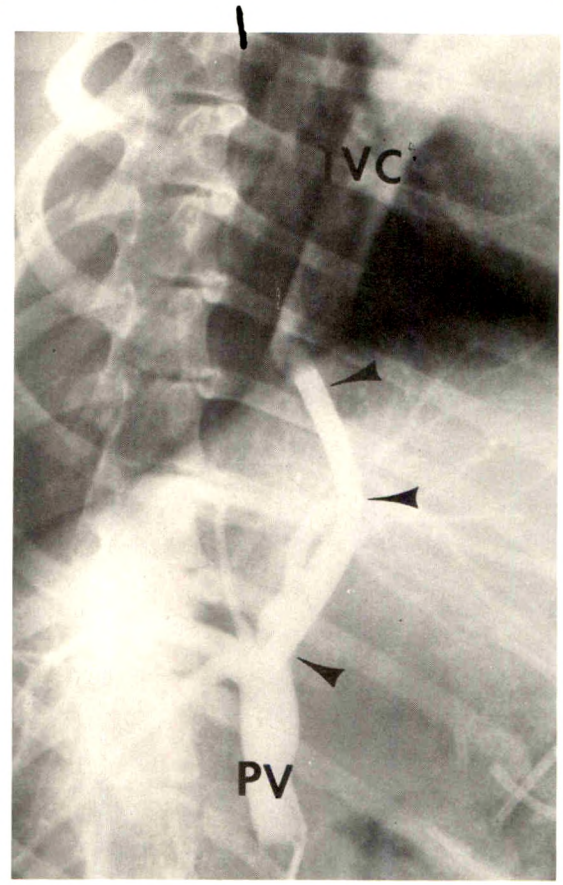

Fig. 7. Portal venogram in a dog with an intrahepatic portacaval shunt created by the transjugular approach. A short tubing (arrowheads) drains part of flow from the portal vein (PV) into the inferior vena cava (IVC).

The application of intravascular tamponade to gastroesophageal varices and the creation of intrahepatic portacaval shunts may be of therapeutic value in otherwise unmanageable massive gastrointestinal bleeding from varices. The transperitoneal transhepatic route has already been used to control such bleeding by obliteration of varices.[7,18] We consider the transjugular, transvenous route preferable to the transperitoneal because of greater safety and the opportunity to effect portal decompression by an intrahepatic portacaval shunt. However, before the sleeve shunt technique explored in animals becomes clinically applicable, more research

will be needed, especially in achieving continuing shunt patency. Research should also be directed to other possible means for transhepatic shunting such as the creation of a direct portovenous fistula by simple dilatation of the transhepatic puncture tract (which might remain patent in the cirrhotic liver with portal hypertension), or the formation of a neovascular lumen around a temporary shunt, techniques which have been successfully applied in the clinical management of arteriosclerotic obstructions.[2,15]

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REFERENCES

1. Britton, R. C., Brown, C. H., and Shirey, E. K. Intrahepatic veno-occlusive disease in cirrhosis with chronic ascites: diagnosis by hepatic phlebography and results of surgical treatment. *Ann. Surg.*, 1963, *158*, 370–382.
2. Dotter, C. T., Judkins, M. P., and Rösch, J. Nonoperative treatment of arterial occlusive disease. *Radiol. Clin. North America*, 1967, *5*, 531–542.
3. Galmarini, D., Fassati, L. R., Riquier, G., Trivellini, G., and Zanoli, P. G. Value of angiographic methods in diagnostic assessment of liver damage in portal hypertension, *South African M. J.*, 1969, *43*, 743–746.
4. Gyepes, M. T., and Rösch, J. Experimental mesenteric venography. *Invest. Radiol.*, 1970, *5*, 257–260.
5. Hanafee, W. N., and Weiner, M. Transjugular percutaneous cholangiography. *Radiology*, 1967, *88*, 35–39.
6. Hanafee, W. N., Rösch, J., and Weiner, M. Transjugular dilatation of biliary duct system. *Radiology*, 1970, *94*, 429–432.
7. Lunderquist, A., and Vang, J. Transhepatic catheterization and obliteration of the coronary vein in patients with portal hypertension and esophageal varices. *New England J. Med.*, 1974, *291*, 646–649.
8. Rösch, J., Hanafee, W. N., and Snow, H. Transjugular portal venography and radiologic portacaval shunt: experimental study. *Radiology*, 1969, *92*, 1112–1114.
9. Rösch, J., Hanafee, W. N., Snow, H., Barenfus, M., and Gray, R. Transjugular intrahepatic portacaval shunt: experimental work. *Am. J. Surg.*, 1971, *121*, 588–592.
10. Rösch, J., Lakin, P. C., Antonovic, R., and Dotter, C. T. Transjugular approach to liver biopsy and transhepatic cholangiography. *New England J. Med.*, 1973, *289*, 227–231.
11. Rösch, J., and Dotter, C. T. Retrograde pancreatic venography: experimental study. *Radiology*, 1975, *114*, 275–279.
12. Rösch, J., Goldman, M. L., and Dotter, C. T. Experimental catheter obstruction of gastric coronary vein: possible technique for percutaneous intravascular tamponade of gastroesophageal varices. *Invest. Radiol.*, 1975, *10*, 206–211.
13. Rösch, J. Hepatic Venography. Encyclopedia of Radiology. Springer Verlag, Berlin. In press.
14. Smith, G. W., Westgaard, T., and Björn-Hansen, R. Hepatic venous angiography in evaluation of cirrhosis of liver. *Ann. Surg.*, 1971, *173*, 469–480.
15. Sparks, C. H. Autogenous grafts made to order. *Ann. Thoracic Surg.*, 1969, *8*, 104–113.
16. Viamonte, M., Jr., Warren, W. D., Fomon, J. J., and Martinez, L. O. Angiographic investigation in portal hypertension. *Surg. Gynec. & Obst.*, 1970, *130*, 37–53.
17. Viamonte, M., Jr., Warren, W. D., and Fomon, J. J. Liver panangiography in assessment of portal hypertension in liver cirrhosis. *Radiol., Clin. North America*, 1970, *8*, 147–167.
18. Viamonte, M., LePage, J., Lunderquist, A., Pereiras, R., Russel, E., Viamonte, M., and Camacho, M. Selective catheterization of portal vein and its tributaries. *Radiology*, 1975, *114*, 457–560.
19. Weiner, M., and Hanafee, W. N. Review of transjugular cholangiography. *Radiol. Clin. North America*, 1970, *8*, 53–68.

Vol. 125, No. 3

POLYVINYL ALCOHOL (IVALON)—A NEW EMBOLIC MATERIAL*

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MINNEAPOLIS, MINNESOTA

ABSTRACT:

Successful nonsurgical treatment of gastrointestinal bleeding and arteriovenous malformations by embolization techniques has been previously documented. 1) Compressed Ivalon sponge was found to be a suitable embolic material in animals and in four patients. 2) The material has been extensively used in surgery, and its biocompatibility has been proved. 3) Expansion of the compressed sponge to its original size after embolization makes this material extremely effective. Recanalization did not occur in animals and humans. 4) For the occlusion of larger arteries, Ivalon can be wrapped around the guidewire. Ivalon sponge absorbs blood and serum, unwraps itself allowing withdrawal of the guidewire. 5) Embolization procedures are not without risk since reflux of embolic material may occur. Therefore, these procedures have to be carried out under fluoroscopic control.

IT is the purpose of this communication to report the experience with a synthetic embolic material which can be introduced through catheters in the management of gastrointestinal bleeders, arteriovenous malformations, hemangiomas, and traumatic rupture of blood vessels. Ivalon sponge, previously used extensively in surgery, has proved to be a good synthetic embolic material.

BACKGROUND

Ivalon, a polyvinyl alcohol, is converted into a sponge-like material by foaming agents and hardened with formaldehyde. It has been used as an ion exchange resin since this foam is a negatively charged colloid and strongly adsorbs cations. Ivalon is also well known to housewives who use it as a cleaning sponge. Polyvinyl alcohol foam has the ability to absorb water; and it is, therefore, used in industry for electroplating and photography. The material is inert and withstands the action of dilute acids, strong alkalis, and common detergents. Ivalon sponge has the unique property of being resilient and readily compressible when wet. If the compressed sponge is dried and becomes hard, it retains its compressed shape. If placed into an aqueous medium as blood, the compressed sponge resumes its original uncompressed shape as seen in Figure 1. The compressed 4 mm long Ivalon material expanded to 23 mm in length after being soaked in saline. This excellent plastic memory makes this material particularly attractive for embolization procedures where the size of the embolus is always limited by catheter size.

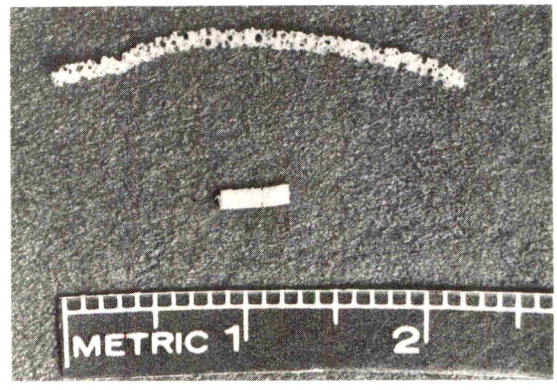

FIG. 1. Compressed Ivalon at the bottom of the figure expanded six times as seen on the top after being soaked in saline.

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This work was supported in part by USPHS Grant 1 T12 HE 5853 and 2 Pol HE 06314.

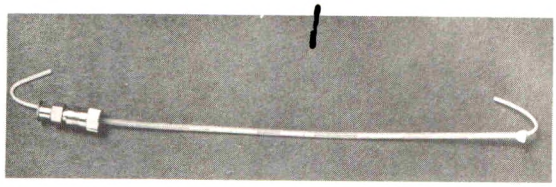

FIG. 2. Ivalon plugs in pre-loaded Clay Adams polyethylene 240 tubes ready for gas sterilization.

PREVIOUS USES OF IVALON

Ivalon sponge has been extensively used in experimental animals as a filling material following pneumonectomy. Autopsy examination revealed that Ivalon sponge is inert and readily invaded by fibrous tissue with a minimal amount of inflammatory cells.[5] In 1949 it was first introduced for medical use by J. H. Grindlay[5] at the Mayo Clinic. When it was used as a skin graft in experimental animals,[7] microscopic examination revealed minimal cellular infiltration. In the 1960's Ivalon was used as a skin substitute in patients with burns. Because of the pores in the foam, it was permeated with serum and adhered to the skin in one or two days. This material appears to serve as a scaffold for ingrowing connective tissue and becomes an integral part of the body. The sponge was also advocated for surgical treatment for rectal prolapse.[3] Very recently Porstmann *et al.*[9] used this material for nonsurgical closure of patent ductus arteriosus with excellent results. Polyvinyl alcohol sponge was the synthetic material of choice in the early days of cardiac surgery. The first surgical closure of septal defects in this institution was accomplished by this material; it was used for the reconstruction of the mitral valve in cases of mitral insufficiency, as reported by others.[1] Although it was used for the repair of aneurysms, increasing rigidity with time made it necessary to replace it by more pliable and long lasting plastics such as dacron and teflon. However, the biocompatibility of Ivalon sponge has been well established.[6]

The Preparation of Ivalon Emboli. Ivalon plastic foam is highly compressible in the dry or wet stage. If the material is soaked in heparin and compressed in a vice, its dimensions can be reduced by a factor of 10–15:1. If dried, the compressed material will retain its shape in spite of gas sterilization. If introduced into the blood stream, it will swell and resume its original shape.

Ivalon sponge was soaked in heparin and compressed in a vice and dried. One half to 1 mm diameter plugs were cut from the dried material with a hole punch similar to a cork borer or the instrument used to cut side holes on angiographic catheters. These plugs measured a few mm in length and were pre-loaded in small plastic tubing (Fig. 2). Depending upon the size of the Ivalon plugs, either Clay Adams 240 or 260 could be used as pre-loaded tubes and gas sterilized. Care must be taken that only gas sterilization and no steam is used. Otherwise, the compressed plugs will expand due to absorption of water and will not be suitable for injection through catheters. At the time of embolization, the pre-loaded tubes are connected to the catheter, and the plugs are flushed into the catheter and blood vessel by forceful manual saline injection. It is very important to eliminate all constrictions between the loading tubes and the selective catheter

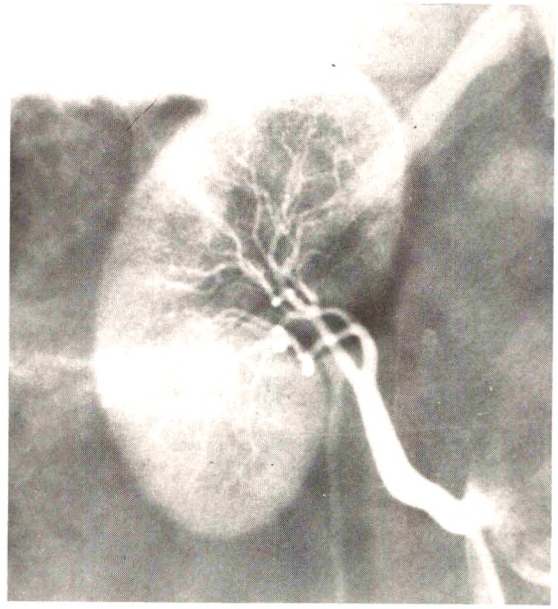

FIG. 3. Selective renal angiogram of a dog prior to Ivalon embolization.

since the compressed Ivalon plugs swell rather rapidly, making prompt delivery of these emboli difficult.

ANIMAL EXPERIMENTS

The renal arteries of dogs were embolized with Ivalon. Angiography was performed 72 hours post Ivalon embolization and revealed obstruction of the renal artery with no evidence of recanalization (Fig. 3; and 4). Aortography performed one week post-embolization revealed occlusion of the renal artery at its most proximal portion due to antegrade thrombosis. The dogs were sacrificed at the end of seven days, and the renal artery and kidneys were subjected to histologic examination. The gross specimen of kidney revealed thrombosis of the main renal artery (Fig. 5). Histologic examination of the canine renal cortex revealed thrombosis of several renal arteries and noncanalization. The renal cortex showed several areas of hemorrhagic necrosis due to thrombosis of the intrarenal arteries. The problem of lysis and fragmentation which is commonly encountered with autogenous clots, therefore, does not exist. This drawback has been well documented by several authors,[2,10] both in humans and canine experiments. It is very important to selectively catheterize and inject the emboli as close to the lesion as possible. Otherwise, the possibility exists that embolic material may enter other arteries resulting in embolic infarctions. Furthermore, it is extremely important that the emboli are injected slowly, particularly after the flow through the artery has decreased. If injection is performed rapidly, embolic material may reflux and embolize other arteries. This has been shown in our laboratory where the renal artery was successfully embolized by Ivalon plugs. At the time of post mortem, however, additional emboli were found in the contralateral renal artery. The gross specimen of the target kidney shows extensive hemorrhagic infarction due to embolization. The contralateral kidney revealed several areas of infarction (Fig. 6). Since the catheter was well in place at the time of embolization, it is likely that the embolization of the contralateral

Fig. 5. The gross specimen of the canine renal artery reveals thrombosis as pointed out in this figure by arrow.

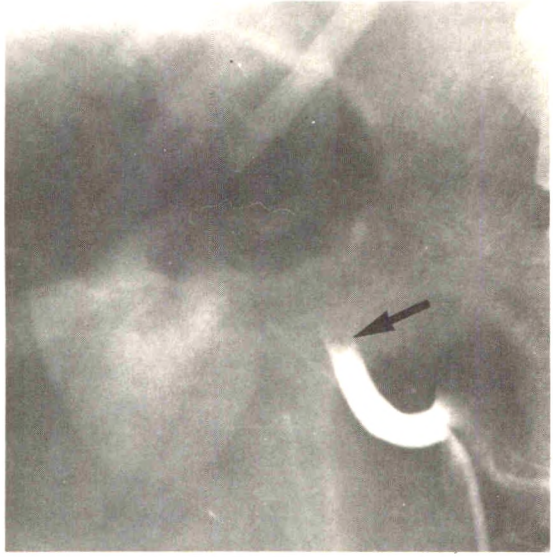

Fig. 4. Selective renal angiography 72 hours post-embolization reveals complete thrombosis of the renal artery (arrow).

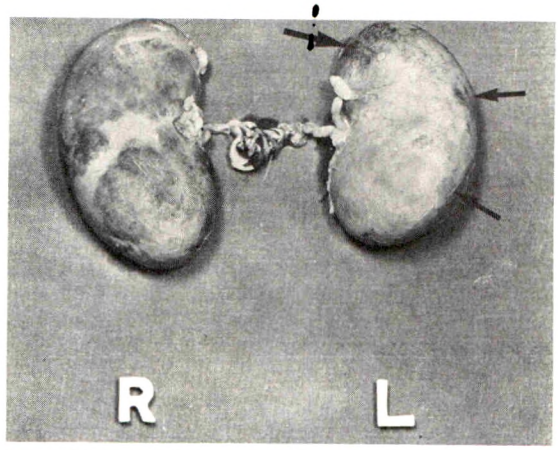

FIG. 6. The right kidney was totally infarcted due to selective embolization. In the contralateral kidney (arrows), certain areas were infarcted due to forceful injection of embolic material.

renal artery was due to reflux by a too forceful injection.

One of the major advantages of this material is expansion to its original size upon exposure to blood. If the sponge is precompressed 10:1, the plug will resume its original proportion after it has been embolized. At the time of embolization, therefore, there is a gradual decrease of flow due to complete obstruction of smaller arteries due to the expansion of the injected Ivalon emboli plugs. This gradual expansion of the embolic material results in a very effective and rapid occlusion of the arterial tree, as evidenced in animal experiments where comparatively few injected emboli resulted in complete and rapid thrombosis of the superior mesenteric artery. Ten dogs were subjected to embolization of various arteries.

Recently, barium impregnated Ivalon plugs have become commercially available, rendering the embolic material visible by roentgenography. At the present time, our experience is limited to non-opaque Ivalon. Another way to make Ivalon plugs radiopaque would be the incorporation of a stainless steel bead.

REPORT OF CASES

CASE 1. N. O. is a four and one-half year old male with a liver tumor which has grown rapidly in size since it was noted at the age of sixteen months. In January, 1973, he had an open biopsy of the liver which was diagnosed as hemangioendothelioma. He was treated by radiation therapy. However, his abdomen continued to increase in size; and a second exploration was carried out in February, 1974, which revealed diffuse involvement of the liver.

At that time he was transferred to the University of Minnesota Hospitals for consideration of a liver transplant as soon as a donor became available. Abdominal angiography revealed a huge liver and hepatic artery supplying multiple vascular foci (Fig. 7). Numerous vascular spaces stayed opacified for a long time without evidence for arteriovenous shunting.

This patient was a candidate for liver transplantation. Decrease in the size of the liver prior to surgery was thought to be essential. Ligation of the hepatic artery was proposed; but due to the large size of the liver, the hepatic artery was not readily accessible by surgery. Therefore, it was decided to attempt trans-

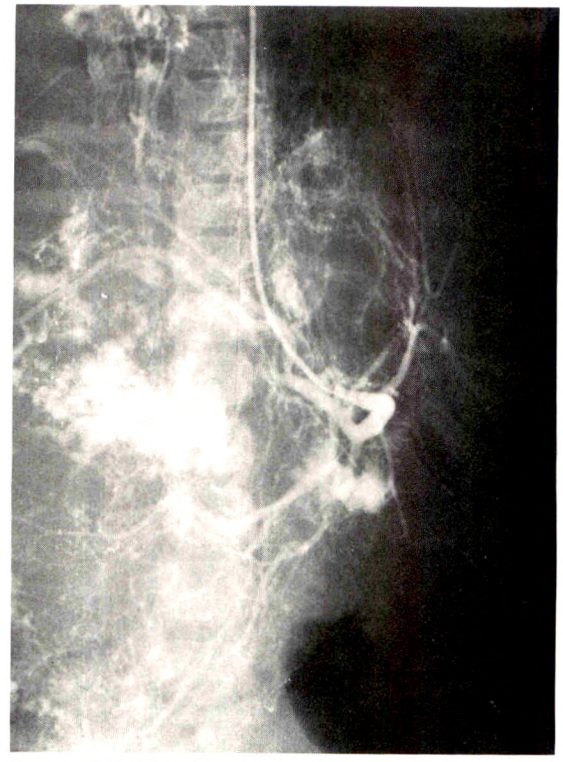

FIG. 7. Selective hepatic angiogram reveals a large hepatic artery and multiple feeding vessels to the huge liver mass. Numerous vascular spaces were identified without arteriovenous shunting.

catheter arterial embolization. Since the hepatic artery was huge, injection of small emboli could not be used in this case. The injection of small emboli would have resulted in obstruction of a few small peripheral hepatic branches, but the main hepatic artery would have been unaffected.

It was, therefore, decided to construct a strainless steel umbrella which could be advanced with the guidewire through the catheter, serving as a baffle for the injected embolic material (Fig. 8).

The No. 7 French teflon catheter was inserted *via* cutdown of the left brachial artery. The hepatic artery was selectively catheterized, and three stainless steel umbrellas were placed through the catheter. Since the small emboli could pass through the steel umbrellas, two small nylon bristle brushes were delivered *via* catheter using a guidewire (Fig. 8).

Ivalon emboli were then delivered and seemed to pile up at the site of the nylon brushes and the metal umbrella. At the completion of the procedure there was practically no blood flow through the embolized hepatic artery as evidenced by angiography. Obviously the foreign material acted as an excellent nidus for subsequent thrombus formation.

Subsequent to the embolization procedure, the patient showed deterioration of liver function with rise of SGOT to 720 units, which ultimately returned to 50 units, his bilirubin rose as high as 5.5 mgm but declined to 2.3 mgm. Shrinkage of the liver by approximately 5 cm above the pubis was noted, but he developed more ascites and his abdominal girth did not change.

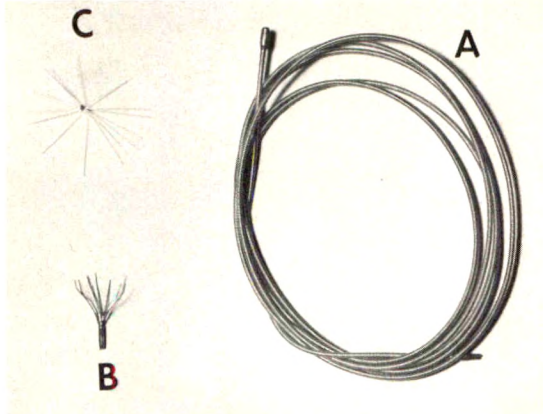

Fig. 8. Guidewire (A) with specially constructed blunt knob that was used to advance the stainless umbrella (B) and nylon brush (C).

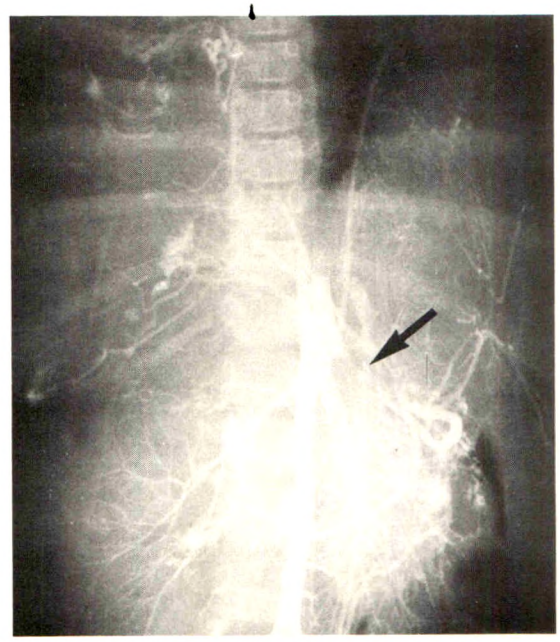

Fig. 9. Thrombosis of hepatic artery at the site of the stainless umbrella (arrow). The collateral flow to the liver was maintained *via* unnamed branches from the aorta and inferior phrenic arteries.

A follow-up angiogram in April, 1974, revealed complete occlusion of the hepatic artery at its origin with demonstration of collateral flow to the liver *via* branches arising directly from the aorta and inferior phrenic arteries (Fig. 9). Venous pooling in the previously demonstrated vascular spaces was markedly diminished.

On the morning of his last admission to the hospital, he was noted to be extremely lethargic and had a respiratory arrest. He was found to have a blood sugar of less than 10 mgm percent and received massive infusions of glucose. A nodule found in the right upper lung field was thought to be metastatic. It was felt that he became hypoglycemic because of his poor oral intake and the fact that his liver was unable to store glycogen.

At autopsy a large nodular reddish-brown tumor replaced the entire liver. A portion of tumor was soft and necrotic possibly secondary to hepatic arterial embolization. The final diagnosis was hemangiosarcoma of the liver with metastases to the lungs. Pathological examination of the common hepatic artery shows the stainless umbrellas, nylon brushes, and the spongy material, namely polyvinyl alcohol (Ivalon) (Fig. 10). There was no distal em-

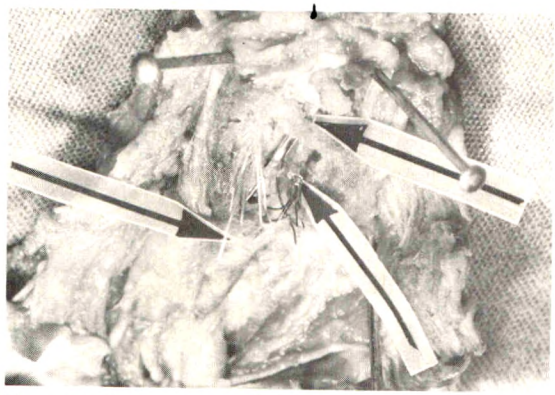

FIG. 10. Postmortem examination of the hepatic artery reveals thrombosis. The arrows point towards the stainless umbrellas, nylon brushes, and Ivalon spongy material covered by fibrin.

bolization due to the baffle created by the stainless steel umbrellas and nylon brushes. There was antegrade thrombosis of the main hepatic artery.

CASE II. Patient J. S. was diagnosed as having a capillary hemangioma of the right ear at the age of two weeks. The lesion grew rapidly and formed a massive hemangioendothelioma on the neck and forehead. Two episodes of massive external hemorrhage were treated with blood transfusions. A second course of prednisone was given, and an angiogram revealed a very vascular hemangioma with little arteriovenous shunting (Fig. 11).

There was congestive heart failure with the liver palpable 5 cm below the right costal margin. The huge heart and pulmonary edema

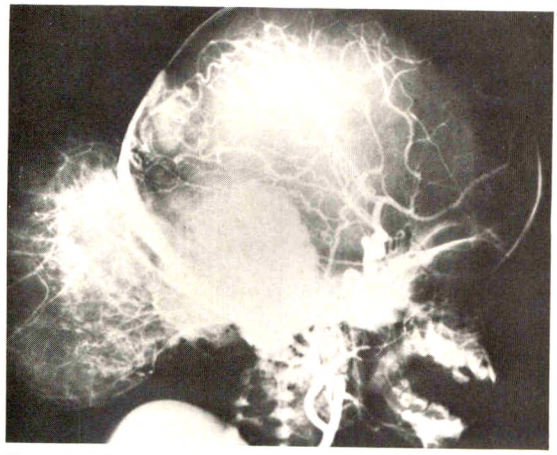

FIG. 11. Common carotid angiography reveals extensive feeders from the external carotid branches to the huge vascular mass.

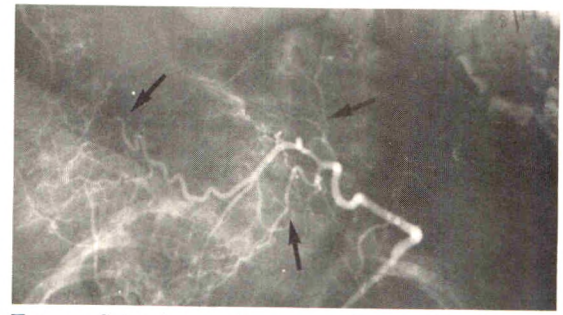

FIG. 12. Selective angiogram of the left thyrocervical artery reveals many feeders (arrors) to the mass.

were felt to be due to high output failure. It was, therefore, decided to treat this patient by extensive Ivalon embolization in order to decrease the vascular supply to the huge mass.

On August 19, 1974, abdominal angiography was performed and excluded additional angiomas in the abdomen. Feeders from the left thyrocervical trunk were selectively catherized and successfully embolized with Ivalon plugs (Fig. 12; and 13). Some of the feeders from the right subclavian artery were also catheterized and embolized as evidenced by sudden amputation of the branches (Fig. 14).

On August 23, 1974, a right brachial cut down was performed, and a No. 5 French red Kifa catheter was introduced selectively into the right thyrocervical trunk which was successfully embolized with Ivalon plugs (Fig. 15; and 16). Some of the other feeders from the right subclavian artery were also selectively entered and embolized. The patient's congestive heart failure did not improve.

Because of the potential danger of embolizing the internal cerebral circulation, embolization of the external circulation was carried out by

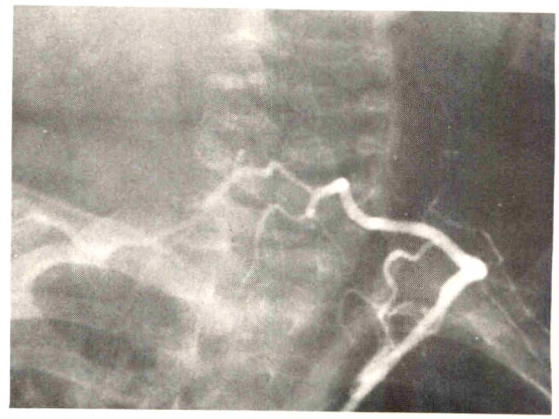

FIG. 13. Note disappearance of major feeders following Ivalon embolization.

Fig. 14. Note obliteration of several feeders which are not named (arrows) from the right subclavian following embolization.

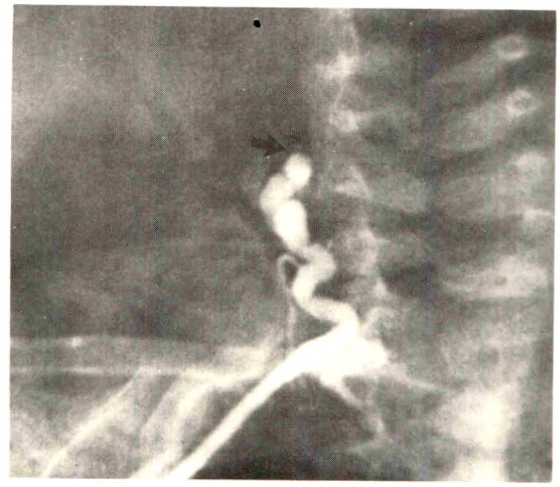

Fig. 16. Note sudden amputation of (arrow) right thyrocervical artery and disappearance of the feeder vessels following embolization.

surgical exposure of the left external carotid artery. A catheter was selectively introduced into the external carotid artery, and an angiogram was performed. Large feeding vessels from the left occipital artery were identified and embolized. At the end of the procedure, no flow was seen to the mass *via* the occipital artery, and the left external carotid artery was surgically ligated. The patient showed improvement as evidenced by increase in food intake and improved alertness. Objectively, there was no definite decrease in the size of the mass.

A follow-up arch aortogram revealed practically no new feeders either from the right subclavian artery or left external carotid arteries. Small feeders from the left subclavian artery were thought to be insignificant and not suitable for selective catheterization embolization (Fig. 17). The patient was discharged with low doses of digitalis for congestive heart failure.

In spite of successful embolization, the patient remained in congestive heart failure which suggested the possibility of a complicating congenital cardiac anomaly in spite of lacking electrocardiographic and clinical findings. Cardiac catherization revealed a cor triatriatum which was corrected surgically.

A third patient with an arteriovenous malformation of the spinal artery underwent successful Ivalon embolization. This

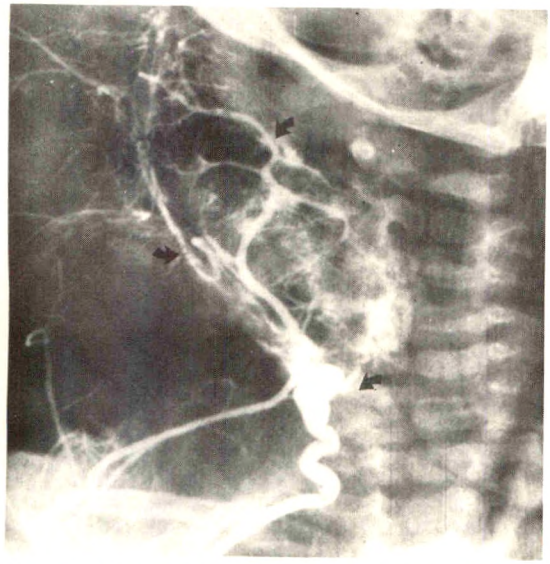

Fig. 15. Note many feeders (arrows) from the right thyrocervical artery to the huge vascular mass.

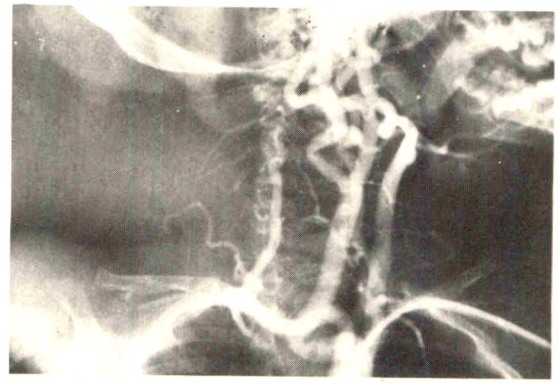

Fig. 17. An arch aortogram after complete embolization shows obliteration of all major feeders (compare with Fig. 11).

case will be the subject of a separate report.

DISCUSSION

Angiomatous malformations with arteriovenous shunting are notoriously difficult to correct by surgery. Ligation of the feeding artery results in rapid recurrence due to development of new feeders. In general, only surgical excision of the malformation results in cure which is commonly not possible due to the location of the angiomatous malformation.

In recent years numerous embolic materials as autogenous clots,[10] silastic emboli, barium impregnated silastic balls, methacrylate,[8] stainless steel pellets,[4] muscle, etc., have been suggested as embolic material. Ideally the injected emboli should reach the site of the arteriovenous malformation and should not enter into the venous component. With the injection of methacrylate, a liquid polymer, the liquid material may enter the venous side and may become embolized into the pulmonary artery.

Barium impregnated silastic balls are attractive. This embolic material is very difficult to handle since it tends to cling to the injection syringe and connecting tubing. Furthermore, since the catheters are very small, such barium balls are minute and difficult to see with the naked eye or by roentgenography. They may cause only incomplete occlusion of the vessel, whereas in the case of Ivalon, the emboli irreversibly result in ultimate complete occlusion.

Autogenous clot tends to break up upon injection through small catheters and embolize arteries which are not feeding the malformation. Furthermore, the possibility of cot lysis and recanalization is one of the major disadvantages of this technique.

Ivalon sponge seems to be an ideal agent for securing permanent hemostasis and promoting thrombosis.[11] The compressed sponge is readily injected and swells upon contact with blood. Incomplete occlusion of small arteries is, therefore, prevented. Once expanded, it is invaded by fibrocytes and becomes part of the patient tissue. Therefore, the material has been extensively used in general and cardiac surgery.[1,3,5–7,9]

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We wish to give special thanks to Miss Mary Scherman for her help in the preparation of this manuscript.

REFERENCES

1. BARNARD, C. N., and SCHIRE, V. Ivalon baffle for posterior leaflet replacement in treatment of mitral insufficiency: follow-up study. *Surgery*, 1968, *63*, 727–730.
2. BOOKSTEIN, J. J., CHLOSTA, E. M., FOLEY, D., and WALTER, J. F. Transcathether hemostasis of gastrointestinal bleeding using modified autogenous clot. *Radiology*, 1974, *113*, 277–285.
3. BOUTSIS, C., and ELLIS, H. Ivalon-sponge-wrap operation for rectal prolapse: experience with 26 patients. *Dis. Colon & Rectum*, 1974, *17*, 21–37.
4. DOPPMAN, J. L., DICHIRO, G., and OMMAYA, A. Obliteration of spinalcord arteriovenous malformation by percutaneous embolization. *Lancet*, 1968, *131*, 477.
5. GRINDLAY, J. H., and CLAGETT, O. T. Plastic sponge prosthesis for use after pneumonectomy: preliminary report of experimental study. *Proc. Staff Meet. Mayo Clin.*, 1949, *24*, 1538–1539.
6. HAWE, A., and RASTELLI, G. C. Late deterioration of intracardiac Ivalan sponge patches. *J. Thoracic & Cardiovasc. Surg.*, 1969, *58*, 87–91.
7. HOGEMAN, K. E., GUSTAFSON, G., and BJORLIN, G. Ivalon surgical sponge used as temporary cover of experimental skin defects in rats: preliminary report. *Acta chir. scandinav.*, 1961, *121*, 83–89.
8. LUESSENHOP, A. J., and SPENCE, W. T. Clinical notes: artificial embolization of cerebral arteries. *J.A.M.A.*, 1960, *172*, 1153–1155.
9. PORSTMANN, W., WIERNY, L., WARNKE, H., GERSTBERGER, G., and ROMANIUK, P. A. Catheter closure of patent ductus arteriosus. *Radiol. Clin. North America*, 1971, *9*, 203–218.
10. ROSCH, J., DOTTER, C. T., and BROWN, M. J. Selective arterial embolization: new method for control of acute gastrointestinal bleeding. *Radiology*, 1972, *102*, 303–306.
11. TADAVARTHY, S. M., KNIGHT, L., OVITT, T. W., SNYDER, C., and AMPLATZ, K. Therapeutic transcatheter arterial embolization. *Radiology*, 1974, *111*, 13–16.

Vol. 125, No. 3

THE ROLE OF LYMPHANGIOGRAPHY IN CHYLOTHORAX*

A REPORT OF SIX NONTRAUMATIC CASES

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ABSTRACT:

Six cases of nontraumatic chylothorax secondary to filariasis, postinflammatory thoracic duct obstruction, idiopathic lymphangiectasis with lymph node hypoplasia, lymphangioma and lymphoma (two cases) are reported. All cases were studied by lymphangiography, which provided considerable diagnostic information. In those cases which are undiagnosed at the time the chylothorax is discovered, a lymphangiogram should be carried out prior to any therapeutic measures.

CHYLOTHORAX is an unusual problem but frequently a difficult one both in establishing the underlying etiology and in management. A review of six nontraumatic cases of chylothorax, which stimulated this report, has led to the conviction that a lymphangiogram is the key diagnostic test and should be utilized in all undiagnosed cases prior to any form of therapy.

In a historical review by Jahsman,[5] quoting Brescia, Bartolet is credited with the initial description of chylothorax in 1633, and Quincke reported the first case in 1875. Kinmouth and Taylor[6] were the first to show that the lymph system could be visualized roentgenographically and the first to demonstrate the thoracic duct by lymphangiography. Wallace *et al.*[18] gave impetus to the technique currently utilized in 1961 with their report of a large series of cases. In 1962 Laumonier *et al.*[8] were the first to study specifically the thoracic duct by lymphangiography.

Van Pernis[17] in 1949 studied over 1,000 thoracic ducts and found the duct to be a variable structure, which may consist of several channels, but under normal conditions is always single from the cisterna chyli to the eighth thoracic vertebra. His measurements determined that the duct normally is two to four mm in diameter. Within the duct, valves are irregularly placed except for a terminal valve within one cm of the subclavian vein. Nusbaum *et al.*[13] found that the thoracic duct varied from two to eight cm, and the variations of normal included two to as many as four ducts emptying into both subclavian veins. In some of their cases the thoracic duct emptied into the jugular vein rather than the subclavian. They also noted multiple valves with a constant one near the termination of the duct at its entrance to the venous system. These authors measured flow rates of chyle in the thoracic duct at 60 to 100 ml per hour and thoracic duct pressure at the height of its maximum flow at ten to 28 cm of water. In cirrhotic patients as many as eight liters of chyle may pass through the duct daily.

All milky effusions are not due to chyle in the thoracic cavity. Cholesterol pleural effusions associated with tuberculosis, rheumatoid arthritis, and other inflammatory conditions may have a similar gross appearance but do not contain triglycerides and should not be confused with chyle.

Table 1 is an etiological classification of chylothorax, which enlarges upon a previous one by Gruwez *et al.*[4] These authors list as a separate category congenital malformation of the thoracic duct. This entity probably falls either into the idiopathic lymphangiectasis or lymphangioma category and has not been listed separately in this Table.

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TABLE I

CLASSIFICATION OF CHYLOTHORAX

I. Traumatic interruption of the thoracic duct
  A. Operative trauma
  B. Penetrating trauma
  C. Blunt trauma
II. Obstruction to normal flow of lymph
  A. Conditions which cause fibrosis
    1. Filariasis
    2. Tuberculosis
    3. Other inflammatory causes
  B. Thrombosis of the left subclavian vein
  C. Idiopathic lymphangiectasis with lymph node hypoplasia
  D. Primary lymphangioma
  E. Other neoplasms (with or without lymphangiectasis)
    1. Lymphoma
    2. Carcinoma
III. Minute trauma superimposed upon II
IV. Neonatal chylothorax
V. Idiopathic chylothorax

REPORT OF CASES

CASE 1. Fibrosis Secondary to Filariasis (Fig. 1). This 54 year old white male was admitted to the Veteran's Administration Hospital of the University of Arizona with swelling of the left side of his neck. Seven years prior to admission, while in India, filariasis was discovered. On one previous occasion since then he had had some swelling on the left side of his neck, which subsided quickly, and he had not sought medical attention. On the current admission the enlargement on the left side of the neck also rapidly subsided. A chest roentgenogram revealed a small bilateral pleural effusion, which on thoracentesis proved to be chylous. A left supraclavicular lymph node biopsy was not rewarding. A lymphangiogram demonstrated unusually small and sparse lymph nodes as well as an interruption in the midportion of the thoracic duct with multiple collateral channels. There was retrograde filling of several channels in the left side of the neck and there was a small accumulation of contrast material in the left supraclavicular space. Because of the left supraclavicular lymph node biopsy, however, it was difficult to draw conclusions from these findings. The chylous effusions rapidly decreased and the patient was discharged without therapy.

Comment. The relationship between the swelling of the left side of the neck and the chylous effusion in this patient is not entirely clear. Both could result from temporary obstruction at the junction of the thoracic duct and the left subclavian vein. Gruwez *et al.*[4] list three inflammatory or parasitic diseases which may be responsible for chylothorax. These are filariasis, tuberculosis, and thrombosis of the subclavian vein. The small and scanty lymph nodes demonstrated in this patient are typical of those seen with fibrosis secondary to filariasis, and the adequacy of the lymphatic system was undoubtedly compromised by the fibrotic lymph nodes. The roentgenograms also demonstrate interruption of the midportion of the thoracic duct with collateral channels, reflux into the lymph channels of the neck, and a lymphocele in the left supraclavicular space at the site of the recent biopsy. Although the exact pathway of the chyle in this case could not be determined, the transient swelling of the left side of the neck must be related to the chylothorax. Minute trauma superimposed on both a compromised lymphatic system and a previously interrupted thoracic duct is probably the best explanation. Temporary obstruction of the left subclavian vein, also discussed by Gruwez, could have played a rôle or could have been entirely responsible.

Filariasis is a more common cause of chyluria than chylothorax. Fifteen cases of chyluria were reported by Koehler and associates.[7]

Thrombosis of the subclavian vein has been incriminated as a cause of chylous reflux into the lungs and pleura by Maier,[9,10] and he believes this may be one cause of idiopathic pulmonary fibrosis. However, obstruction of the thoracic duct experimentally did not produce any long standing chylothorax or chylous ascites but only temporary lymphangiectasia and pleural effusion.[1]

A high obstruction of the thoracic duct was reported by White and Urguhart,[19] and this was probably secondary to trauma. Despite obstruction of the thoracic duct in

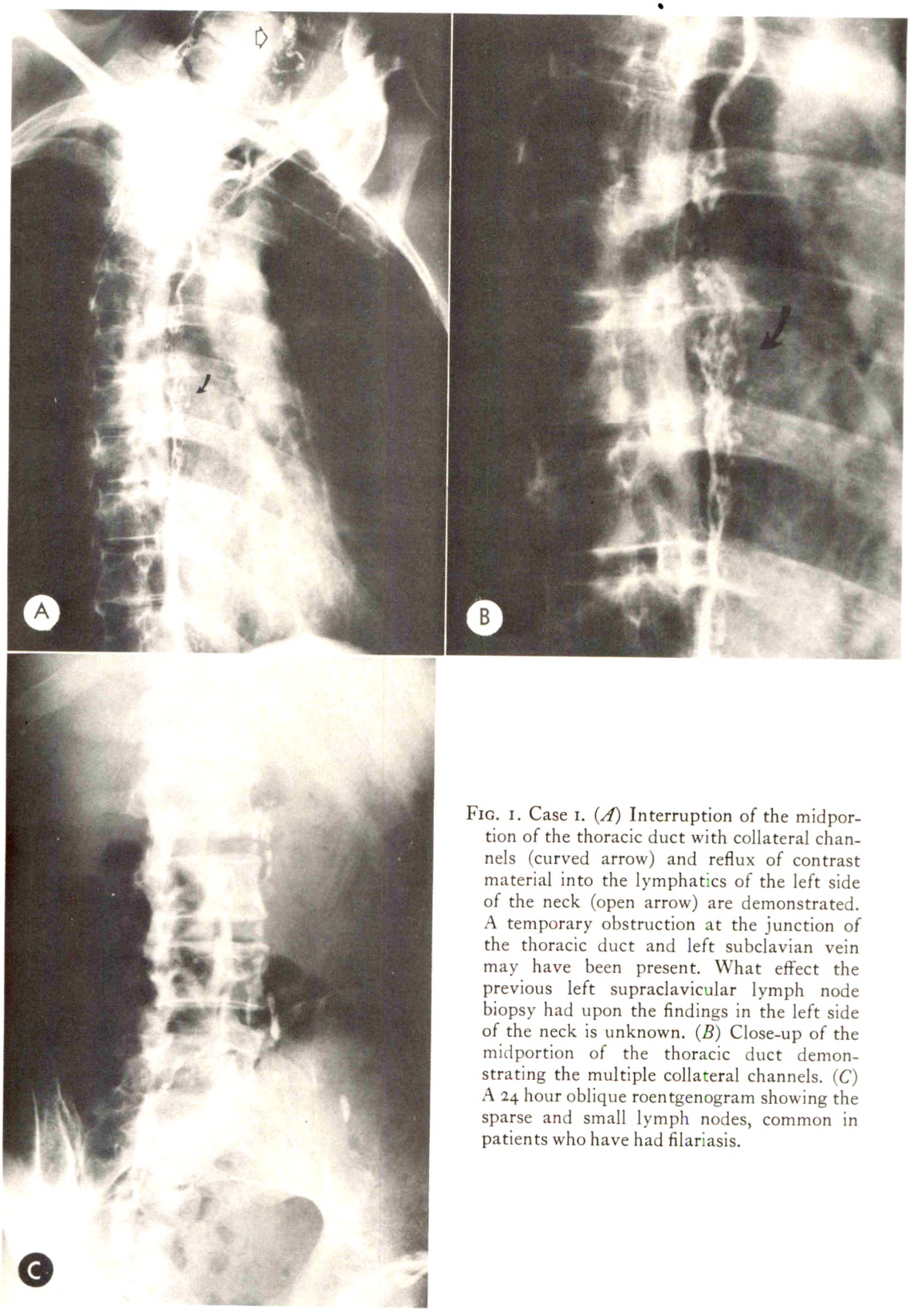

Fig. 1. Case 1. (*A*) Interruption of the midportion of the thoracic duct with collateral channels (curved arrow) and reflux of contrast material into the lymphatics of the left side of the neck (open arrow) are demonstrated. A temporary obstruction at the junction of the thoracic duct and left subclavian vein may have been present. What effect the previous left supraclavicular lymph node biopsy had upon the findings in the left side of the neck is unknown. (*B*) Close-up of the midportion of the thoracic duct demonstrating the multiple collateral channels. (*C*) A 24 hour oblique roentgenogram showing the sparse and small lymph nodes, common in patients who have had filariasis.

the neck in their patient, no swelling of the neck occurred, but dilated mediastinal and pulmonary lymphatics were demonstrated by lymphangiography.

There are a number of benign conditions, which can cause lymph node abnormalities and can be confused with malignancy, as reported by Parker *et al.*[14] Chylothorax, however, was not reported in any of their cases.

Case II. Localized Obstruction of the Thoracic Duct with Lymphangiectasis (Fig. 2). A 72 year old white male was admitted to the Good Samaritan Hospital, Phoenix, Arizona from another institution with a history of recurrent chylothorax. The chylothorax was noted on routine chest roentgenograms during an admission for excision of chronic sialadenitis of the submaxillary gland. Past history was remarkable only in that the patient had a 14 year history of atrial fibrillation treated with digoxin. Repeated thoracentesis at that time yielded chylous fluid on each occasion. On admission to Good Samaritan Hospital, the physical examination was unremarkable except for slight liver enlargement. Laboratory studies were normal. A technetium sulfur colloid scan revealed a defect on the inferior margin of the liver. A lymphangiogram showed partial obstruction of the thoracic duct at the level of the 11th thoracic vertebra with questionable erosion of the anterior margin of the 12th vertebra. Aside from postinflammatory and fibrotic changes in the lower abdominal and pelvic lymph nodes, no other disease was seen. A liver biopsy was unremarkable and the patient was subjected to laparotomy. At surgery, a large inclusion cyst and several smaller cysts of the liver were noted, which pathologically were benign, unrelated ductal cysts. A "softball size" mass at the base of the mesentery was palpated, and was fixed to the mesentery. Multiple deep biopsies revealed ectatic lymph vessels within adipose connective tissue associated with mild chronic inflammation. A review of this tissue revealed no evidence of lymphangioma. The patient was discharged with no specific therapy and required several thoracenteses thereafter. Reaccumulation of the chylothorax gradually ceased, however, and the patient is apparently well one year later.

Comment. This patient evidentally developed an inflammatory reaction around the origin of his thoracic duct, which caused partial obstruction and led to lymphangiectasis and chylothorax. The etiology of the inflammation as well as the significance of the change in the vertebral body are entirely unclear. Since the abnormality apparently was localized to one area and the patient made a good recovery, this case appears unrelated to Case III.

Case III. Idiopathic Lymphangiectasis with Lymph Node Hypoplasia (Fig. 3). This 30 year old white female was referred to the University of Arizona Medical Center for evaluation of a left pleural effusion. Upon admission the physical findings were limited to the left side of the chest and on roentgen examination a large left effusion was noted with a smaller one on the right side. Thoracentesis on the left revealed a chylous effusion. A diagnostic left thoracotomy was carried out but was unrewarding. Because of rapid reaccumulation of the left effusion following surgery, a second unsuccessful thoracotomy was made in an attempt to find and ligate the thoracic duct. Thereafter the patient was treated over an extended period of time with bilateral chest tube drainage. Shortly after the second thoracotomy a lymphangiogram revealed hypoplastic lymph nodes, and multiple abnormal collateral channels in the abdomen extending to the left side of the diaphragm, many of which were ectatic. The patient was later released from the hospital and treated with a dietary regimen. She was readmitted on several occasions with chylous ascites, lymphedema, and recurrent pleural effusions. Gradual deterioration occurred with increasing shortness of breath secondary to the recurrent pleural effusions. Despite all supportive measures she expired 15 months after the initial admission to the hospital. Autopsy revealed idiopathic lymphangiectasia with widespread lymphatic dilatation and an increased number of lymphatic channels in the lungs, gastrointestinal tract, and nodal tissue. There was no evidence of a lymphangioma or other neoplasm.

Comment. Despite careful interrogation of this patient no past illness could be elicited to account for the lymphangiectasia or hypoplasia of the lymph nodes. Although in some cases pulmonary fibrosis and smooth muscle hyperplasia are associated with lymphangiectasia, none was present

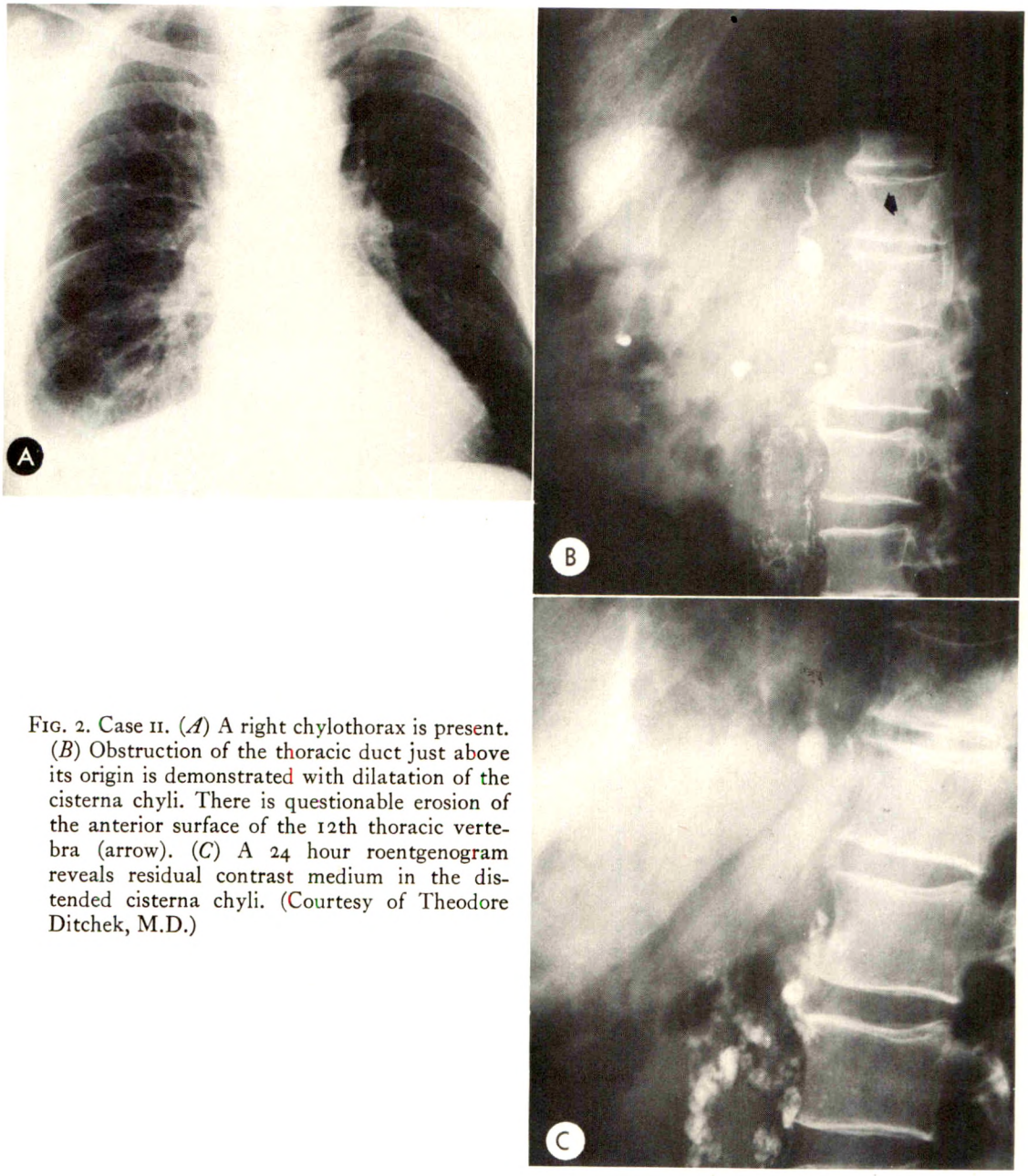

Fig. 2. Case II. (*A*) A right chylothorax is present. (*B*) Obstruction of the thoracic duct just above its origin is demonstrated with dilatation of the cisterna chyli. There is questionable erosion of the anterior surface of the 12th thoracic vertebra (arrow). (*C*) A 24 hour roentgenogram reveals residual contrast medium in the distended cisterna chyli. (Courtesy of Theodore Ditchek, M.D.)

in this patient. Camiel *et al.*[3] in 1971 reported a similar case. Their patient also had chylous effusions in the abdomen and chest and hypoplastic lymph nodes, but they did not report lymphangiectasis. Lymphocytopenia was present, however, which they believed to be due to lymphocyte depletion, as at autopsy no malignancy was found. These authors were able to demonstrate free contrast medium within the chylothorax following lymphangiography. In our patient the thoracic duct was well demonstrated, and in both cases the possibility of minute trauma superimposed upon a congenital lymphatic insufficiency must be considered.

Case IV. Lymphangioma (Fig. 4). This eight and one half year old white female child initially was admitted to the University of Utah Hospital for a limp associated with a short extrem-

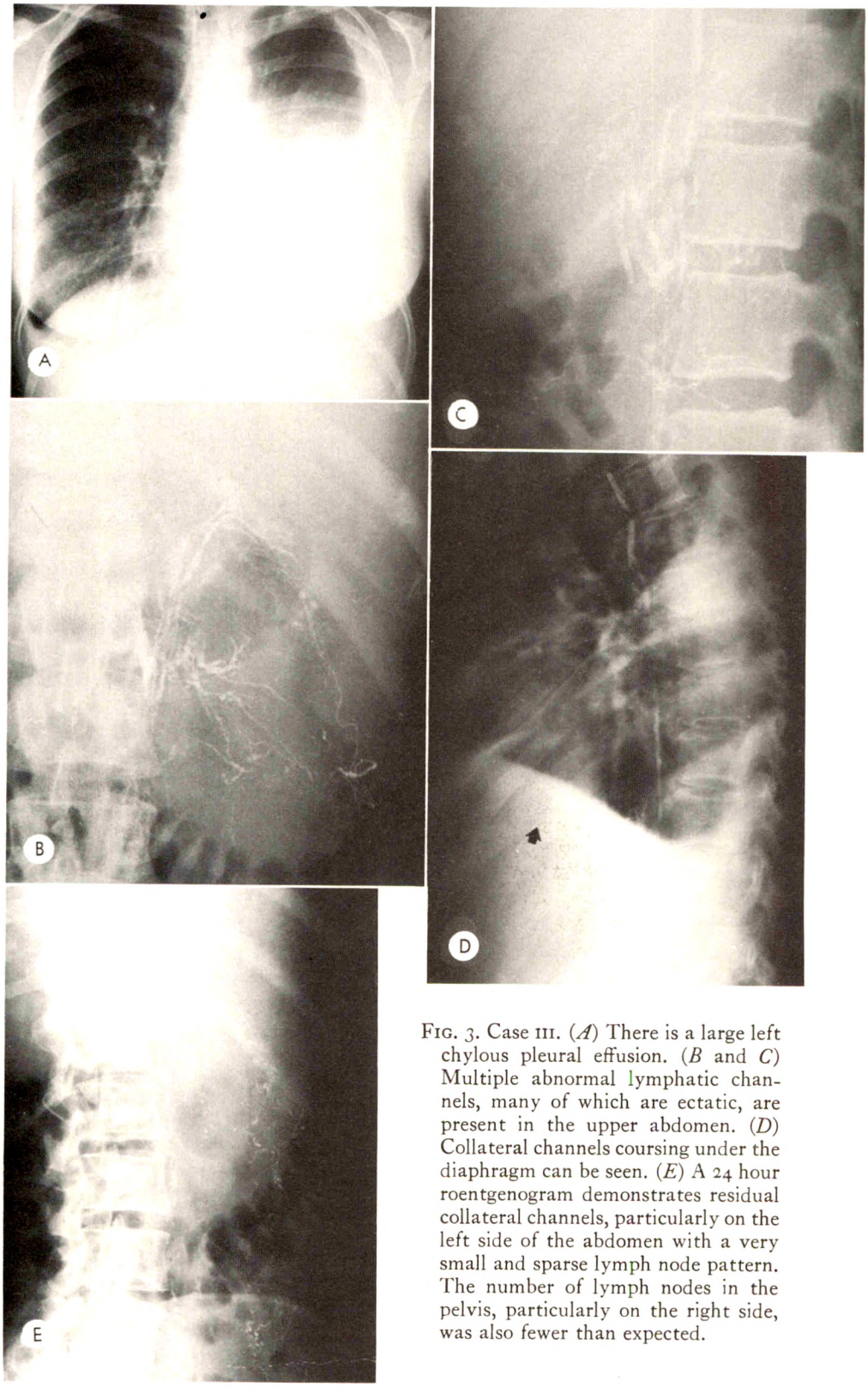

FIG. 3. Case III. (*A*) There is a large left chylous pleural effusion. (*B* and *C*) Multiple abnormal lymphatic channels, many of which are ectatic, are present in the upper abdomen. (*D*) Collateral channels coursing under the diaphragm can be seen. (*E*) A 24 hour roentgenogram demonstrates residual collateral channels, particularly on the left side of the abdomen with a very small and sparse lymph node pattern. The number of lymph nodes in the pelvis, particularly on the right side, was also fewer than expected.

ity. A bone lesion was noted roentgenographically in the left femur and a subsequent biopsy was interpreted as a capillary hemangioma. The patient later sustained multiple pathological fractures of the extremity. An arteriogram was carried out in a retrograde manner through the right femoral artery and showed accelerated filling of the venous system in the left thigh, but there was no typical blush of a hemangioma. A lymphangiogram demonstrated abnormal lymphatic vessels corresponding to the level of soft tissue involvement but the contrast medium became too dilute to demonstrate a communication with the osseous lesion. Despite perfusion with sclerosing agents the lesion increased in size and after a fourth pathological fracture, amputation was carried out. Milky lymphatic fluid, probably chyle, was noted exuding both from the fracture site initially, and later, following the amputation, from the stump as well. The final diagnosis was angioma, probably lymphangioma. The patient was readmitted approximately four years following the first admission with shortness of breath. Chest roentgenograms revealed a large right effusion, which upon examination following thoracentesis was chylous. Later during the same admission mediastinal widening was noted and a lymphangioma of the mediastinum was suspected. A lymphangiogram carried out at that time demonstrated multiple lymphatic vessels coursing into the mediastinum compatible with a lymphangioma. The patient was discharged from the hospital on supportive therapy.

Comment. A primary lymphangioma or lymphangiomatosis is usually characterized by tumors in the mediastinum or retroperitoneal area which involve the lymphatic system and usually the thoracic duct. The chylothorax is probably secondary in these cases to obstruction by the tumor with collateral channels formed between the chyle bearing lymphatics of the abdomen and the pleural space. The tumor is probably hamartomatous in origin, nonmetastasizing, and consists histologically of smooth muscle bundles interspersed with anastomosing lymphatic channels.[12] The channels and cystic spaces are lined with flat cuboidal endothelial cells and are separated by fibrous septa of varying thickness. While a lymphangioma or lymphangiomatosis is a true neoplasm, histologically distinct and quite different from lymphangiectasis, the two conditions can be at times confused even histologically.[15] A localized collection of ectatic lymph vessels, lymphangiectasis, whether primary or secondary, should not be classified as a neoplasm.

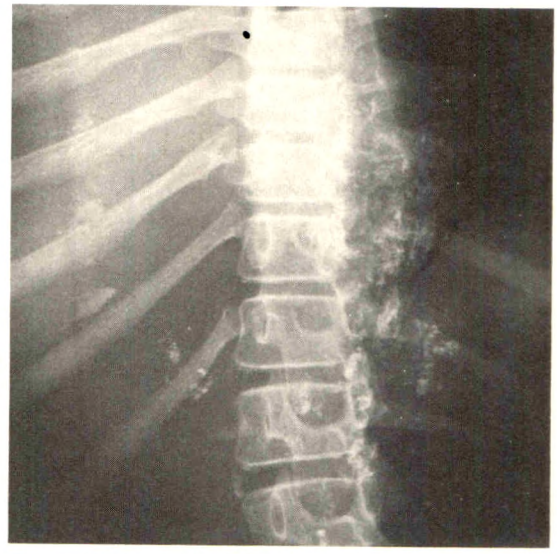

Fig. 4. Case iv. Multiple bizarre, ectatic channels are demonstrated in the mediastinum compatible with lymphangiomatosis. (Courtesy of P. Ruben Koehler, M.D.)

Case v. Obstruction Secondary to Lymphoma (Fig. 5.) This 59 year old white female was admitted to the University of Arizona Medical Center for treatment of lymphoma. Four months prior to admission the patient noticed a lump on the right side of her neck for which she did not seek medical care until two weeks prior to admission. At that time she was admitted to another institution and a biopsy was carried out. Pathologic examination of the specimen revealed nodular lymphocytic lymphoma. Physical examination upon admission to the University of Arizona Medical Center revealed diffuse bilateral superficial lymphadenopathy and moderate splenomegaly. A chest roentgenogram demonstrated a left pleural effusion as well as probable mediastinal and hilar lymphadenopathy. A left thoracentesis was carried out and 1,200 cc of a milky brownish-white fluid was removed, which on

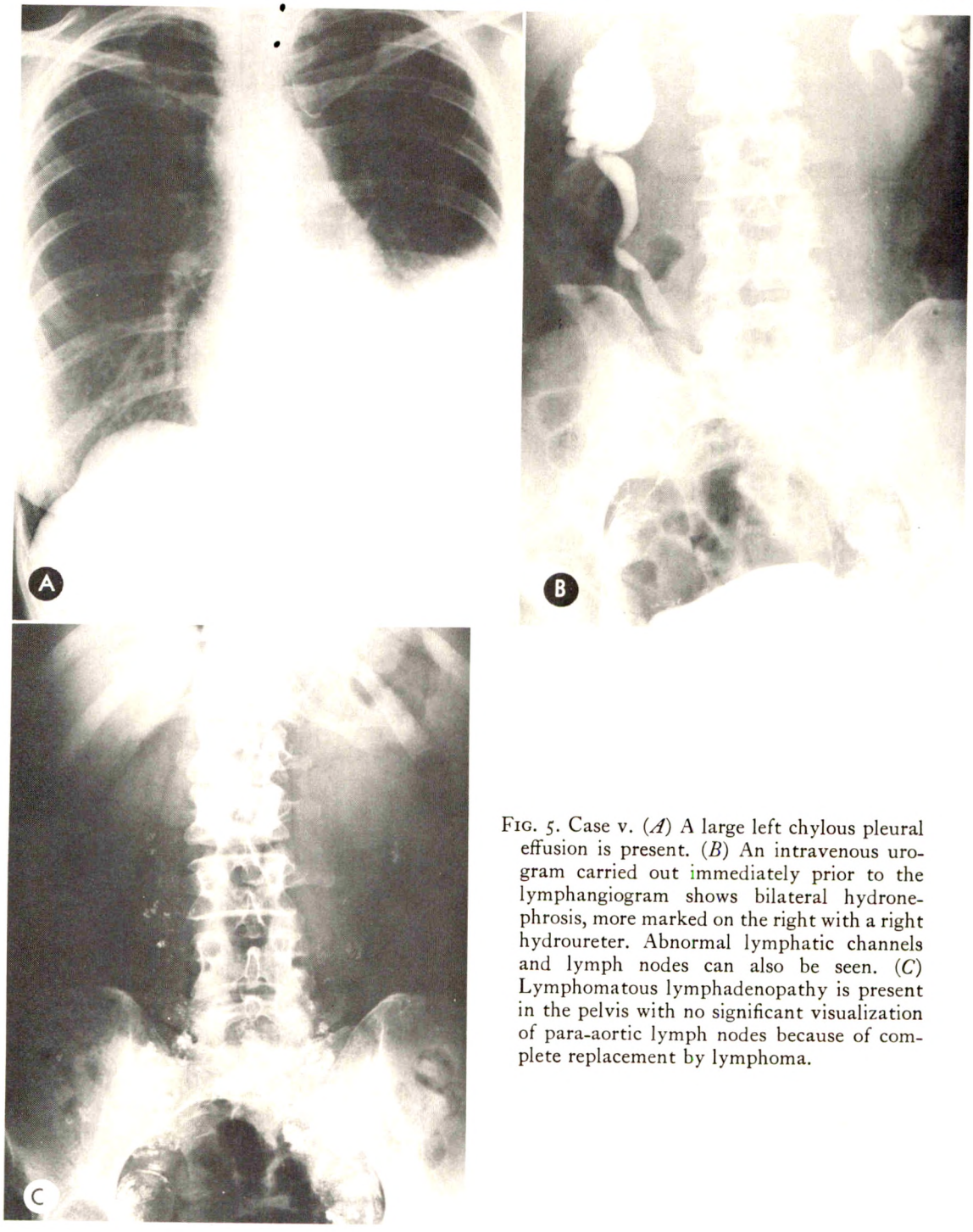

FIG. 5. Case v. (*A*) A large left chylous pleural effusion is present. (*B*) An intravenous urogram carried out immediately prior to the lymphangiogram shows bilateral hydronephrosis, more marked on the right with a right hydroureter. Abnormal lymphatic channels and lymph nodes can also be seen. (*C*) Lymphomatous lymphadenopathy is present in the pelvis with no significant visualization of para-aortic lymph nodes because of complete replacement by lymphoma.

examination was found to be chyle. Two days after admission an intravenous urogram revealed retroperitoneal masses displacing each ureter, and a lymphangiogram on the same day demonstrated massive lymphoma with involvement of all the lymph nodes in the pelvis. There was very poor filling of the retroperitoneal para-aortic lymph nodes. The patient then un-

derwent a staging laparotomy, which included splenectomy and biopsy of the liver, multiple lymph nodes, and bone marrow. The entire retroperitoneal space on both sides was filled with large firm tumor masses. Pathologic examination of the surgical specimens revealed nodular lymphocytic lymphoma involving the pelvic and mesenteric lymph nodes, spleen and liver. Following recovery from surgery the patient was treated with limited local irradiation to the pelvis and placed on maintenance chemotherapy.

Comment. Although lymphangiectasis secondary to lymphoma was not visualized in this patient, chyle was undoubtedly carried by collateral channels through the diaphragm into the left pleural space.

Case vi. Obstruction Secondary to Lymphoma (Fig. 6). This 36 year old white male was admitted to the M. D. Anderson Tumor Institute and Hospital with a previously biopsy proved lymphoma. On initial examination peripheral lymphadenopathy was present as well as an abdominal mass. He was treated as an out-patient with a chemotherapeutic agent and readmitted to the hospital a few months later. Physical examination revealed peripheral cervical lymphadenopathy, a small midabdominal mass, minimal hepatomegaly, and splenomegaly. Chest roentgenograms revealed a massive left pleural effusion, which was found to be chylous. A lymphangiogram demonstrated multiple collateral channels in the upper abdomen with probable obstruction of the thoracic duct in the lower portion of the thorax. Biopsy of the right inguinal lymph node area revealed a nodular poorly differentiated lymphocytic lymphoma. The patient was referred for radiation therapy and maintenance chemotherapy.

Comment. Chylothorax is much more common secondary to malignancies other than a primary lymphangioma. In a series of cases from the Mayo Clinic, Roy and coworkers[16] found 67 percent of their 52 cases secondary to some neoplastic condition. Only one of these was found to be benign while 23 out of 34 malignancies were due to lymphoma. McFarland and Holman[11] found malignancy as a cause of chylothorax in seven out of 15 cases, and lymphoma was responsible for four of these. Bower[2] in 20 cases found ten secondary to a neoplastic condition with six due to lymphoma. It has been postulated that the malignant involvement of the para-aortic lymph nodes and cisterna chyli leads to obstruction and chyle then seeks collateral lymphatic pathways

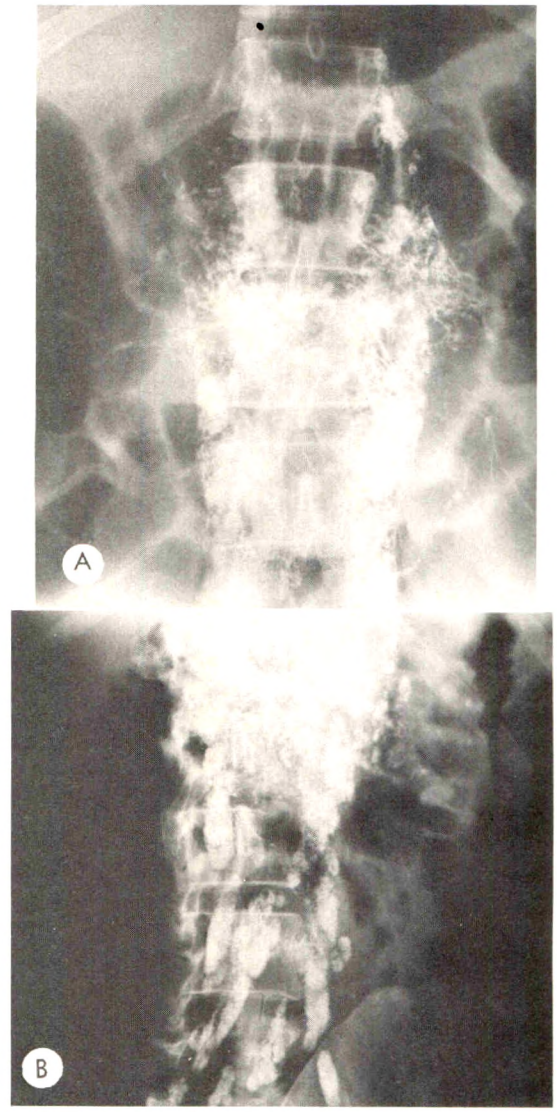

Fig. 6. Case vi. (*A*) Multiple abnormal lymphatic channels, some of which are moderately ectatic, were demonstrated in this patient who had chylothorax at this time. (*B*) A 24 hour roentgenogram demonstrates multiple abnormal lymph nodes in the upper abdomen, mostly on the left side. (Courtesy of Sidney Wallace, M.D.)

through the abdomen and diaphragm often ending in the pleural space, although chylous ascites may also occur. Secondary lymphangiectasis in these cases is common. While the eventual outcome of the neoplastic condition is rarely favorable, treatment of the underlying disease with radiation or chemotherapy frequently leads to a regression of the chylothorax if the obstruction is relieved.

NEONATAL CHYLOTHORAX

Yancy and Spack[20] in a thorough review of the literature in 1967 reported on 36 cases of neonatal pleural effusion, 18 of which were chylous. The etiology is unknown and birth trauma has been suspected. However, although most recovered, in seven of their autopsied cases, no damage to the thoracic duct was found.

CONCLUSIONS

It can be safely assumed that chylothorax is secondary either to interruption or obstruction of the lymphatic system. In the absence of a traumatic incident, except for neonatal chylothorax, one may conclude that the chylothorax is due to lymphatic obstruction with formation of collateral channels. Chylous lymphatic fluid finds its way into the pleural space through these collateral pathways, which are often ectatic. A lymphangiogram done at this point should determine the site and possibly the cause of the obstruction. The most important differentiation will be between a neoplastic and a benign condition. If neoplasm can be excluded, the next differential point will be to determine whether the patient has an inadequate lymphatic system secondary to postinflammatory fibrosis or idiopathic lymph node hypoplasia. A local obstructing condition, whether benign or neoplastic, may well be amenable to therapy, even though the therapy may be only temporarily beneficial. The lymphangiogram is certainly the key diagnostic test as it should determine whether the basic disease is benign or malignant, generalized or localized, and may even point toward a specific diagnosis. In addition, it should provide useful information for the surgeon or radiation oncologist in planning therapy.

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REFERENCES

1. Benninghoff, D., Camiel, M., and Takashima, T. Clinical and experimental studies of chylous reflux. *Prog. in Lymphol.*, 1970, *2*, 269–271.
2. Bower, G. Chylothorax. *Dis. Chest*, 1964, *46*, 464–468.
3. Camiel, M., Benninghoff, D., and Alexander, L. Chylous effusions, extravasation of lymphographic contrast material, hypoplasia of lymph nodes and lymphocytopenia. *Chest*, 1971, *59*, 107–110.
4. Gruwez, J., Cardoen, G., Dive, C., and Beart, A. Chylothorax. *Prog. Lymphol.*, 1967, *1*, 30–36.
5. Jahsman, W. Chylothorax: brief review of literature: report of 3 non-traumatic cases. *Ann. Int. Med.*, 1944, *21*, 669–678.
6. Kinmouth, J., and Taylor, G. Lymphatic circulation in lymphedema. *Ann. Surg.*, 1954, *139*, 129–136.
7. Koehler, P. R., Chiang, T.-C., Lin, C.-T., Chen, K.-C., and Chen, K-Y. Lymphography in chyluria. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *102*, 455–465.
8. Laumonier, P., Lachapéle, A., Couraud, L., Hugues, A., Lagarde, C., and Mage, J. Une application de la lymphographie au diagnostic et au traitement du chylothorax. *Presse med.*, 1962, *70*, 2630–2635.
9. Maier, H. Pulmonary and pleural lymphatics: challenge to thoracic explorer. *J. Thoracic & Cardiovasc. Surg.*, 1966, *52*, 155–163.
10. Maier, H. C. Chylous reflux in lungs and pleurae. *Thorax*, 1968, *23*, 281–296.
11. McFarland, J., and Homan, C. Chylothorax. *Am. Rev. Resp. Dis.*, 1972, *105*, 287–291.
12. Miller, W. T., Cornog, J. L., Jr., and Sullivan, M. A. Lymphangiomyomatosis: clinical-roentgenologic-pathologic syndrome. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *111*, 565–572.
13. Nusbaum, M., Baum, S., Hedges, R., and Blakemore, W. Roentgenographic and direct visualization of thoracic duct. *A.M.A. Arch. Surg.*, 1964, *88*, 105–113.
14. Parker, B. R., Blank, N., and Castellino, R. A. Lymphographic appearance of benign

conditions simulating lymphoma. *Radiology*, 1974, *111*, 267–274.
15. Rappaport, H. Tumors of hematopoietic system. In: Atlas of Tumor Pathology. Armed Forces Institute of Pathology, Washington, D. C., 1966, 3, 359–363.
16. Roy, P., Carr, D., and Payne, W. Problem of chylothorax. *Mayo Clin. Proc.*, 1967, *42*, 457–467.
17. Van Pernis, P. Variations of thoracic duct. *Surgery*, 1949, *26*, 806–809.
18. Wallace, S., Jackson, L., Schaffer, B., Gould, J., Greening, R., and Kramer, S. Lymphangiograms: their diagnostic and therapeutic potential. *Radiology*, 1961, *76*, 179–199.
19. White, W., and Urguhart, W. Demonstration of pulmonary lymphatics by lymphography in patient with chylothorax. *Clin. Radiol.*, 1966, *17*, 92–94.
20. Yancy, W., and Spack, A. Spontaneous neonatal pleural effusion. *J. Pediat. Surg.*, 1967, *2*, 313–319.

PERIPHERAL VASCULAR PERFUSION SCANNING*

CORRELATION WITH THE ARTERIOGRAM AND CLINICAL ASSESSMENT IN THE PATIENT WITH PERIPHERAL VASCULAR DISEASE

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ABSTRACT:

Peripheral vascular perfusion scan during rest and reactive hyperemia does not necessarily offer the same information as the arteriogram or the clinical assessment of the peripheral pulses. The information obtained from the peripheral vascular perfusion scan offers potentially clinically useful information regarding the physiologic significance of various arterial lesions to the surgeon in selection of his therapeutic approach. The peripheral vascular perfusion scan uniquely and under varied physiologic conditions permits assessment of the effect of arterial disease on the distribution of perfusion within the extremity. The peripheral vascular perfusion scan is not in competition with, but is complementary to, arteriography and clinical assessment of the patient with peripheral vascular disease.

A DETAILED anatomical study of the major vessels of the extremity, such as that provided by contrast arteriography, is essential in the preoperative evaluation of the patient with suspected peripheral vascular disease. However, neither the physiologic significance of the detected large vessel disease, in terms of the effect on the relative distribution of perfusion, nor the extent and distribution of perfusion from visualized or nonvisualized collaterals can be assessed by what is essentially a morphologic study. The relative distribution of perfusion in an extremity, both at rest and under various physiologic stresses, can, however, be visually assessed by scintillation scanning of the distribution of intra-arterially injected radioactive albumin microspheres.[6,10]

The purpose of this study is to correlate the relative distribution of perfusion within an extremity, as depicted in peripheral vascular perfusion scans performed at rest and during the vasodilatation of reactive hyperemia, with the arteriographic and clinical findings. This was done to assess the physiologic effect of the morphologic changes seen on the arteriogram and to answer this question: Does the peripheral vascular perfusion scan offer the same information as that obtained from contrast arteriography and the clinical assessment of the peripheral pulses? For, if we are indeed evaluating a different aspect of peripheral vascular disease and thereby supplying, not identical, but additional, potentially useful data regarding the nature of the disease, then the answer to this question should be no.

MATERIAL AND METHOD

A consecutive group of 30 patients with symptomatic peripheral vascular disease, who were referred to our cardiovascular laboratory for contrast angiography, was selected for the study. All patients had their perfusion studies at the time of the translumbar arteriogram. The method used is essentially the same as previously described[7] and is briefly as follows: 15 minutes after the completion of translumbar arteriography, with the patient again in the

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Supported by GM10548 and GM01496.

basal state, 5–10 mCi of ^{99m}Tc labelled human serum albumin microspheres (15–30 μ in diameter) were rapidly injected through the aortic catheter, whose tip had been fluoroscopically positioned just below the renal arteries. The patient's legs were then inserted into full leg inflatable stockings which were inflated to 50 mm of Hg above the patient's systolic blood pressure and maintained at that pressure for 5 minutes. After release of the pressure and during the peak of the reactive hyperemic response, 5–10 mCi of ^{113}In labelled albumin microspheres (15–30 μ in diameter) were rapidly injected through the catheter. Anteroposterior rectilinear scans of the extremities, using 5:1 minification were then obtained. One set of scans was made for each isotope.

The perfusion scans and the arteriograms were then evaluated independently as to extent and location of the disease and the possible clinical findings which might be expected. For this, it was assumed that if there was a stenosis of a major vessel of greater than 50 percent, then significant clinical findings such as claudication, absence of peripheral pulses, or an abnormal perfusion pattern on the scan might be expected distal to the stenosis or occlusion.

RESULTS

The following two cases are presented to illustrate examples of what we define as a normal study and one which is abnormal. Figure 1 is a normal rest and reactive hyperemia scan in a 71 year old Caucasian female with vague shooting pains down both legs. The translumbar arteriogram was completely normal. The distribution of the radiopharmaceutical both at rest and during reactive hyperemia is proportional to muscle mass with a relative hypoperfusion to the non-muscular structures of the extremity, *i.e.*, the knee and ankles. In reviewing the six normal cases done to date, it became apparent that, in the normal, the relative perfusion to the calf can be as little as 60 percent of that of the thigh. This is, most likely, related in part to the relative muscle mass difference between the thigh and the calf. Figure 2 is a rest and reactive hyperemia scan of a 41 year old Caucasian male with left calf claudication. The arteriogram demonstrated bilateral superficial femoral artery occlusion with reconstitution at the popliteal level and good run-off bilaterally. As can be seen in comparing the two scans when the circulation is stressed, it is the left calf, where the patient is symptomatic, which is relatively hypoperfused. It should be noted that, although the left calf is relatively hypoperfused, flow which does get to the knee and below tends to go primarily to muscular structures rather than to the knee and ankle, which are primarily non-muscular.

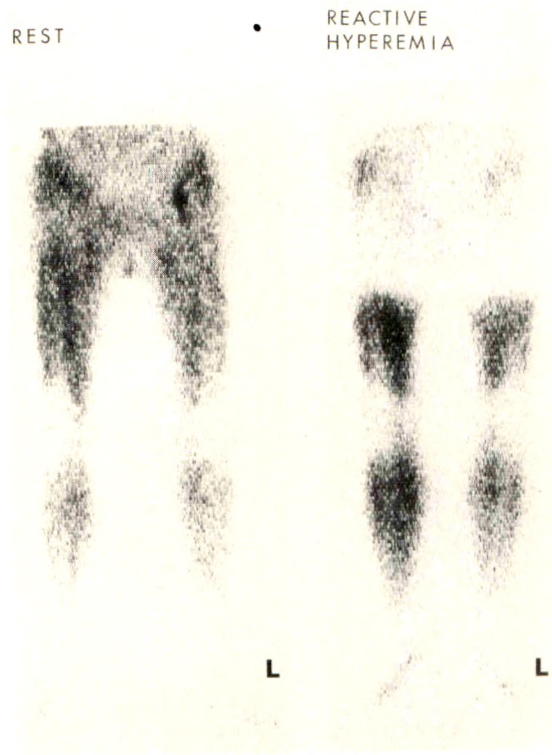

Fig. 1. Normal rest and reactive hyperemia scan in 71 year old female with vague symptomatology and normal arteriogram. Note that the radioactivity is distributed in proportion to muscle mass.

Correlating the extent and location of the disease seen on the rest scans with the arteriogram, we essentially verified the poor correlation as noted in a previous study by Giargiana *et al.*[3] In our present group of patients, 37 percent had a posi-

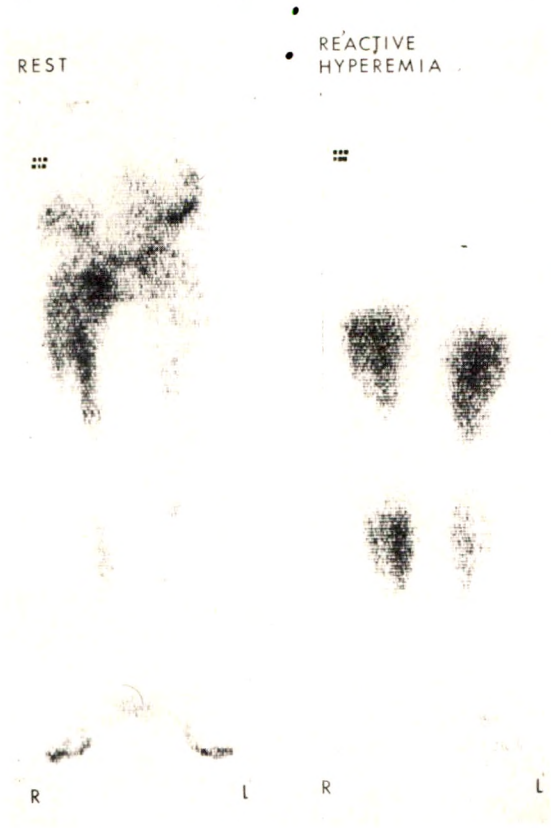

Fig. 2. An abnormal rest and reactive hyperemia scan in 41 year old male with left calf claudication. Note that when circulation is stressed it is the left calf which demonstrates a relative hypoperfusion.

tive correlation between scintillation and arteriographic findings, while 50 percent had less extensive disease and 13 percent had more extensive disease (Table I). Utilizing the reactive hyperemia scan, the distribution of results was quite similar with 40 percent positive correlation, 20 percent more, and 40 percent less extensive disease seen on the scan (Table I). We noted that the vast majority of the patients with more extensive disease on the scan were diabetics with essentially normal arteriograms, in which the scans suggested diffuse small vessel disease.[6,7]

We then attempted to reconstruct the perfusion scan from the knowledge of the presence or absence of the various peripheral pulses. For instance, if the femoral pulse was present, one might expect a normal thigh perfusion pattern, while if a superficial femoral artery was obstructed, with subsequent loss of the popliteal pulse, one might expect an abnormal pattern or relative decrease in perfusion in the ipsilateral calf. Rather than duplicate a previously published and rather tedious investigation,[3] we used only the reactive hyperemia scan for this aspect of the study. We found that if the femoral pulse was present, there was a normal perfusion pattern noted in the thigh of 100 percent of the cases. If the femoral pulse was not palpable, an abnormal perfusion pattern was seen in only 31 percent, while 69 percent still demonstrated a normal perfusion pattern (Table II).

In reviewing the calf perfusion pattern in relation to the presence or absence of the various pulses we noted the following: (1) when the popliteal pulse was present, 90 percent of the cases had a normal calf perfusion pattern, while ten percent had an abnormal pattern; (2) when the popliteal pulse was absent there was approximately a 50 percent chance of still having a normal perfusion pattern in the calf; (3) the distribution of the data in relation to the dorsalis pedis and posterial tibial pulses was quite similar in that there was a high probability of having a normal perfusion pattern when the pulse was present and

Table I

CORRELATION OF PERFUSION SCANS AND ARTERIOGRAMS

Category	No. of Patients	Percent of Total
Rest Scan		
Positive correlation of scan and arteriogram	11	37
Less extensive disease on scan	15	50
More extensive disease on scan	4	13
Reactive Hyperemic Scan		
Positive correlation of scan and arteriogram	12	40
Less extensive disease on scan	12	40
More extensive disease on scan	6	20

TABLE II

CORRELATION OF REACTIVE HYPEREMIA PERFUSION OF THE THIGH AND PRESENCE OF FEMORAL PULSE

	Total No. of Extremities	Normal Perfusion Scan Pattern (Percent)	Abnormal Perfusion Scan Pattern (Percent)
Femoral pulse present	47	100	0
Femoral pulse absent	13	69	31

approximately a 50 percent chance of still having a normal perfusion pattern when the pulse was absent (Table III).

The final comparison of the arteriogram, the perfusion scans, and the clinical data involved the ability to reconstruct, without prior clinical knowledge of the patients, their symptomatology using the arteriogram and the scans. A major muscle group of the extremity was either symptomatic or not, with no attempt made to grade or correlate the severity of the symptoms with the finding on the arteriogram or scans.

Using the arteriogram our predictions agreed with the patients' symptomatology in only 53 percent of cases. Using the rest scan alone, the results were similar, with agreement in 60 percent of cases. However, when viewing the rest and reactive hyperemia scans and noting the changes which occur in the relative distribution of perfusion to the muscular and non-muscular elements of the extremity, there was agreement in 83 percent of the cases (Table IV).

DISCUSSION

One prerequisite for acceptance of a new diagnostic or prognostic procedure is that it must offer previously obtainable information more easily or offer new, potentially clinically useful information about a disease process.

At present, information of various types can be obtained concerning the status of the peripheral circulation. In addition to the history and physical examination, numerous instrumental methods have been applied to comprehend more fully the pathologic process of peripheral vascular disease.[5,11] Some, such as ^{133}Xe clearances, examine the actual blood flow per weight of tissue of a very minute portion of the involved extremity,[1] while others rely on the presence of pulsatile flow in major vessels.[9] The most widely accepted method, contrast angiography, is a morphologic study and, therefore, although it offers detailed anatomical information of the major vessels, it offers very little information about

TABLE III

CORRELATION OF REACTIVE HYPEREMIA PERFUSION PATTERN OF THE CALF AND PRESENCE OF PERIPHERAL PULSES

	Total No. of Extremities	Normal Perfusion Scan Pattern (Percent)	Abnormal Perfusion Scan Pattern (Percent)
Popliteal pulse present	18	92	8
Popliteal pulse absent	42	55	45
Dorsalis pedis pulse present	16	88	12
Dorsalis pedis pulse absent	44	57	43
Posterior tibial pulse present	15	87	13
Posterior tibial pulse absent	45	54	46

TABLE IV

CORRELATION OF PATIENT'S SYMPTOMATOLOGY WITH ARTERIOGRAM AND SCAN FINDINGS

	No. of Patients	Percent of Total
Arteriograms and Patient's Symptomatology		
Agreement	16	53
No agreement	14	47
Rest Scans and Patient's Symptomatology		
Agreement	18	60
No agreement	12	40
Reactive Hyperemia Scans and Patient's Symptomatology		
Agreement	25	83
No agreement	5	17

the physiologic effect of the visualized disease in terms of perfusion. For this reason, in a manner analogous to the pulmonary perfusion scan, the intra-arterial injection of radioactive particles was applied to the evaluation of perfusion of the extremities.[4] This permitted an evaluation of the relative distribution of perfusion in the muscular and non-muscular portions of the extremity at the level of the microcirculation. It also permits one to assess the physiologic significance of the vascular disease in terms of alterations in relative perfusion, and the relative effectiveness of visualized and non-visualized collateral vessels to perfuse the muscular and non-muscular segments of the extremity.

As was seen by the poor correlation in both the rest and reactive hyperemia scans, the stenotic lesion on the arteriogram does not necessarily imply an alteration in relative perfusion within the extremity as defined by our study. However, the ability to visualize the changes which do occur in relative distribution of perfusion when the circulation is stressed has permitted our surgical colleagues to appreciate more fully which of the frequently multiple, arteriographic abnormalities are physiologically significant. Although at this time our experience in utilizing this information in this manner is limited, it appears to be helpful in terms of noting points of fixed resistance and noting which end organ receives its blood supply. For example, the scans in Figure 2 suggest that in this case if increased flow could be brought down the patent channels in the extremity, by bypassing the physiologically significant proximal obstruction, its distribution will be primarily to the muscular structures, where it is needed. As we have previously reported,[3,6] many patients do not have this distribution pattern and tend to distribute the perfusion in their extremity in proportion to surface area rather than muscle mass or, with stress, to increase the relative proportion of perfusion to the non-muscular structures. The decision as to which surgical approach, if any, is indicated in a particular patient is frequently difficult.[2,8] This information, although only recently applied in this manner, has aided in formulating the optimum surgical approach to the particular patient.

Our findings of poor (55 percent) correlation of the rest scan and good correlation (85 percent) of the reactive hyperemia scan with the patient's symptomatology supports the concept that when looking at the two studies together one is looking at the distribution of perfusion both at rest, when many patients are asymptomatic, and under stress, when the patient is symptomatic. The poor correlation between the arteriogram and the patient's symptomatology does not in any way degrade the arteriogram but affirms the hypothesis that the arteriogram is basically a morphologic study and does not necessarily yield data regarding the physiologic significance of the disease it detects.

Finding a normal distribution of relative perfusion in approximately 50 percent of cases with no palpable pulses proximal to the muscle bundle in question supports the concept that a palpable pulsatile arterial flow is not a prerequisite for normal relative distribution of flow in an extremity. In this same regard, we also see that the presence of palpable arterial flow indicates a 90 percent probability that the relative dis-

tribution of perfusion distal to that pulse will be normal.

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The authors gratefully acknowledge the expert technical assistance in performing these studies of Carole Plaza, R.T., Marie Pasinski, R.T., Wayne Kasecamp, R.T., and Madleen Murrell, R.T.

REFERENCES

1. Alpert, J. S., Larson, O., and Lassen, N. A. Evaluation of arterial insufficiency of legs: comparison of arteriography and ^{133}Xe walking test. *Cardiovasc. Res.*, 1968, *2*, 161–169.
2. Dickenson, P. H., McNeil, I. F., and Morrison, J. M. Aorto-iliac occlusion: review of 100 cases treated by direct surgery. *Brit. J. Surg.*, 1967, *54*, 764–770.
3. Giargiana, F. A., Jr., Siegel, M. E., James, A. E., Rhodes, B. A., Wagner, H. N., Jr., and White, R. I., Jr. Preliminary report on complementary roles of arteriography and perfusion scanning in assessment of peripheral vascular disease. *Radiology*, 1973, *108*, 619–627.
4. Jones, E. L., Wagner, H. N., Jr., and Zuidema, G. D. New method for studying peripheral circulation in man. *A.M.A. Arch. Surg.*, 1965, *91*, 725–734.
5. Petersen, F. B., and Sizzaard-Anderson, J. Simultaneous venous occlusion plethysmography and ^{133}Xe clearance in patients with arteriosclerosis of lower extremities: attempt to evaluate blood flow in skin and muscle. *Scandinav. J. Thoracic & Cardiovasc. Surg.*, 1969, *3*, 30–35.
6. Rhodes, B. A., Greyson, N. D., Siegel, M. E., Giargiana, F. A., White, R. I., Jr. Williams, G. M., and Wagner, H. N., Jr. Distribution of radioactive microspheres in patients with peripheral vascular disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *118*, 820–826.
7. Siegel, M. E., Giargiana, F. A., Jr., Rhodes, B. A., White, R. I., Jr., and Wagner, H. N., Jr. Effect of reactive hyperemia on distribution of radioactive microspheres in patients with peripheral vascular disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *118*, 814–819.
8. Strandness, D. E., Jr. Selection of patients for direct arterial surgery. In: Peripheral Arterial Disease: A Physiologic Approach. Little, Brown & Company, Boston, 1969, pp. 129–144.
9. Strandness, D. E. Jr., Schultz, R. D., and Sumnew, D. S. Ultrasonic flow detection: useful technique in evaluation of peripheral vascular disease. *Am. J. Surg.*, 1967, *113*, 311–320.
10. Wagner, H. N., Jr., Jones, E. L., Tow, D. E., and Langan, J. K. Method for study of peripheral circulation in man. *J. Nuclear Med.*, 1965, *6*, 150–154.
11. Winsor, R. Instrumental methods for studying peripheral arterial circulation. *Cardivasc. Clin.*, 1971, *3*, 17–35.

EXPERIENCE IN PARATHYROID SCANNING*

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ABSTRACT:

In our experience, the low success rate of ^{75}Se parathyroid studies does not justify continuance of the test as a routine diagnostic procedure. The initial results using the $^{99m}Tc\,O_4^-$—^{131}I subtraction technique are encouraging but further experience is required in order to ascertain whether this procedure will justify a place as a routine test in the localization of parathyroid adenoma.

THE greater awareness and consequent earlier diagnosis of hyperparathyroidism on clinical and biochemical grounds have increased the importance of improving methods of preoperative localization of parathyroid adenomas. While there have been significant advances in angiographic and radioimmunoassay techniques[5,7,8] over the past few years, a satisfactory non-invasive technique is yet to be found, although preliminary experience with parathyroid thermography is encouraging.[9] Parathyroid scanning has never become a routine diagnostic investigation because the standard selenomethionine (^{75}Se) studies have in most series been shown to give a low diagnostic yield.[3,4] In view of this, it was decided to try new methods of parathyroid adenoma visualization with the aim of evolving a more satisfactory scanning technique. This article reviews the first 50 parathyroid studies performed on 42 patients in the Department of Nuclear Medicine, Repatriation General Hospital, Melbourne, over the past two years. The results confirm the inadequacy of routine ^{75}Se studies in the localization of parathyroid adenomas. Our experience with various subtraction techniques is documented, and follow-up in these cases suggests that parathyroid subtraction scanning may prove to be a worthwhile investigation in the diagnosis of parathyroid adenoma.

MATERIAL AND METHOD

All 42 patients were suspected of having a parathyroid adenoma on clinical and biochemical grounds. In more than half the patients there was a preceding history of recurrent renal calculi. Eight of the patients

TABLE I

RELIABILITY OF PARATHYROID SCANNING

	Adenomas No.	+VE Scans		Confirmed −VE Scans	Total Scans	Correct Scan Result	
		No.	Percent	No.	No.	No.	Percent
Dual peak ^{75}Se	14	3	21	14	29	17	58
Sequential ^{99m}Tc	4*	3	75	4	6	5	83
^{75}Se—^{131}I subtraction	3	1	33	1	4	2	50
^{75}Se—^{99m}Tc subtraction	1	1	100	1	2	2	100
^{99m}Tc—^{131}I-subtraction	6	4	66	2	9‡	6	75
	28†	12	42.8	20	50	32	64

* One patient had three parathyroid adenomas.
† Eight patients had two different scanning techniques used.
‡ In one case the study was positive but follow-up nine months later revealed that the patient was advised to have operation, but refused.

* From the Department of Nuclear Medicine, Repatriation General Hospital, Heidelberg, Melbourne, Victoria, Australia.

TABLE II

SIZE OF PARATHYROID ADENOMAS IN $^{99m}Tc\ O_4^-$—^{131}I SUBTRACTION STUDIES

Case No.	Size	Position*
(1) +ve scan	2.1×0.8×0.6 cm	Left lower
(2) +ve scan	1.5×0.8 cm	Left lower
(3) +ve scan	2.5×1.5 cm	Left upper
(4) +ve scan	2.0×1.0 cm	Left upper
(5) −ve scan	1.5×1.0 cm	Right upper
(6) −ve scan	1.3×1.0 cm	Right upper

* All the adenomas were situated in conventional positions behind the thyroid gland.

had two different scanning techniques performed.

Five different methods were used (Table 1). Twenty-nine studies were performed with a dual peak ^{75}Se technique.[10] Two hundred and fifty μCi of ^{75}Se were given intravenously following a course of triiodothyronine (T-3) 80 μgs daily for one week. Four sequential scans of the neck were then performed over a two hour period. Eight of these patients had parenteral glucagon[2] (0.5–1.0 mg intramuscularly) prior to commencement of the study. Six patients had sequential $^{99m}Tc\ O_4^-$ studies, one following T-3 thyroidal suppression and five utilizing potassium perchlorate suppression or washout techniques. Four patients had ^{75}Se—^{131}I, and two, ^{75}Se—$^{99m}Tc\ O_4^-$ subtraction studies. Nine patients had $^{99m}Tc\ O_4^-$—^{131}I subtraction studies. Fifty μCi of ^{131}I were given orally 24 hours before the study and 2 mCi of $^{99m}Tc\ O_4^-$ intravenously 15 minutes before commencement of three sequential subtraction scans. In the latter group, all patients had both $^{99m}Tc\ O_4^-$ and ^{131}I thyroid scans as well as the subtraction studies performed. Dual channel facility enabled simultaneous performance of the ^{131}I thyroid scan. The upper head of a Selo digital dual head rectilinear scanner using a high energy collimator was used for all these studies.

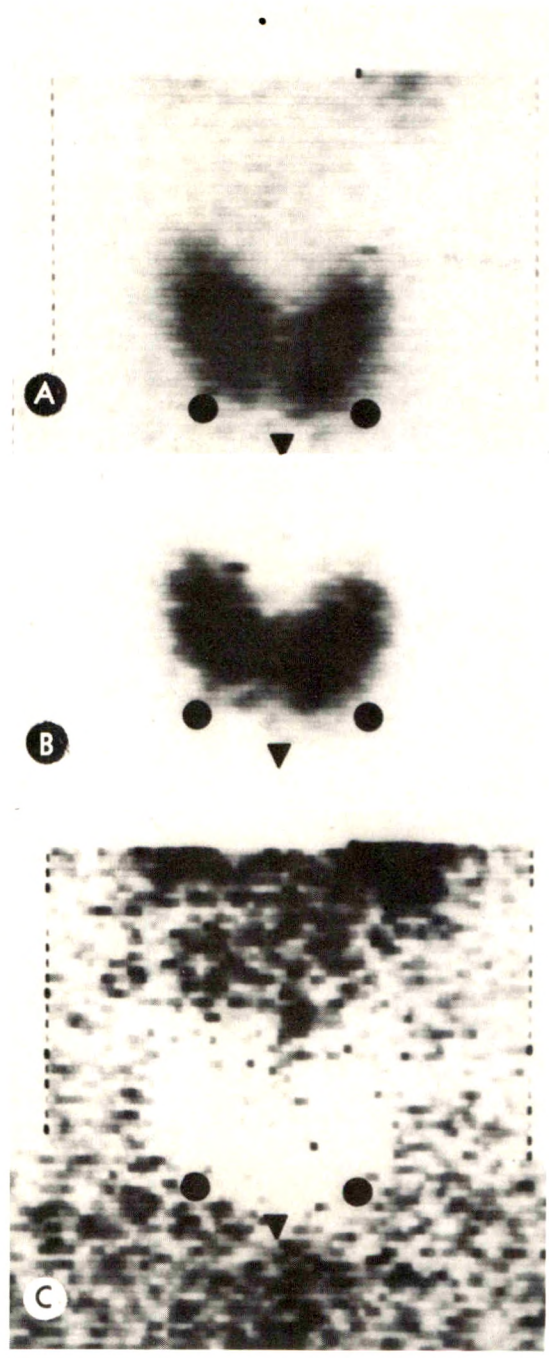

FIG. 1. (*A*) Normal preliminary $^{99m}Tc\ O_4^-$ quantitative thyroid scan. (*B*) Twenty-four hour ^{131}I scan. (*C*) Normal subtraction study.

RESULTS

The results are as shown in Table 1. It can be seen that although the over-all results are acceptable when the confirmed negative scans are included, when reviewing only those patients with proved adenomas, the ^{75}Se techniques are unsatisfactory with a success rate of only 27 percent. The success rate in those studies using the

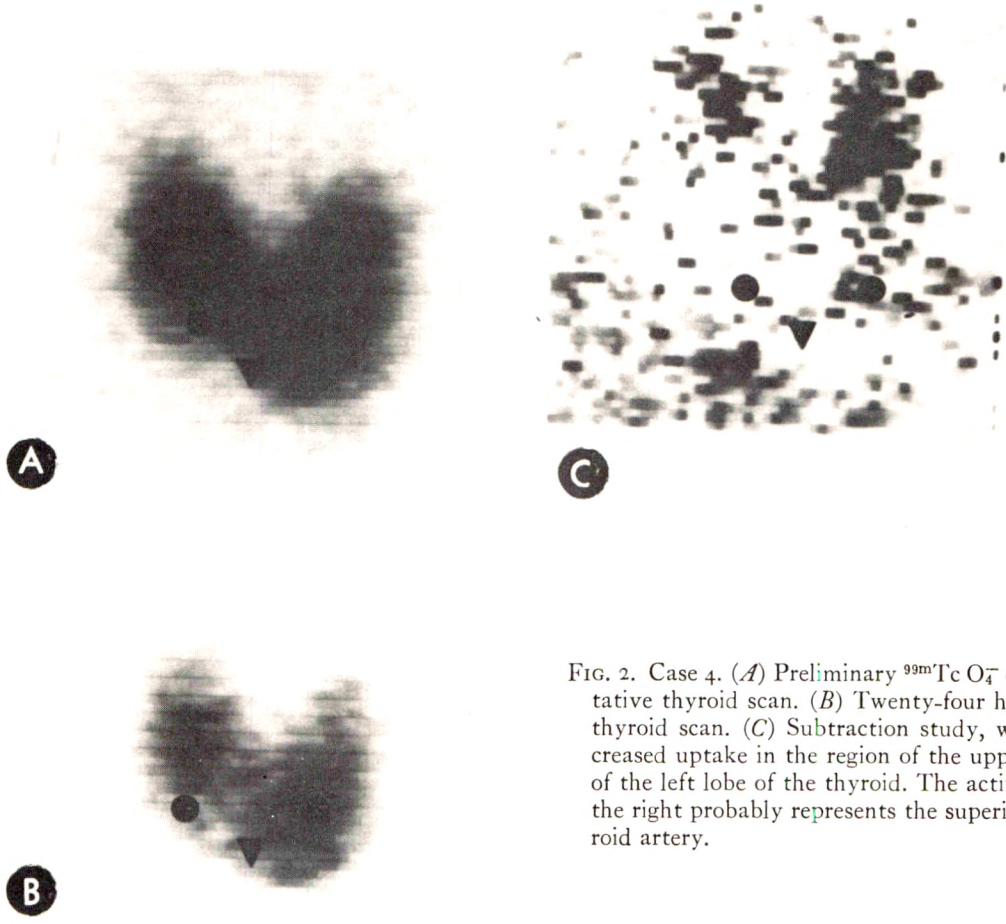

FIG. 2. Case 4. (*A*) Preliminary $^{99m}Tc\ O_4^-$ quantitative thyroid scan. (*B*) Twenty-four hour ^{131}I thyroid scan. (*C*) Subtraction study, with increased uptake in the region of the upper pole of the left lobe of the thyroid. The activity on the right probably represents the superior thyroid artery.

$^{99m}Tc\ O_4^-$—^{131}I subtraction technique was 66 percent. The diagnosis was confirmed in all cases by neck exploration and removal of the adenoma except in one case in which the diagnosis was confirmed at post mortem. The size of the parathyroid adenomas in this group are as indicated in Table II. Cases were considered negative (Table I) on follow-up when the final diagnosis was thought not to be parathyroid adenoma. The follow-up period varied from a minimum of seven months to two years. One negative scan result was confirmed at neck exploration when four normal parathyroid glands were found. There was one false positive sequential $^{99m}Tc\ O_4^-$ study and one false positive ^{75}Se study.

DISCUSSION

The small size and retrothyroid situation of most parathyroid adenomas, coupled with a significant variation in cell type and vascularity, make it unlikely that any simple scanning method will be consistently successful in the localization and possible diagnosis of parathyroid adenomas. It is generally accepted that ^{75}Se studies are of limited value, particularly in small adenomas, and this is confirmed in our experience, where despite such refinements as dual peak utilization, sequential studies, quantitative counting, and glucagon stimulation they have proved disappointing. The fact that ^{75}Se is incorporated within thyroid tissue, which in most instances directly overlies the adenoma, precludes good resolution. Following suppression of the thyroid gland a variable amount of activity within the thyroid may still persist, in addition to significant blood pool activity in the

neck. Subtraction of the thyroid image from the scan by electronic means provides satisfactory resolution of an underlying lesion (Fig. 1–4).

It is of interest that in those studies using ^{75}Se the over-all success rate in the 18 confirmed adenomas (Table 1) was only 27 percent in contrast to the eleven studies using $^{99m}Tc\,O_4^-$ with a success rate in the adenomas of 72 percent. This is probably related to the marked improvement in statistical quality and resolution of scans using this latter isotope. The sequential post suppression $^{99m}Tc\,O_4^-$ studies were not persisted with, as the false negative scan in this group also gave a false positive result due to an autonomous thyroid nodule, a documented cause of ^{75}Se false positive parathyroid scans.[6] The ^{75}Se—^{131}I subtraction studies were also not continued with in view of the excessive contribution of ^{131}I to the ^{75}Se channel and the fact that localization of parathyroid adenomas was found to be more satisfactory with $^{99m}Tc\,O_4^-$ than the ^{75}Se. Two patients with confirmed parathyroid adenomas had positive $^{99m}Tc\,O_4^-$—^{131}I subtraction scans and negative ^{75}Se studies.

The most recent group of patients had $^{99m}Tc\,O_4^-$—^{131}I subtraction studies performed. This technique has several advantages. The statistical quality of the study is far superior to that using ^{75}Se, and the ^{131}I contribution to the $^{99m}Tc\,O_4^-$ channel can be compensated for by giving an appropriate dose of $^{99m}Tc\,O_4^-$ and setting a narrow window for the 140 kev energy peak of this isotope. The problem of a false positive result from a thyroid nodule is also overcome, as the nodule should be apparent on the ^{131}I scan and thus subtracted from the final image. It must be appreciated, however, that the kinetics of these radioisotopes differ as the $^{99m}Tc\,O_4^-$ reflects early thyroidal trapping only, and the 24 hour ^{131}I scan indicates late organified activity. Some nodules may therefore show greater affinity for $^{99m}Tc\,O_4^-$ at the time of the subtraction study. This need not negate the value of the study, as long as late phase sequential scans are performed (Fig. 4*C*).

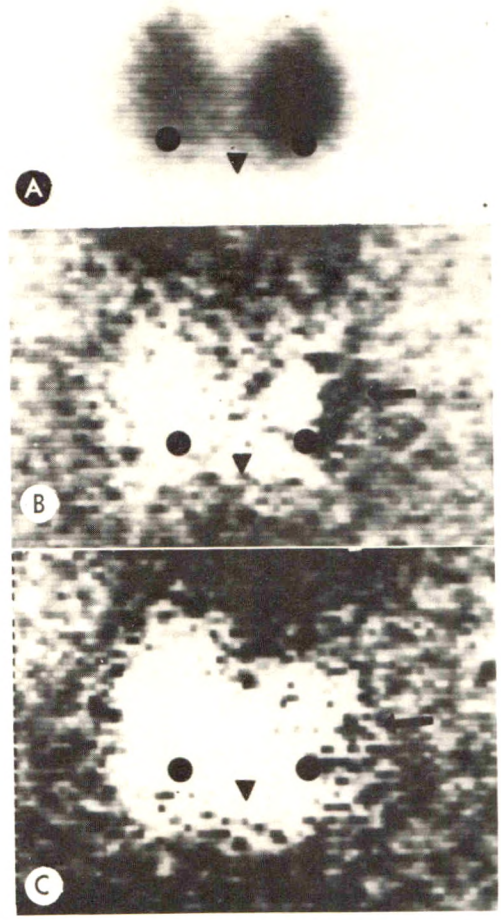

FIG. 3. Case 1. (*A*) Twenty-four hour ^{131}I scan. (*B*) Fifteen minute subtraction study, note increased activity on the left. (*C*) Forty-five minute subtraction study, note persistent increase in this region.

The study can be completed within 24 hours without the need for prior suppression of thyroid activity, and can therefore be performed with short notice. There is no problem in regard to long term radioisotope interference should parathyroid hormone radioimmunoassay or other *in vitro* radioisotopic tests be required. This factor deterred us from using ^{125}I for subtraction studies. All patients had preliminary $^{99m}Tc\,O_4^-$ quantitative thyroid scans[1] in order to determine their suitability for subtraction scanning. Thus, suppressed uptake due to recent roentgenographic investiga-

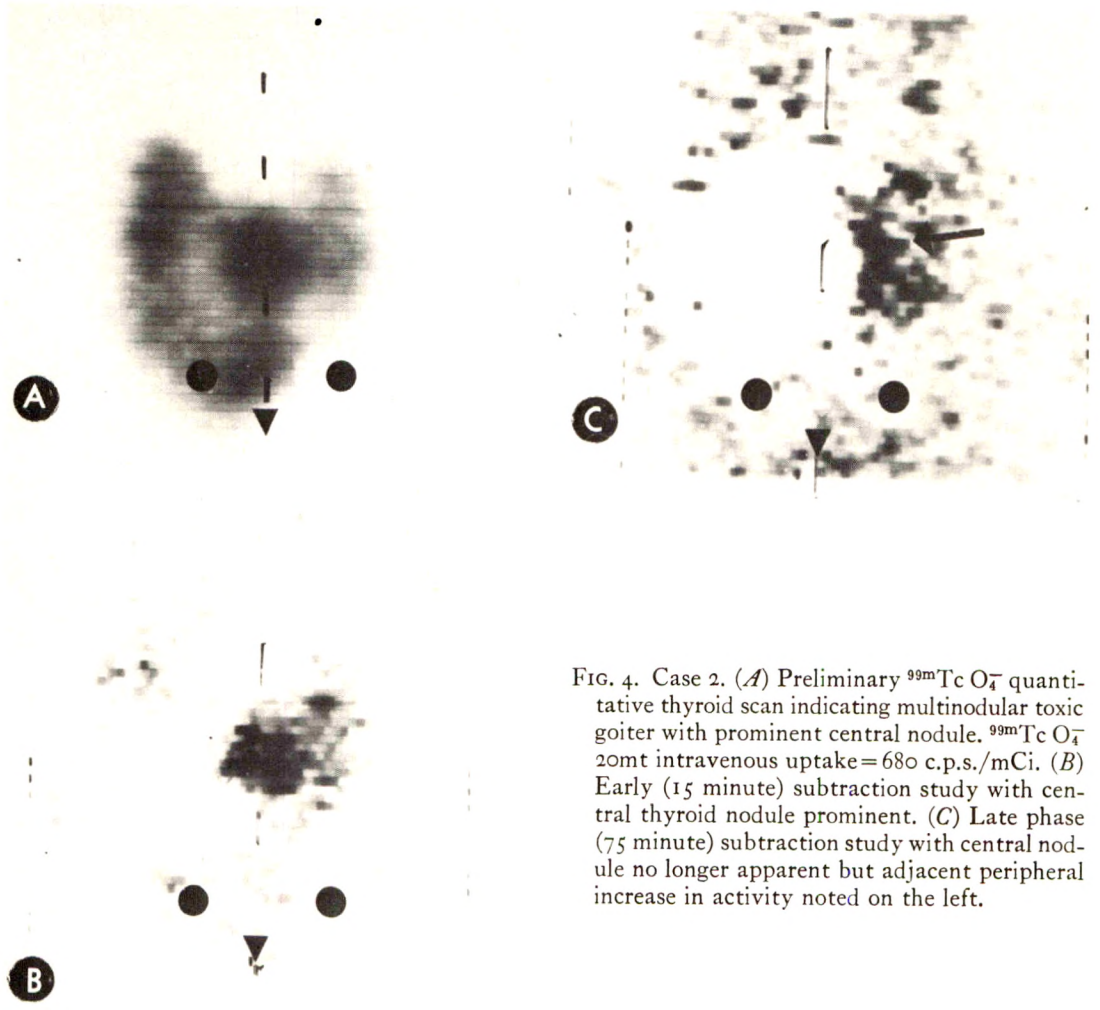

FIG. 4. Case 2. (*A*) Preliminary $^{99m}Tc\ O_4^-$ quantitative thyroid scan indicating multinodular toxic goiter with prominent central nodule. $^{99m}Tc\ O_4^-$ 20mt intravenous uptake = 680 c.p.s./mCi. (*B*) Early (15 minute) subtraction study with central thyroid nodule prominent. (*C*) Late phase (75 minute) subtraction study with central nodule no longer apparent but adjacent peripheral increase in activity noted on the left.

tions may be noted, or the presence of a multinodular or toxic goiter detected. The former would preclude a subtraction study, and knowledge of the latter conditions would be helpful in the subsequent performance of the scans. The setting up procedure for the subtraction study is not easy and it would be reasonable to anticipate a higher success rate as more experience is gained with this technique. The main limitation of this method is that it is unlikely to be of value in those parathyroid tumors situated well within the mediastinum. This did not apply to any patient in this series.

It is not clear why some parathyroid adenomas show an affinity for $^{99m}Tc\ O_4^-$. This may be attributable to $^{99m}Tc\ O_4^-$ incorporation within the tumor cells. It is more likely, however, that it reflects, at least in part, the vascularity of the gland.

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I wish to thank the physicians and surgeons at the Repatriation General, Austin, and Prince Henry's Hospitals for referring their patients for parathyroid studies, and nucleographers; Mrs. R. Martin, Mrs. D. Kent, Miss J. Shaw and Mr. R. Matchan for assisting in the performance of these studies. Thanks are also due to Miss C. Dwyer for assisting in the preparation of

the manuscript, and to the Repatriation Department, for allowing publication of this paper.

REFERENCES

1. Arkles, L. B. Quantitative thyroid scanning: reliable thyroid function test. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *121*, 705–713.
2. Ashkar, F. S., Naya, J. L., and Smith, E. M. Parathyroid scanning with ^{75}Se—selenomethionine and glucagon stimulation. *J. Nuclear Med.*, 1971, *12*, 751–753.
3. Di Giulio, W., and Morales, J. O. Value of selenomethionine ^{75}Se scan in preoperative localization of parathyroid adenomas. *J.A.M.A.*, 1969, *209*, 1873–1880.
4. McGeown, M. G., Bell, T. K., Soyannwo, M. A. O., Fenton, S. A., and Oreopoulus, D. Parathyroid scanning in human with selenomethionine—^{75}Se. *Brit. J. Radiol.*, 1968, *41*, 300–306.
5. O'Riordan, H. H., Kendall, B. E., and Woodhead, J. A. Preoperative localization of parathyroid tumours. *Lancet*, 1971, *2*, 1172–1175.
6. Potchen, E. J., and Studer, R. Preoperative localization of parathyroid tumours. *Progr. Clin. Cancer*, 1970, *4*, 160–174.
7. Powell, D., Shimkin, P. M., Doppman, J. L., Wells, S., Aurbach, G. D., Marx, S. J., Ketcham, A. S., and Potts, J. T. Primary hyperparathyroidism: preoperative tumor localization and differentiation between adenoma and hyperplasia. *New England J. Med.*, 1972, *286*, 1169–1175.
8. Reiss, E., and Canterbury, J. M. Primary hyperparathyroidism: application of radioimmunoassay to differentiation of adenoma and hyperplasia and to preoperative localization of hyperfunctioning parathyroid glands. *New England J. Med.*, 1969, *280*, 1381–1385.
9. Samuels, B. I., Dowdy, A. H., and Lecky, J. W. Parathyroid thermography. *Radiology*, 1972, *104*, 575–578.
10. Steven, L. W., Andrews, J. T., and Arkles, L. B. Optimum measurement of selenium-75 for pancreas scanning. *Australian Radiol.*, 1969, *13*, 418–420.

RADIONUCLIDE STUDIES IN BRONCHOGENIC CARCINOMA OF THE HILUM*

SCINTIGRAPHY AND TOMOGRAPHY: THEIR COMPLEMENTARY FEATURES

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ABSTRACT:

Thirty-eight cases with direct or indirect signs of hilar masses were investigated by roentgenologic, radioisotopic and surgical methods. Reasonable correlation between tomography and scintigraphy was confirmed, substantiating their complementary nature. Bronchogenic carcinoma of the central airways was most frequent among the hilar masses. Masses as well as other involvement of the bronchovascular structures of the hilum on conventional tomography were confirmed by the gallium-67 scan, and inhalation and perfusion scintigraphy. Some cases which simulated bronchogenic carcinoma were presented. Hilar masses without destruction of the bronchovascular structures showed normal inhalation and perfusion scintigrams with positive gallium-67 accumulation. These lesions were metastatic cancer, malignant lymphoma, and sarcoidosis. If these diseases involve the airways and the vessels of the hilum, differentiation from bronchogenic carcinoma may naturally be difficult.

ROUTINE chest films in posteroanterior and lateral projections are useful for disclosing abnormalities in the thorax, but the mediastinal silhouette in the roentgenograms often obscures lesions at the hilum as well as the central pulmonary regions. Therefore, bronchogenic carcinoma which arises most often in the lobar and segmental bronchi[6] is rather difficult to detect in its early stage. Much effort has been devoted to finding characteristic signs of bronchial carcinoma of the hilum.[23,24] Hilar enlargement and secondary changes such as oligemia and atelectasis of the peripheral lung fields are most important,[24] and tomography is a most reliable means of confirming these signs among noninvasive roentgenologic methods.[23,40] In addition to this, lung scintigraphy is also useful for studying the hilar region. Gallium-67 scintigraphy is accepted as valuable for detecting hilar as well as mediastinal masses.[17] Perfusion scintigraphy for the assessment of regional pulmonary blood flow is also useful for screening for bronchogenic carcinoma of the hilum.[5] Inhalation scintigraphy with radioaerosol has proved to be a convenient procedure for determining partial obstruction of the central airways.[33] Since there is no specific picture of bronchogenic carcinoma with any type of scintigraphy alone,[5,17,33] the combined use of these three isotope methods enhances the detectability of bronchogenic carcinoma of the hilum as tomography simultaneously reveals the hilar masses, oligemia, and bronchial stenosis. In this report, usefulness of this combination of scintigraphy and tomography is presented.

MATERIAL AND METHOD

Thirty-eight patients who were suspected of having hilar masses on routine chest roentgenograms were selected for the present investigation. Hilar enlargement as well as secondary changes due to the hilar

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TABLE I

SUMMARY OF FINDINGS IN 38 CASES SUSPECTED OF HAVING HILAR MASSES

Diagnosis	Cases	Tomography			Scintigraphy		
		Masses	Stenosis	Oligemia	Ga-67	Inhalation	Perfusion
Bronchogenic carcinoma*	18	+	+	+	+	+	+
Cases simulating bronchogenic carcinoma							
Bronchial adenoma	1	+	+	+	−	+	+
Breast cancer*	1	+	+	+	+	+	+
Bronchial tuberculosis							
chronic*	1	−	+	+	±	+	+
active	1	+	+	+	+	+	+
Unresolved pneumonia	1	+	+	+	+	+	+
Conglomerated silicosis	1	+	+	+	+	+	+
Metastasis	3	+	−	−	+	−	−
Lymphoma,* sarcoidosis	8	+	−	−	+	−	−
Miscellaneous							
Remote infectious	2	+	−	−	−	−	−
Embolism	1	−	−	−	−	−	+

* Cases presented in Figures 1 to 5.
− = Negative finding; + = Definite positive finding; ± = Slight uptake of gallium-67.

masses, such as atelectasis, infiltrative opacities, and oligemia were carefully examined. These findings were confirmed by tomograms in anteroposterior and lateral projections. Inhalation scintigraphy was carried out according to the conventional method of Taplin *et al.*[33] Ten mCi of technetium-99m albumin or stannous colloid suspended in 2 ml of normal saline were prepared as a radioaerosol by a highly efficient ultrasonic nebulizer.* This aerosol could be completely inhaled in about one minute under the maximum output of this nebulizer. The patient was advised to breathe as quietly as possible at normal tidal ventilation. A scintillation camera† was more desirable than a scanner for the inhalation scintigraphy, because the so-called hot spot of the aerosol would disappear as a result of ciliary activity, if more time were spent for obtaining multiple views.[26] After inhalation scintigraphy, 200 μCi of I-131 macroaggregated albumin (MAA) were administered intravenously for the perfusion scintigraphy. When Tc-99m MAA was available, this scintigraphy was performed one day after the inhalation scintigraphy. The gallium-67 scintigraphy was done three days after intravenous administration of 2 mCi of gallium-67 citrate. Radiospirometry with xenon-133 was performed in some cases. The findings obtained from these three or four studies were correlated with those of the tomography. When masses were disclosed in the central airways by any of these noninvasive methods, both bronchography and bronchoscopy were performed for the final diagnosis of bronchogenic carcinoma. If the central airways were considered to be intact in spite of the hilar masses, other surgical procedures such as mediastinoscopy were preferred.

* Ultrasonic Nebulizer NB 108 LKB Medical AB of Sweden.
† Pho gamma III, Nuclear Chicago, Illinois, U.S.A.

RESULTS

The results of the examinations of 38 cases are summarized in Table 1. These cases can be classified into four groups according to the findings of tomography and scintigraphy. The first group, consisting of 18 cases of bronchogenic carcinoma proved histopathologically, showed definite abnor-

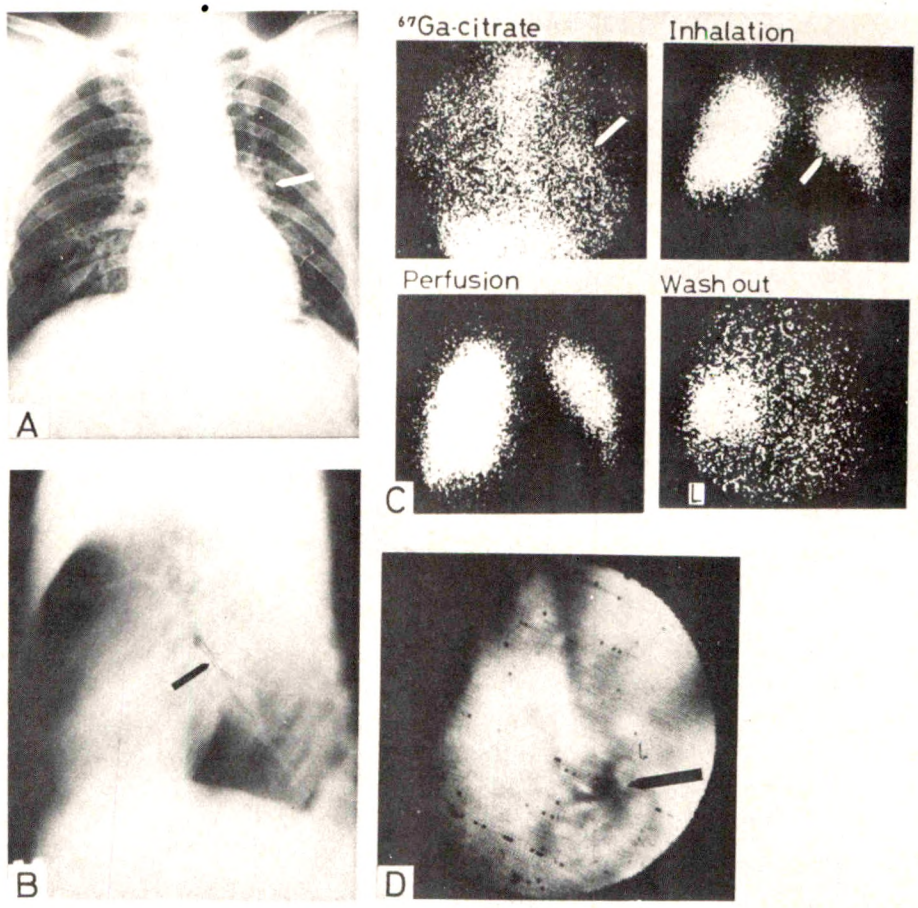

FIG. 1. Bronchogenic carcinoma in a 47 year old man. A posteroanterior plain film and a left lateral tomogram (*A* and *B*) show hilar masses, bronchial stenosis, and oligemia of the left lung. Four scintigrams (*C*) confirm those findings. Bronchoscopic examination reveals severe stenosis of the lower lobe bronchus of the left lung (*D*).

mal findings both on tomography and scintigraphy simultaneously with hilar masses and involvement of the airways and vessels. The second group contained six cases that simulated bronchogenic carcinoma. The third group consisted of eleven cases with either metastatic carcinoma to the hilum, malignant lymphoma, or sarcoidosis. This group was characterized by hilar masses without involvement of the airways and hilar vessels. Other cases were classified into the fourth group.

REPORT OF CASES

Typical examples of each group are presented in the following five cases (Fig. 1 to Fig. 5).

(1) The first group: Bronchogenic carcinoma (18 cases)

CASE 1. A 47 year old male was admitted to this hospital because of persistent cough with occasional bloody sputum. A posteroanterior chest roentgenogram showed slight left hilar enlargement (Fig. 1*A*). A left lateral tomogram revealed hilar masses around the lower lobe bronchus (Fig. 1*B*). Stenosis of the lower lobe bronchus and moderate oligemia of the lower lung field were also recognized. All four scintigrams showed abnormal findings (Fig. 1*C*). Gallium-67 accumulated in the left hilum. The inhalation scintigram showed a clear hot spot of the aerosol at the left hilum with an inhalation defect in the left lower lung. A perfusion defect was also observed in the perfusion scintigram. The washout of xenon-133 from the same area

was markedly delayed. These scintigrams indicate hilar masses causing bronchial stenosis and oligemia, which suggest the probability of bronchogenic carcinoma. Bronchoscopic examination disclosed severe stenosis of the left lower lobe bronchus (Fig. 1*D*). The histopathological diagnosis was oat cell carcinoma.

Case II. A 57 year old male had noticed a hard mass in the left supraclavicular region about one month before admission. Undifferentiated cell carcinoma probably of pulmonary origin was proved by biopsy of this mass. A posteroanterior chest roentgenogram showed hilar enlargement on both sides with reticulo-nodular shadows in the left lung (Fig. 2*A*). An anteroposterior tomogram disclosed right hilar masses which nearly obstructed the lower lobe bronchus, but the central airways of the left lung were normal in spite of masses around them (Fig. 2*B*). These roentgenograms were considered to indicate bronchogenic carcinoma on the right and metastatic extension to the left. Gallium-67 accumulated in the right hilum, and a diffuse uptake was seen in the left lung (Fig. 2*C*), which was consistent with the roentgenographic findings. Inhalation, perfusion, and xenon-133 scintigraphy showed findings similar to those observed in Case I (Fig. 2*C*). The inhalation scintigram showed a hot spot in the right hilum with an inhalation defect below. A perfusion defect and marked delay of the washout of xenon-133 were also observed in the lower lung field. Severe stenosis of the middle lobe bronchus and almost complete obstruction of the lower lobe bronchus were shown by bronchography (Fig. 2*D*). Bronchoscopic examination and biopsy revealed an undifferentiated cell carcinoma of the right lung, but the central airways of the left lung were normal. In summary, the hilar masses on the right caused bronchial stenosis and oligemia, which were confirmed by both tomography and scintigraphy. In contrast, the hilar masses and reticulo-nodular shadows on the left were due to metastatic extension without definite involvement of the airways and vessels of the hilum. This should be compared with Case V of the third group.

(2) The second group: Cases simulating bronchogenic carcinoma (six cases)

Case III. A 54 year old female had suffered from a dry cough for five months before admission. She had undergone a left mastectomy five years before for cancer. A plain chest roentgenogram showed hilar enlargement and reticular shadows in both lungs (Fig. 3*A*). Complete obstruction of the middle lobe bronchus and partial obstruction of the lower lobe bronchus were observed in a right lateral tomogram (Fig. 3*B*). Partial obstruction of the lower lobe bronchus and moderate narrowing of the left pulmonary artery were shown by the left lateral tomogram (Fig. 3*B*). Gallium-67 scintigraphy revealed accumulation in both hilar regions and in the left lung (Fig. 3*C*). The inhalation and perfusion scintigrams showed bilateral bronchial stenosis and perfusion decrease in the lower portion of each lung (Fig. 3*C*). Bronchoscopic examination proved complete obstruction of the middle lobe bronchus (Fig. 3*D*) and adenocarcinoma was considered certain. But other airways, such as the lower lobe bronchus of each lung were stenotic without the appearance of malignancy. The findings obtained from tomography and scintigraphy were quite similar to those of Case II. But this case was classified metastatic breast cancer with lymphangitic spread on the basis of the subsequent clinical course.

Case IV. A 24 year old female was referred to this hospital because of moderate stridor. Plain chest roentgenograms at total lung capacity (Fig. 4*A*) and at residual volume (Fig. 4*B*) disclosed air trapping in the left lung. Marked oligemia of the left lung was also noted. An anteroposterior tomogram showed severe stenosis of the left main bronchus but no mass lesion was clearly shown (Fig. 4*C*). Slight uptake of gallium-67 was seen in the mediastinum as well as in the left lung (Fig. 4*D*). A clear hot spot of the aerosol was observed in the left main bronchus in the inhalation scintigram and severe perfusion decrease of the left lung was also recognized (Fig. 4*D*). Severe but benign tuberculous stenosis of the left main bronchus was confirmed by bronchoscopy.

(3) The third group: Metastatic carcinoma, malignant lymphoma and sarcoidosis (eleven cases)

Case V. A 49 year old male was admitted to this hospital because of a neck tumor, which was proved reticulum cell sarcoma. During radiation therapy abnormal hilar shadows appeared (Fig. 5*A*). An anteroposterior tomo-

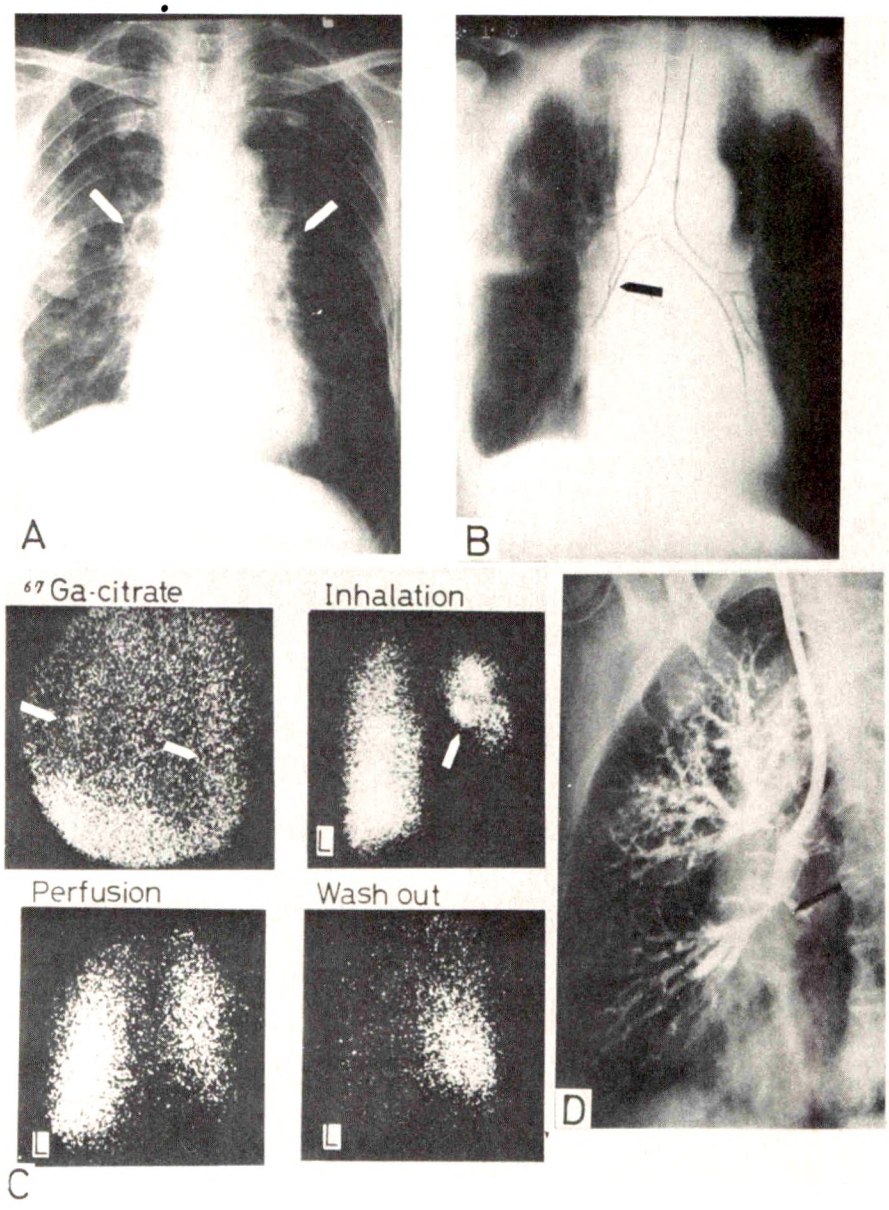

Fig. 2. A 57 year old man with bronchogenic carcinoma which originated in the right hilum and spread to the left lung. A posteroanterior plain film (*A*) and an anteroposterior tomogram (*B*) show bilateral hilar masses which are also confirmed by the gallium-67 scintigraphy (*C*), but the airways and vessels of the left lung are normal by inhalation, perfusion, and xenon-133 scintigraphy (*C*). (*D*) is the bronchogram of the right lung which shows severe stenosis of the middle and lower lobe bronchi.

gram revealed hilar masses on the left, but the main, lobar, and segmental bronchi were all normal (Fig. 5*B*). Inhalation and perfusion scintigrams were normal, although higher accumulation of gallium-67 was observed in the left hilum (Fig. 5*C*). Similar observations were also seen in sarcoidosis without definite pulmonary involvement.

(4) Other cases that did not fall into these three groups. (three cases)

Three cases that were suspected of hav-

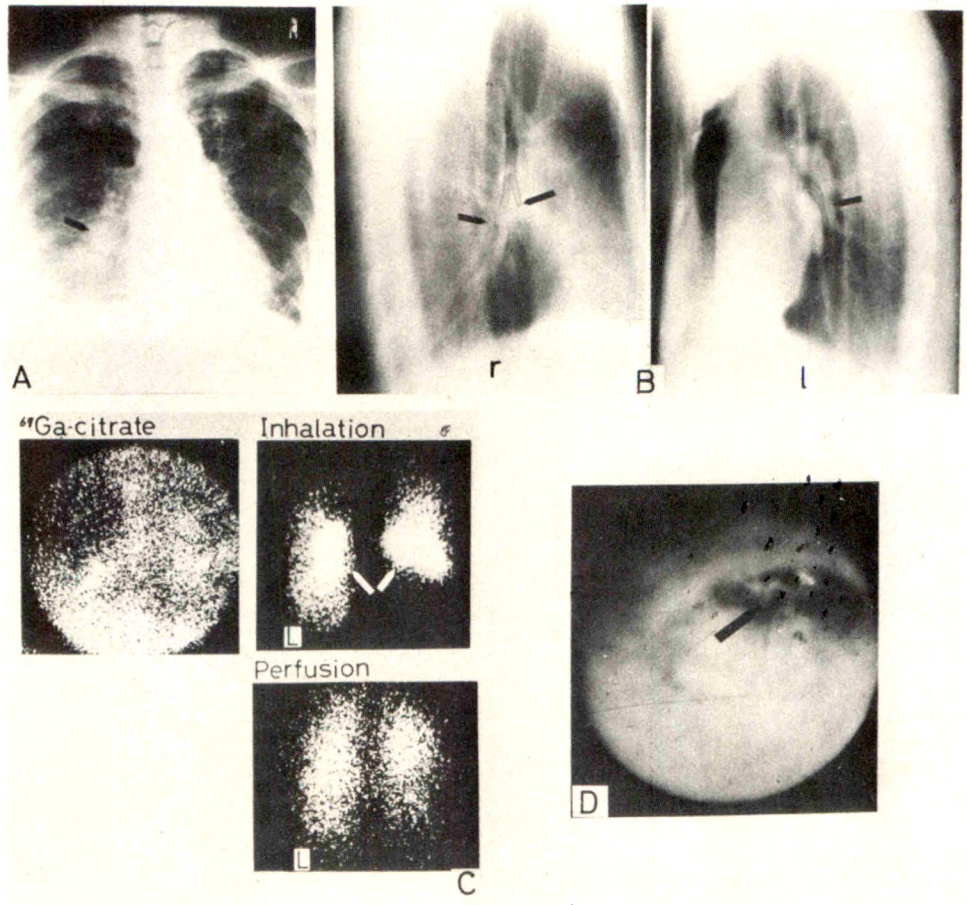

Fig. 3. A 54 year old female with metastatic breast cancer simulating bronchogenic carcinoma. A postero-anterior plain film (*A*), lateral tomograms of right and left sides (*B*), and three scintigrams (*C*) show bilateral hilar masses which cause bronchial stenosis or obstruction and oligemia. (*D*) shows the obstructed right middle lobe bronchus.

ing hilar masses were patients with lymphadenopathy of the hilum and pulmonary embolism. The precise findings of each case are summarized in Table 1.

DISCUSSION

In considering mediastinal masses, the site in the mediastinum is the first important diagnostic clue.[14,18] Among these masses, those of the mid-mediastinum present an additional problem: that is, whether or not they involve the hilar structures such as the bronchi and vessels. Diagnostic procedures listed in Table II have been used to differentiate bronchial carcinoma from other mid-mediastinal masses.[18] But the usefulness of scintigraphy as a diagnostic method in bronchial carcinoma has not yet been evaluated clearly. Therefore, an attempt to correlate scintigraphy with other established methods such as tomography should be undertaken, to establish the complementary features of these two procedures.

Bronchogenic carcinoma is characterized by hilar masses and changes secondary to them such as atelectasis or oligemia shown on routine chest roentgenograms.[23,24] These signs can be well confirmed by tomography.[18,23,40] Tomography is, as is shown in this report, an excellent noninvasive method which reveals clearly the hilar

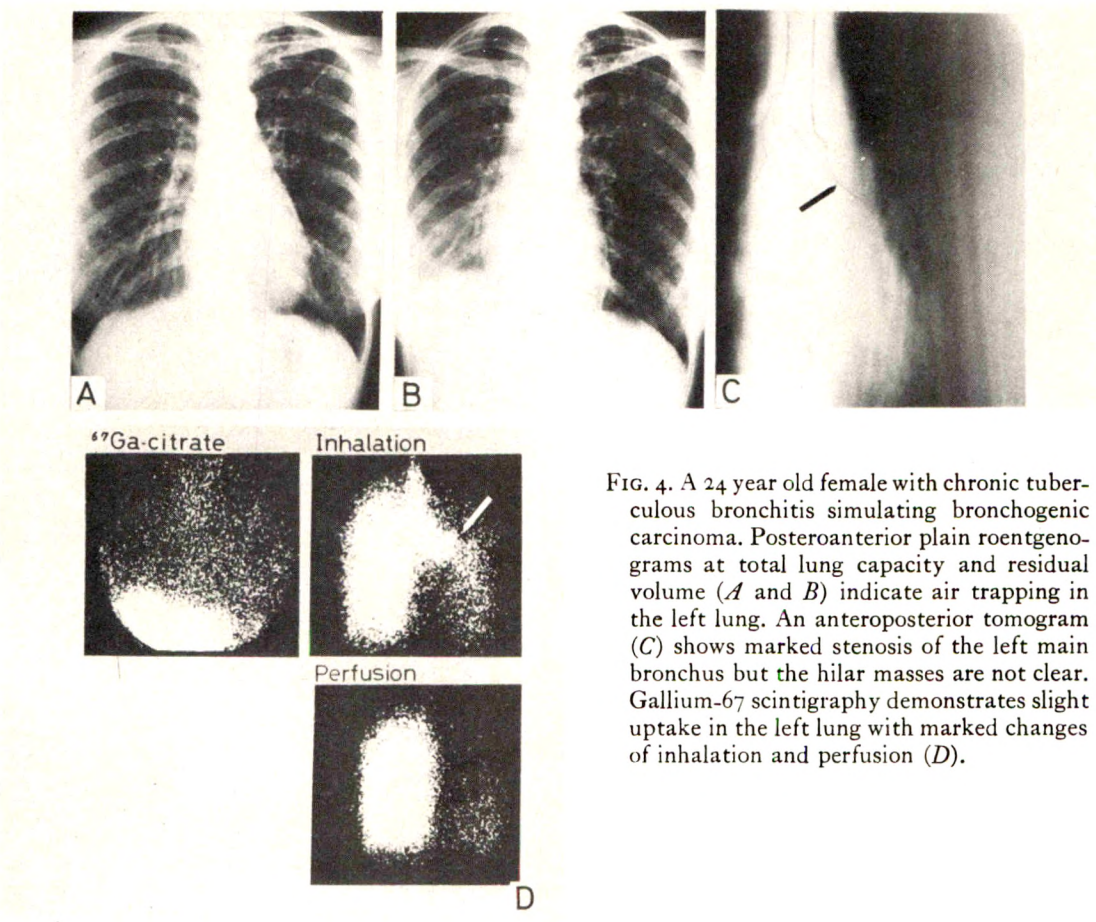

FIG. 4. A 24 year old female with chronic tuberculous bronchitis simulating bronchogenic carcinoma. Posteroanterior plain roentgenograms at total lung capacity and residual volume (*A* and *B*) indicate air trapping in the left lung. An anteroposterior tomogram (*C*) shows marked stenosis of the left main bronchus but the hilar masses are not clear. Gallium-67 scintigraphy demonstrates slight uptake in the left lung with marked changes of inhalation and perfusion (*D*).

masses, complete or partial obstruction of the airways, and oligemia, simultaneously.

Lung scintigraphy is also valuable for examining the hilar region. Gallium-67 scintigraphy is a useful tumor seeking method[4] and its sensitivity in bronchogenic carcinoma of the hilar region is a well known fact.[3,16,17] Accumulation of gallium-67 in inflammatory lesions may limit its specificity for malignancy.[19] However, its merit in detecting hilar masses which are often overlooked on the roentgenograms makes it useful. Perfusion scintigraphy with radioactive particles such as I-131 MAA or Tc-99m MAA has been accepted as a valuable method for estimating regional pulmonary blood flow since the reports of Taplin *et al.*[31] and Wagner and associates.[38] Although comprehensive studies of perfusion abnormalities in various chest diseases have been reported,[9,19,22] no feature specific for bronchial carcinoma has been identified. For example, benign disease such as pulmonary emphysema shows a perfusion defect similar to that which occurs in bronchogenic carcinoma.[5,22] But there is one specific feature of perfusion scintigraphy in bronchogenic carcinoma of the hilum: that is, the area of the perfusion defect is often greater than expected from the roentgenograms.[5,19,27,39] In addition, lobar and segmental oligemia is not always easy to detect on the plain films, and therefore perfusion scintigraphy is useful for screening bronchial carcinoma of the hilum.[5]

Since inhalation scintigraphy with radioaerosol was introduced by Taplin and Poe,[32] and Pircher and associates,[21] it has be-

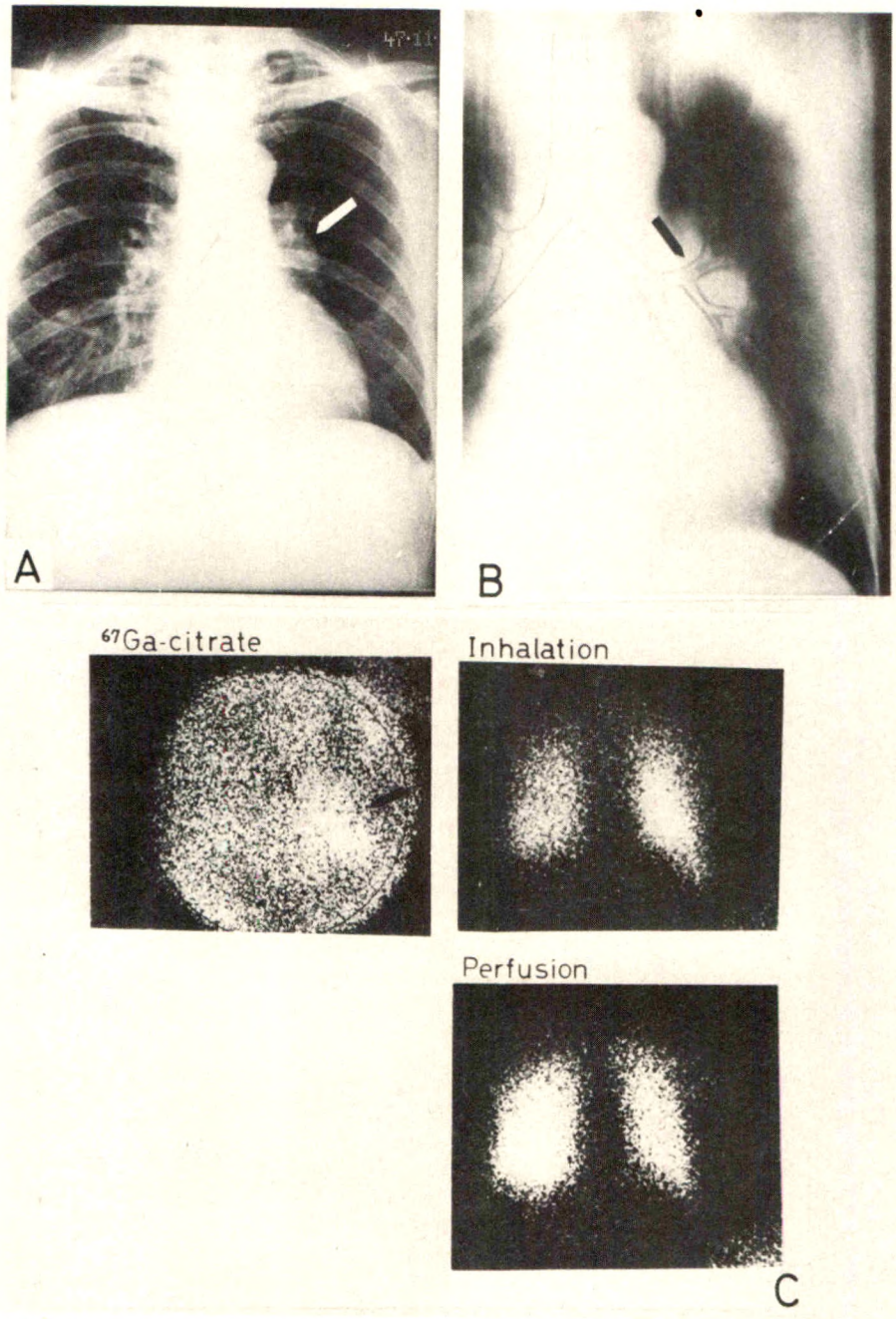

Fig. 5. Reticulum cell sarcoma of the left hilum in a 49 year old man. A posteroanterior chest film (*A*) and an anteroposterior tomogram (*B*) show hilar masses on the left but the airways and vessels of the left lung are normal, as is confirmed by the scintigrams (*C*).

come possible to investigate the airways and pulmonary arteries simultaneously by combining it with perfusion scintigraphy.[10,32,33,35] Aerosol deposition in the human airways has been investigated in various clinical fields other than nuclear medicine, such as aerosol therapy in chest disease[1,20] and study of the pathogenesis of bronchial carcinoma.[28] Theoretical and experimental considerations in aerosol depo-

TABLE II

DIAGNOSTIC PROCEDURES IN HILAR MASSES

Roentgenographic Studies
Routine x-ray
Tomography
Bronchography
Angiography
Surgical Procedures
Bronchoscopy
Scalene lymph node biopsy
Mediastinoscopy
Biopsy of superficial lymph nodes
Thoracotomy
Others
Skin test for specific granuloma
Sputum studies

sition in human airways have been elucidated,[2,30,36] and conventional inhalation scintigraphy should be reviewed from this view point. The aerosol used for inhalation scintigraphy is wet, although dry aerosols have become available experimentally.[35] It should be noted that the physical properties of these wet and dry aerosols may be different in the respiratory tract,[20] but some common features exist. Major mechanisms of the aerosol deposition in the human airways are a combination of impaction, sedimentation and diffusion.[30] Since our ultrasonic nebulizer is found to generate aerosol particles of about 2 microns in median diameter, impaction and sedimentation may be dominant in the central and peripheral airways, respectively.[11,30] Impaction becomes more important when partial obstruction, carinal widening, and abrupt change of flow direction exist,[12] causing excessive deposition of the aerosol at that point. This excessive deposition may be visualized as a hot spot in the inhalation scintigram. Bronchial stenosis[34] may produce a similar picture. This hot spot, however, is also observed in chronic obstructive pulmonary disease which also causes bronchial stenosis,[10] and it is impossible to differentiate bronchial carcinoma from chronic obstructive pulmonary disease by inhalation scintigraphy alone.

In summary, combined use of gallium-67 scintigraphy and inhalation and perfusion scintigraphy, is valuable for disclosing the nature of hilar masses, because each of these methods alone has proved nonspecific for bronchogenic carcinoma.

The correlation of these scintigraphic methods with tomography is good in bronchogenic carcinoma as is shown in Table I. Therefore it is reasonable to consider that the procedures, tomography and scintigraphy, are complementary in the diagnosis of bronchial carcinoma. The high accumulation of gallium-67 in our series of bronchogenic carcinoma may be due to the advanced nature of the disease.[16] When metastatic carcinoma compresses or erodes the central airways, the findings of both tomography and scintigraphy are quite similar to those of bronchogenic carcinoma (Case III). The same result may be observed in lesions other than bronchial carcinoma as reported by others.[15,25,37] On the other hand, some metastatic cancer with or without lymphangitic spread,[7] malignant lymphoma,[8,29] and sarcoidosis[8,9] do not greatly destroy the bronchovascular systems, if not advanced. Thus, inhalation and perfusion scintigraphic studies were almost normal as in the left lung of Cases II, and V, though higher uptake of gallium-67 was observed in both cases. These findings were considered to be specific for this group (group 3), which differed greatly from bronchogenic carcinoma.

It is reasonable to consider that the probable diagnosis of bronchogenic carcinoma can be made according to the present context, but bronchoscopic examination is still necessary for the final diagnosis. Inhalation and gallium-67 scintigraphy play an important role in determining the indication for this examination. When the central airways are considered normal by tomography and scintigraphy, bronchoscopic examination is not always necessary. Other surgical procedures such as mediastinoscopy should be performed to determine the nature of the hilar masses. Before final disposition, further studies should be required especially for early bronchial carcinoma

involving the hilum, because all three scintigraphic methods may not always show abnormal findings in such a case.

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REFERENCES

1. Allan, D. Humidification and mist therapy. In: International Anesthesiology Clinics. Edited by D. Allan. Little, Brown & Company, Boston, 1970, *8*, p. 3.
2. Beekmans, J. M. Deposition of aerosols in respiratory tract: 1. Mathematical analysis and comparison with experimental data. *Canad. J. Physiol. Pharmac.*, 1965, *43*, 157–172.
3. Deland, F. H., Sauerburn, B. J. L., Boyd, C., Wilkinson, R. H., Jr., Friedman, B. I., Moinuddin, M., Preston, D. F., and Kniseley, R. M. 67-Ga citrate imaging in untreated primary lung cancer: preliminary report of cooperative group. *J. Nuclear Med.*, 1974, *15*, 408–411.
4. Edwards, C. L., and Hayes, R. L. Tumor scanning with 67-Ga citrate. *J. Nuclear Med.*, 1969, *10*, 103–105.
5. Fletcher, J. W., James, A. E., and Holman, B. L. Regional lung function in cancer. In: Progress in Nuclear Medicine. Regional Pulmonary Function in Health and Disease. Edited by B. L. Holman and J. F. Lindeman. Karger, Basel and University Park Press, Baltimore 1973, *3*, pp. 135–148.
6. Garland, H. L., Beier, R. L., Coulson, W., Heald, J. H., and Stein, R. L. Apparent site of origin of carcinomas of lung. *Radiology*, 1962, *78*, 1–11.
7. Goldsmith, H. S., Bailey, H. D., Callahan, E. L., and Beattie, E. J., Jr. Pulmonary metastasis from breast carcinoma. *A.M.A. Arch. Surg.*, 1967, *94*, 483–488.
8. Heitzman, E. R. Sarcoidosis and Hodgkin's disease. In: The Lung. Radiologic-Pathologic Correlations. C. V. Mosby Company, St. Louis, 1973, pp. 258–275 and 313–329.
9. Isawa, T. Studies on distribution of pulmonary arterial blood flow in lungs by radioisotope scanning. *Sc. Rep. Res. Inst. Tohoku Univ. (Ser. C)*, 1966, *13*, 123–185.
10. Isawa, T., Wasserman, K., and Taplin, G. V. Lung scintigraphy and pulmonary function studies in obstructive airway disease. *Am. Rev. Resp. Dis.*, 1970, *102*, 161–172.
11. Itoh, H. Ishii, Y., Mori, T., Hamamoto, K., Mukai, T., Torizuka, K., and Takahashi, K. Aerosol deposition in the respiratory tract. In: Proceedings of the First World Federation of Nuclear Medicine and Biology, 1974, pp. 1101–1103.
12. Itoh, H. Unpublished data.
13. James, E. A., White, R., Cooper, M., and Wagner, H. N., Jr. Pretreatment diagnostic evaluation with reference to pulmonary scans in cancer. *J. Thoracic & Cardiovasc. Surg.*, 1971, *61*, 530–540.
14. Joseph, W. L., Murray, J. F., and Mudler, D. G. Mediastinal tumors—problems in diagnosis and treatment. *Dis. Chest*, 1966, *50*, 150–160.
15. Kalbian, V. V. Bronchial involvement on pulmonary sarcoidosis. *Thorax*, 1957, *12*, 18–23.
16. Kinoshita, F., Ushio, T., Maekawa, A., Arita, R., and Kubo, A. Scintiscanning of pulmonary diseases with 67-Ga citrate. *J. Nuclear Med.*, 1974, *15*, 227–233.
17. Langhammer, H., Glaubitt, G., Grabe, S. F., Hampe, J. F., Haubold, U., Hör, G., Kaul, A., Loeppe, P., Koppenhagen, J., Roedler, H. D., and Vander Schoot, J. B. 67-Ga for tumor scanning. *J. Nuclear Med.*, 1972, *13*, 25–30.
18. Lyons, H. A., Calvy, G. L., and Sammons, B. P. Diagnosis and classification of mediastinal masses. 1. Study of 782 cases. *Ann. Int. Med.*, 1959, *51*, 879–932.
19. Maynard, C. D., and Dowan, J. Role of scan in bronchogenic carcinoma. *Seminars Nuclear Med.*, 1971, *1*, 195–205.
20. Muir, D. C. F. Deposition and clearance of inhaled particles and physical properties of therapeutic aerosols. In: Clincial Aspects of Inhaled Particles. Edited by D. C. F. Muir. William Heinemann Ltd., London, 1972, pp. 1–20 and 173–185.
21. Pircher, F. J., Temple, J. R., Kirsch, W. J., and Reeves, R. J. Distribution of pulmonary ventilation determined by radioisotope scanning: preliminary report. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *1*, 185–194.
22. Quinn, J. L. Perfusion scanning in chronic obstructive lung disease. *Seminars Nuclear Med.*, 1971, *1*, 185–194.
23. Rigler, L. G. Roentgen signs of carcinoma of lung. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1955, *74*, 415–428.
24. Rigler, L. G. Earliest roentgenographic signs of carcinoma of lung. *J.A.M.A.*, 1966, *195*, 655–657.
25. Samuels, M. L., Howe, C. D., Dodd, G. D., Jr., Fuller, L. M., Shullenberger, C. C., and Leary, W. L. Endobronchial malignant lymphoma: report of 5 cases in adults. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *85*, 87–95.
26. Sanchis, J., Dolovich, M., Chalmers, R., and

Newhouse, M. Quantitation of regional aerosol clearance in normal human lung. *J. Appl. Physiol.*, 1972, *33*, 757–762.

27. Secker-Walker, R. H., Provan, J. L., Jackson, J. A., and Goodwin, S. Lung scanning in carcinoma of bronchus. *Thorax*, 1971, *26*, 23–32.
28. Shlesinger, R. B., and Lippman, M. Particle deposition in casts of human upper tracheobronchial tree. *Am. J. Ind. Hyg. Assoc. J.*, 1972, *33*, 237–251.
29. Stolberg, H. O., Patt, N. L., MacEwen, K. F., Warwick, O. H., and Brown, T. C. Hodgkin's disease of lung: roentgenologic-pathologic correlation. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 96–115.
30. Takahashi, K., and Itch, H. New computational model for deposition of aerosol particles inhaled in human respiratory systems. *J. Jap. Health Phys. Sc.*, 1974, *9*, 3–10.
31. Taplin, G. V., Johnson, D. E., Dore, E. K., and Kaplin, H. S. Lung photoscans with macroaggregates of human serum radioalbumin: experimental basis and initial clinical trials. *Health Physics*, 1964, *10*, 1219–1227.
32. Taplin, G. V., and Poe, N. D. Dual lung-scanning technique for evaluation of pulmonary function. *Radiology*, 1965, *85*, 364–368.
33. Taplin, G. V., Poe, N. D., Dore, E. K., Swanson, L. A., Isawa, T., and Greenberg, A. Scintiscanning and roentgenographic procedures in managing major pulmonary disorders. In: Medical Radioisotope Scintigraphy 11, Proc. Symp., Salzburg, 6–15 August 1968. I.A.E.A., Vienna, 1969, pp. 111–158.
34. Taplin, G. V., Poe, N. D., Dore, E. K., Greenberg, D. A., and Isawa, T. Radioaerosol inhalation scanning. In: Pulmonary investigation with radionuclides. Edited by A. J. Gilson and W. M. Smoak. Charles C Thomas, Publisher, Springfield, Ill., 1970, pp. 296–317.
35. Taplin, G. V., Poe, N. D., Isawa, T., and Dore, E. K. Radioaerosol and xenon gas inhalation and lung perfusion scintigraphy. In: Regional Lung Function and Closing volume. Scand. J. Resp., Dis., Suppl. 85, 1974, pp. 144–158.
36. Task Group on Lung Dynamics. Deposition and retention models for internal dosimetry of human respiratory tract. *Health Physics*, 1965, *12*, 173–207.
37. Trinidad, S., Lisa, J. R., and Rosenblatt, M. D. Bronchogenic carcinoma simulated metastatic tumors. *Cancer*, 1963, *16*, 1521–1529.
38. Wagner, H. N., Jr., Sabiston, D. C., Jr., McAfee, J. G., Tow, D. E., and Stern, H. S. Diagnosis of massive pulmonary embolism in man by radioisotope scanning. *New England J. Med.*, 1964, *271*, 377–384.
39. Wagner, H. N., Jr., Lopez-Majano, V., Tow, D. E., and Langan, J. K. Radioisotope scanning of lungs in early diagnosis of bronchogenic carcinoma. *Lancet*, 1965, *1*, 344.
40. Wright, F. W. The radiological diagnosis of lung and mediastinal tumors. Butterworth & Co., Ltd., 1973.

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ROENTGENOLOGY OF SPOROTRICHOSIS*

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ABSTRACT:

Localized cutaneous as well as unifocal and multifocal systemic sporotrichosis may all produce roentgenographic findings. Localized cutaneous nodules may occasionally extend to the bone below producing a locally erosive lesion similar to those more commonly found in blastomycosis. More frequently, sporotrichosis involves the joints, either alone or with accompanying skin nodules. The joint changes are those of a pyogenic arthritis of the knees, elbows, hands, or feet which is difficult to distinguish from pyarthrosis produced by other organisms.

The pulmonary findings depend upon whether or not the lung is the only site of infection. If there are no other areas of involvement, the pulmonary findings may be indistinguishable from those of secondary tuberculosis. However, if the skin or joints are also involved, small nodules appear and apparently do not proceed to cavitation.

SPOROTRICHOSIS, an infection by the fungus *Sporotrichum schenckii*, may present in a variety of forms (Table I). The most common form of this rare infection is localized cutaneous involvement in which ulcerating nodules appear at the site of inoculation. This localized disease does not spread to cause systemic disease but may involve underlying bone. Much rarer is systemic sporotrichosis which is thought to result from inhalation of spores. This systemic disease may be unifocal or multifocal and involve one or more organs such as lung, bone, skin, or meninges. This paper describes the roentgenographic manifestations in the lungs of both unifocal and multifocal systemic disease as well as the roentgenographic changes which occur in bone due to direct extension of localized cutaneous disease. In addition, we have included two new cases of bone and joint involvement in systemic disease.

MATERIAL AND METHOD

All cases of systemic and cutaneous sporotrichosis seen at the University of Michigan Hospital since 1960 were reviewed. Patients were selected for this study if they had either systemic disease or localized cutaneous disease with symptoms referable to bone. Cases of cutaneous disease only, both localized and systemic, were discarded. The available roentgenograms were then reviewed with special attention to bone and chest roentgenograms. For inclusion, lesions must have been proved to be sporotrichosis infection by either culture or smear of that particular lesion. Positive skin cultures were not assumed to prove that an accompanying arthritis or lung nodule was due to *Sporotrichum* infection.

REPORT OF CASES

CASE I. U.F., a 64 year old white male, developed red, tender nodules distributed over his entire body, followed one month later by a migratory arthritis. Six months later the patient

TABLE I

SPOROTRICHOSIS

I. Localized cutaneous
II. Systemic
A. Unifocal (lung, bone, widespread cutaneous)
B. Multifocal

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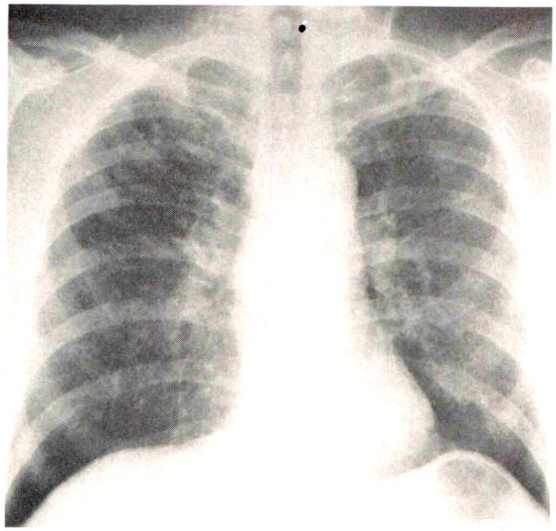

FIG. 1. Case I. Chest roentgenogram demonstrates small ill-defined nodules at both apices.

was admitted with arthritis, cutaneous nodules, weight loss, and a cough. The small joints of the hands, wrists, elbows, knees, and feet were red, tender and swollen. Rales were heard over the lungs. Skin tests for blastomycosis, histoplasmosis, coccidiomycosis, and tuberculosis were negative. The chest roentgenogram showed multiple small non-calcified nodular densities concentrated at both apices (Fig. 1; and 2). There was no cavitation or calcification. Joint roentgenograms of the hands and feet demonstrated demineralization, but were otherwise within normal limits. Cultures of the skin lesions grew *Sporotrichum schenckii.*

The patient died of a pulmonary embolus before therapy could be instituted. Cultures of the apical nodules, of pleural fluid, and of both wrists grew *Sporotrichum schenckii.*

CASE II. M.J., a 51 year old Black alcoholic, presented with a productive cough, dyspnea, and intermittent fever in 1973. The chest roentgenogram (Fig. 3) demonstrated bilateral infiltrates and cavitation suggestive of tuberculosis, but repeated sputum culture and stains were negative for acid fast bacilli. However, fungal cultures grew out *Sporotrichum schenckii.* The patient was treated with amphotericin but he signed out of the hospital against medical advice before the entire course could be administered. Six months later he appeared again with anorexia, weight loss, shortness of breath and hemoptysis. The chest roentgenogram showed enlarging cavities (Fig. 4). Cultures were positive for *Sporotrichum schenckii* despite 2,300 mgs amphotericin and 5-Fluorocytosene. He is currently under treatment with a newly developed fungicide.

CASE III. A 40 year old white male presented with a nodule on the dorsum of the right hand. He had no joint, or pulmonary symptoms. Roentgenograms of the right fifth metacarpal demonstrated a round destructive lesion with slight periosteal elevation (Fig. 5). Cultures grew out *Sporotrichum schenckii.* After curettage, the lesion healed with sclerosis and ingrowth of bone. The patient has had no recurrence.

FIG. 2. Case I. Close-up of the right apex again shows multiple small, ill-defined apical nodules.

FIG. 3. Case II. Cavitating upper and middle lobe infiltrates simulate tuberculosis.

Case iv. A.A., a 45 year old white male, noted swelling and pain in the left knee. A local physician "drained the knee" but three months later symptoms returned. He entered a local hospital where synovectomy revealed "granulomatous changes" and culture demonstrated *Sporotrichum schenckii*. He was treated with a total of 2 gm intravenous amphotericin B but several months later symptoms returned. On admission to the Ann Arbor Veterans Hospital roentgenograms of the left knee showed diffuse demineralization, moderate joint space narrowing, erosions of the medial and lateral tibial plateaus and of the lateral femoral condyle, and a permeative lesion of the proximal tibia with periosteal elevation (Fig. 6). Needle aspiration of the left knee produced no fluid but cultures of the needle tip grew *Sporotrichum schenckii*. The patient subsequently underwent a left knee synovectomy and arthrodesis.

Case v. J.C., a 53 year old white male with Stage ivb Hodgkin's disease and chronic alcoholism noted swelling of the metacarpophalangeal and proximal interphalangeal joints of both hands one year prior to admission. He obtained no relief from either aspirin or prednisone, but much later the symptoms subsided somewhat. One year after initial onset of symptoms the patient noted swelling of both wrists with aching and limited motion. There was loss of extension at the metacarpophalangeal joints of the fourth and fifth digits of the left hand. Hand and wrist roentgenograms on admission showed considerable demineralization, soft tissue swelling over the dorsum of both hands, and a small erosion on the radial side of the proximal left fifth metacarpophalangeal joint (Fig. 7). Subsequently a synovectomy and extension tendon repair were performed. *Sporotrichum schenckii* grew from the synovectomy specimen and from a later needle aspiration of the right wrist.

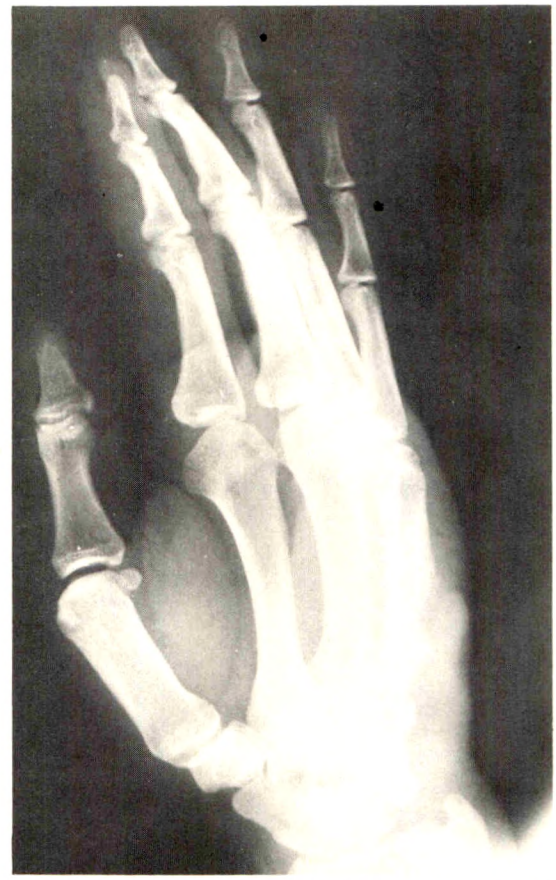

Fig. 5. Case iii. Erosion of the fifth metacarpal and periosteal elevation are associated with a subcutaneous nodule.

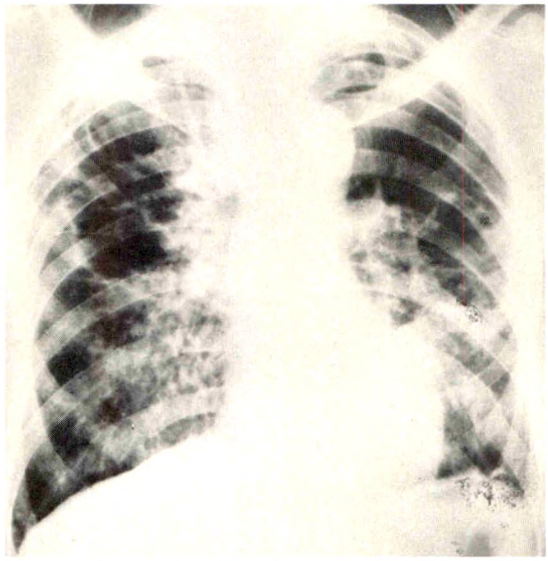

Fig. 4. Case ii. Cavities have enlarged and the infiltrates have not subsided despite amphotericin therapy.

DISCUSSION

Sporotrichum schenckii is a fungus with no apparent limitation in geographic distribution. It may be implanted in the skin of human beings by trauma such as a rose thorn prick. The resulting localized cutaneous lesion may develop satelite nodules

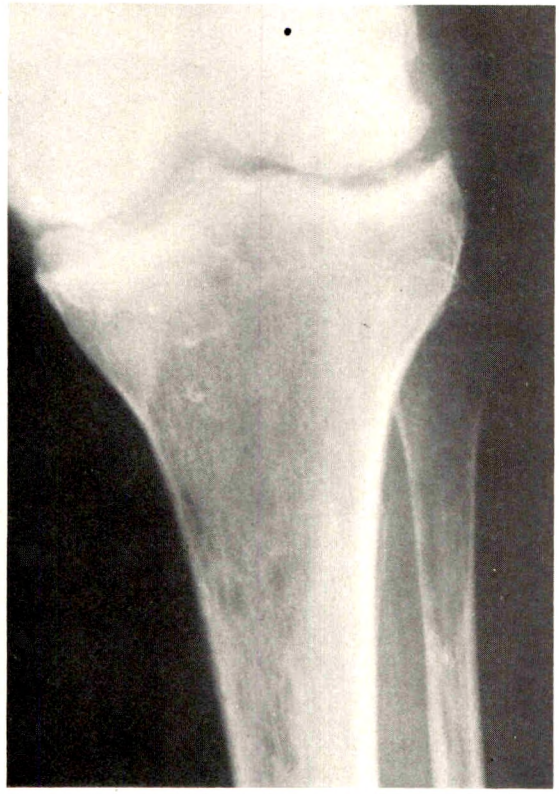

FIG. 6. Case IV. Joint space narrowing and subchondral eburnation are associated with faint periosteal elevation along the medial tibial metaphysis and with metaphyseal mottling.

in the lymphatic distribution of the affected limb, but there is no spread beyond this area. The method of infection in the systemic variety is unknown, although hypothesized by many to be due to inhalation of spores. Systemic infection may involve only one organ, such as the lungs, bone, or skin; or any of these in combination. Skin involvement in systemic disease involves several widespread areas of the body, rather than just one extremity, as in the localized cutaneous form.[3]

PULMONARY DISEASE

Approximately 30 cases of systemic sporotrichosis involving only the lung have been reported.[1,2,4–6] These case reports have all appeared in the internal medicine literature and have described unilateral or bilateral apical infiltrates progressing to cavitation, usually without air-fluid levels. Case II, presented above, is typical of this type of disease. Shortness of breath, hemoptysis, cough, and bilateral apical infiltrates with cavitation suggested tuberculosis. However, when multiple cultures for tuberculosis are negative (as in this case) or when anti-tuberculous therapy fails, sporotrichosis should be considered. No other area of infection, such as skin, bones, or meninges has been reported when this

FIG. 7. Case V. A small erosion at the base of the fifth metacarpal is associated with diffuse osteoporosis of the hand.

pulmonary pattern has been observed. In all cases either sputum or biopsy cultures have been positive.

When multiple organs are infected in systemic sporotrichosis, the pulmonary pattern is quite different than when the lungs alone are involved. In a review of the clinical aspects of sporotrichosis, Wilson *et al.*[7] mentions one case, reported in a Chinese medical journal, of a woman with generalized subcutaneous nodules with a 1 cm solitary smooth-margined, apical lung nodule which did not cavitate and was not accompanied by infiltrates. *Sporotrichum schenckii* grew out of the sputum. Our Case I is the only other reported proved case of pulmonary involvement in multifocal systemic sporotrichosis. The lung changes are similar to other fungal diseases demonstrating multiple small nodules. However, they were concentrated at the apices and did not cavitate. Sputum cultures were repeatedly negative.

BONE FINDINGS

Occasionally, a skin lesion in the localized cutaneous form of sporotrichosis may involve the underlying bone, as in our Case III, but this pattern is much more typical of blastomycosis. The erosive lesion is eccentric, is related in position to a skin nodule, and raises the periosteum. After curettage and amphotericin B therapy it healed with sclerosis and filling in of bone.

Our Cases IV, V and VI illustrate the much more common bone findings of purulent arthritis previously described by Winter and Pearson.[8] Soft tissue swelling, early osteoporosis, erosions, and cartilage narrowing are the most frequently described changes. Although Winter and Pearson claim that lack of osteoporosis may distinguish sporotrichal joint infection from tuberculous infection, our experience indicates that in early sporotrichal joint involvement osteoporosis is present, although it may disappear with treatment. As in tuberculosis, sporotrichosis appears to involve the joint primarily, although occasionally spread occurs to adjacent bone producing a typical osteomyelitic appearance. This pattern of spread distinguishes sporotrichosis from blastomycosis which spreads from skin to underlying joint or from skin to underlying metaphysis to adjacent joint.

The knees, elbows, hands, wrists, and feet are the most frequent sites of sporotrichal infection. Unlike the other fungal diseases and tuberculosis, the vertebrae have not been reported as sites of infection nor have the mandible and ribs, as in actinomycosis.

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REFERENCES

1. Beland, J. E., Mankiewicz, E., and MacIntosh, D. J. Primary pulmonary sporotrichosis. *Canad. M. A. J.*, 1968, *99*, 813–817.
2. Cruthirds, T. P., and Patterson, D. O. Primary pulmonary sporotrichosis. *Am. Rev. Dis.*, 1967, *95*, 845–847.
3. Lynch, P. J., Voorhees, J. J., and Harrell, E. R. Systemic sporotrichosis. *Ann. Int. Med.*, 1970, *73*, 23–30.
4. Ridgeway, N. A., Whitcomb, F. C., Erickson, E. E., and Law, S. W. Primary pulmonary sporotrichosis: report of two cases. *Am. J. Med.*, 1962, *32*, 153–160.
5. Scott, S. M., Peasley, E. D., and Crymes, T. P. Pulmonary sporotrichosis: report of two cases with cavitation. *New England J. Med.*, 1961, *265*, 453–457.
6. Trevathan, R. D., and Phillips, S. Primary pulmonary sporotrichosis: case report. *J.A.M.A.*, 1966, *195*, 181–183..
7. Wilson, D. E., Mann, J. J., and Bennett, J. E., and Utz, J. P. Clinical features of extracutaneous sporotrichosis. *Medicine*, 1967, *46*, 265–279.
8. Winter, T. Q., and Pearson, K. D. Systemic sporotrichosis. *Radiology*, 1972, *104*, 579–584.

XEROGRAPHIC MANIFESTATIONS OF MALE BREAST DISEASE*

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ABSTRACT:

Male breast disease, although overshadowed by its female counterpart, is still a problem which often comes to the attention of the radiologist. The major importance of mammography is differentiating unilateral gynecomastia from cancer. We now recognize four xeroradiographic patterns of gynecomastia: (1) increased ducts only; (2) ductal hyperplasia mimicking adenosis; (3) small ducts with stromal proliferation; and (4) fatty replacement only. These reflect the duration of the process.

Gynecomastic masses are central, smooth, and most important, extend from the nipple outward, usually bisecting the midplane. Ducts may be visible. Carcinoma is usually central, dense, with irregular spiculated margins and, unlike gynecomastia is rarely in the midplane of the nipple, although it can be retroareolar in location. There may be concomitant skin changes or lymphadenopathy seen on the xerogram and these never occur in gynecomastia.

There are numerous etiologies for gynecomastia, and these have been discussed in detail. It is thought that the radiologist should be aware of the pathophysiology of male breast disease and understand its significance.

SINCE the discovery of new and more accurate mammographic techniques, renewed interest in the field of breast roentgenology has been awakened. One of the more recent techniques is xeroradiography, a photoconductive imaging process whose applications to female breast disease have been aptly described and advanced by Wolfe[33] and others. However, little has been written about the xerographic patterns of male breast disease and, more important, about the complex and interesting evaluation of a man with breast complaints. We shall describe the xerographic patterns associated with benign and malignant male breast disease as well as the etiology and pathology of these various entities.

MATERIAL AND METHOD

From November, 1972, to January, 1975, xeromammography was performed on 4,817 patients at the Massachusetts General Hospital, 32 of whom were male, of this group, two had carcinoma, one had an epithelial inclusion cyst, one was judged normal, four had fat replacement, and the remaining 24 had gynecomastia, nine bilaterally.

DISCUSSION

The normal male breast is a rudimentary organ consisting of a nipple, a small branching duct system lined with cuboidal epithelium, and a fatty stroma. Like the female breast, both the stroma and epithelium are responsive to hormonal stimulation. Roentgenographically, the normal male breast is characterized by a minimal amount of fat in the subareolar area. Occasionally a few short ducts may be recognized.

The most common male breast complaint is gynecomastia, a subareolar swelling, sometimes painful, which can be unilateral or bilateral. Pathologically, it represents hyperplasia of normal breast elements. Histologically, the duct proliferates, but no acini are produced. There is also considerable increase in the surrounding

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stroma, as well as fatty replacement. Nipple discharge rarely occurs.

Roentgenographically, gynecomastia presents as a symmetrical cone in the midplane of the nipple. This is because it represents hyperplasia of ducts and connective tissue stroma which normally fan out from the subareolar area.

Although Bannayan and Hajdu[1] state that pathologically the proliferative changes may be eccentric, in our experience all gynecomastia was roentgenographically subareolar in origin, extending in a fan-like configuration, and evenly distributed above and below the midplane of the nipple. We have noted four basic patterns: (1) an increase in ducts only (Fig. 4); (2) ductal hyperplasia mimicking adenosis (Fig. 1; 5; and 6); (3) small ducts with stromal proliferation (Fig. 3); and (4) fatty replacement only (not true gynecomastia—Fig. 6).

The evolution of the histopathology is secondary to the duration of the gynecomastia. Initially, fibroepithelium, stroma, and ducts proliferate. Eventually, there are hyalinization of the stroma and regression of the ducts.[22] Most commonly gynecomastia is idiopathic and found at the time of puberty[16] or senescence. Figure 2 shows a typical pubescent gynecomastia in an 18 year old boy who developed a tender mass under his left nipple. Nydick *et al.*[23] state that this is a self-limiting process usually lasting six months. They found this entity in 38.7 percent of a series of 1,855 boys, and in 75 percent it was bilateral. Likewise, the senescent form will regress spontaneously and will present as a tender, subareolar mass, usually bilateral, but commonly asymmetrical. Both forms roentgenographically display prominent ducts with fat and thickened stroma.

The second major group is iatrogenic

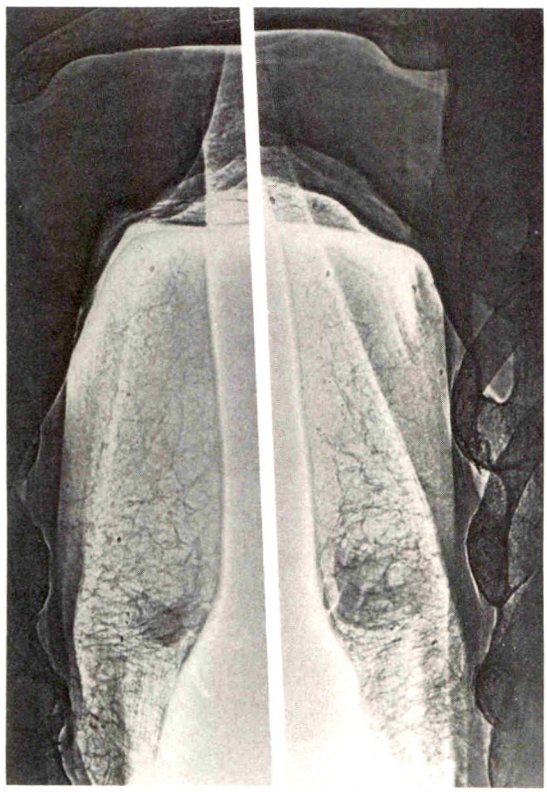

Fig. 1. Moderate ductal hyperplasia on the left, with minimal changes on the right. This type of asymmetry is not unusual.

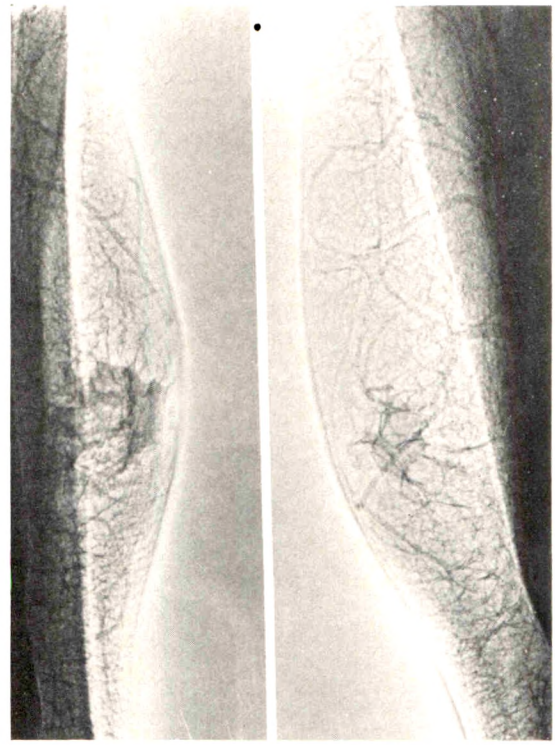

Fig. 2. Bilateral gynecomastia in an 18 year old male. The right side is more severely involved. Note the ill-defined subareolar density fanning out from the nipple midplane. Slight deep eccentricity is appreciated on the right.

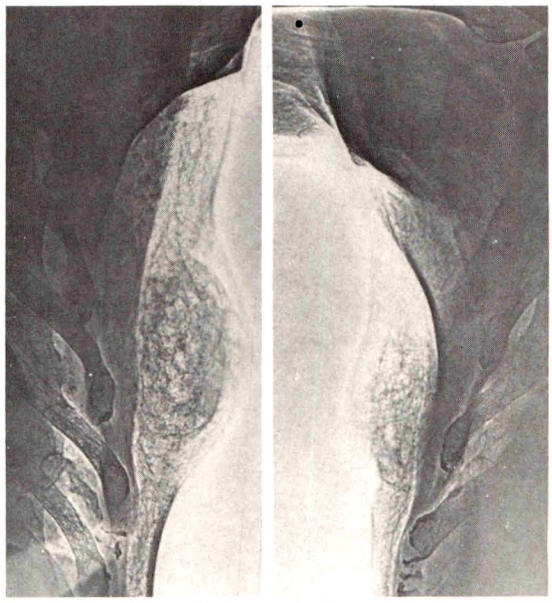

FIG. 3. Numerous small discrete fine ducts intermeshed with stromal proliferation.

gynecomastia secondary to medications. Numerous drugs have been implicated in producing gynecomastia including digitalis,[24] INH,[20] thiazides,[12] steroids,[1] and spironolactone.[4,10] Figure 3 demonstrates a 42 year old laborer who developed unilateral gynecomastia with ductal proliferation after nine months on a thiazide diuretic prescribed for hypertension. After removal of the drug his gynecomastia regressed. Recently, marihuana has also been implicated as a cause of gynecomastia,[14] as has chronic renal dialysis.[21,25]

Exogenous hormone therapy is a well known cause of gynecomastia. Figure 4 shows a 74 year old man with metastatic carcinoma of the prostate treated with stilbestrol. Endogenous hyperestrinism due to failure of hepatic detoxification of naturally produced male estrogens is felt to be the etiology of gynecomastia in liver disease. This includes cirrhosis as well as other chronic hepatic insult.[15,18] Clinically, the testes are usually shrunken symmetrically. Interestingly, Summerskill and Adson[29] described gynecomastia as a manifestation of hepatoma. Gynecomastia, secondary to estrogen effects, manifests considerable stromal increase with duct proliferation (Fig. 5). Schwartz and Wilens[27] claim that well-developed acini are found in approximately 30 percent of patients treated with

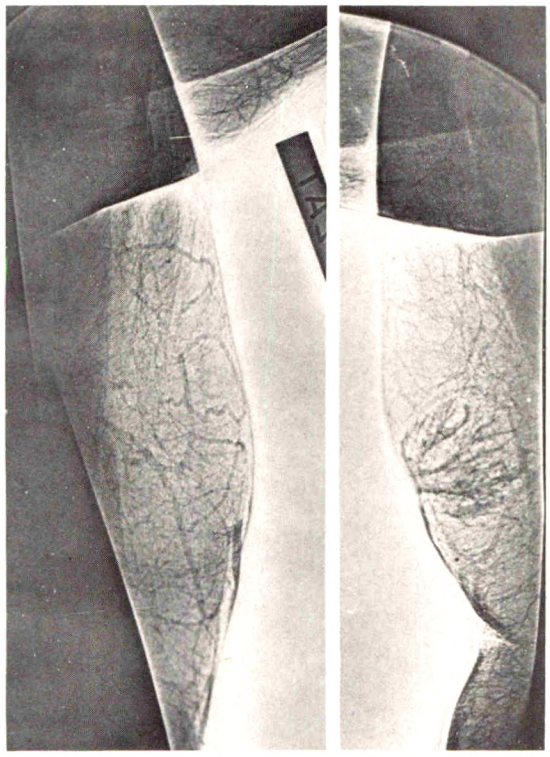

FIG. 4. Individual ducts are readily seen in this case of unilateral gynecomastia. No mass effect is seen.

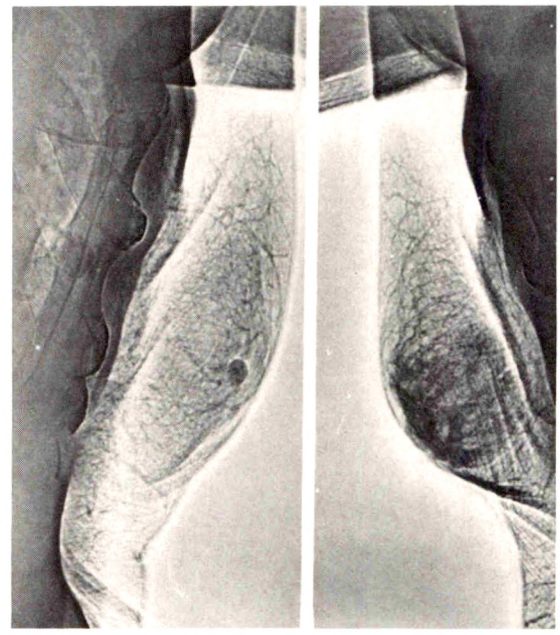

FIG. 5. Severe ductal hyperplasia on the left with normal opposite side.

stilbestrol for prostatic carcinoma. Xerographically the patient shows some ductal hyperplasia which may mimic adenosis (Fig. 1).

There are numerous organic causes of gynecomastia. Treves[30] describes a 10 percent incidence of gynecomastia in testicular neoplasms, the most common type being embryonal carcinoma. Other testicular neoplasms include teratomas, benign or malignant,[7] and an occasional mixed seminoma.[17,31] Choriocarcinoma not only can produce gynecomastia, but may also produce a milk-like substance which exudes from the nipple.[5,11] Carcinoma of the adrenal gland,[28] pituitary adenoma,[19] and hyperthyroidism[2,3] have all been implicated as occasionally producing gynecomastia.

Other possible testicular causes include atrophy due to infection, trauma, or even hydrocele.[12] Klinefelter's syndrome, eunuchism, certain other hypogonadal syndromes, and several developmental anomalies in the lower genitourinary system may manifest gynecomastia. For this reason the testes should be evaluated clinically. If they are atrophied, liver disease should be suspected; if they are unilaterally enlarged, either tumor or hydrocele should be suggested.

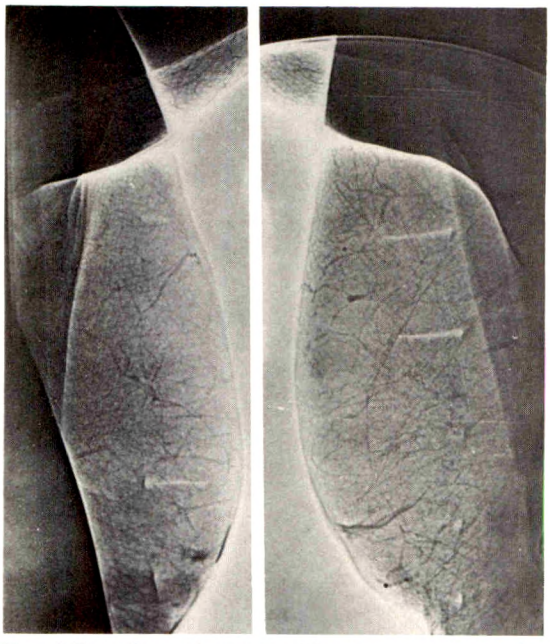

Fig. 6. Pure fat replacement. Not true gynecomastia. No ductal or stromal elements are present. (The linear white streaks are artifacts.)

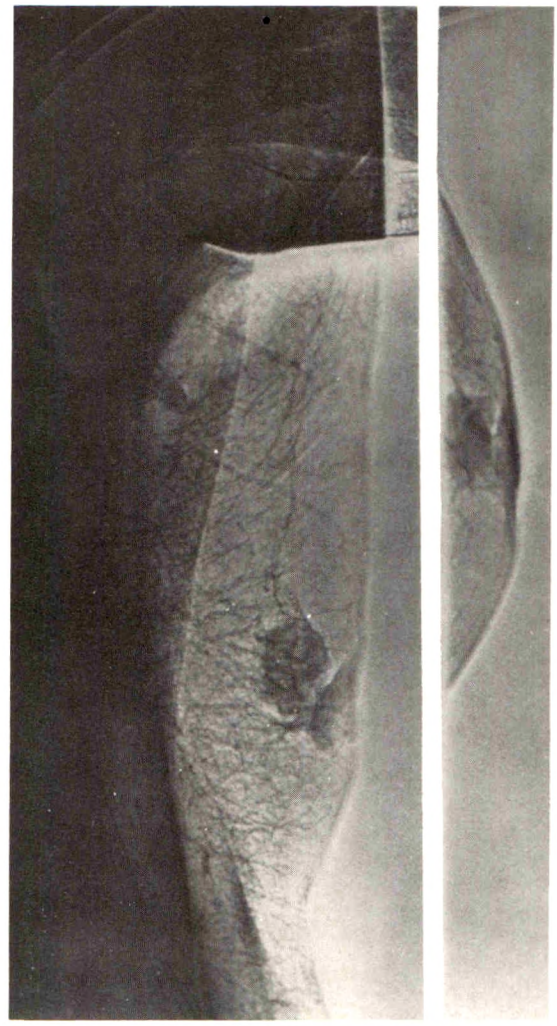

Fig. 7. This carcinoma is a discrete irregular retroareolar mass with concomitant nipple thickening. An enlarged lymph node can be observed in the axilla.

Hardy[13] has described gynecomastia in association with chronic thoracic disease including lung cancer, tuberculosis, and empyema. Sbar[26] described 80 patients with gynecomastia associated with lung disease, mainly carcinoma. Interestingly, 34 were unilateral and 32 of these were on the same side as the pulmonary lesion. The pathophysiology of this is poorly understood. Watson[32] claims that gynecomastia may be associated with leprosy.

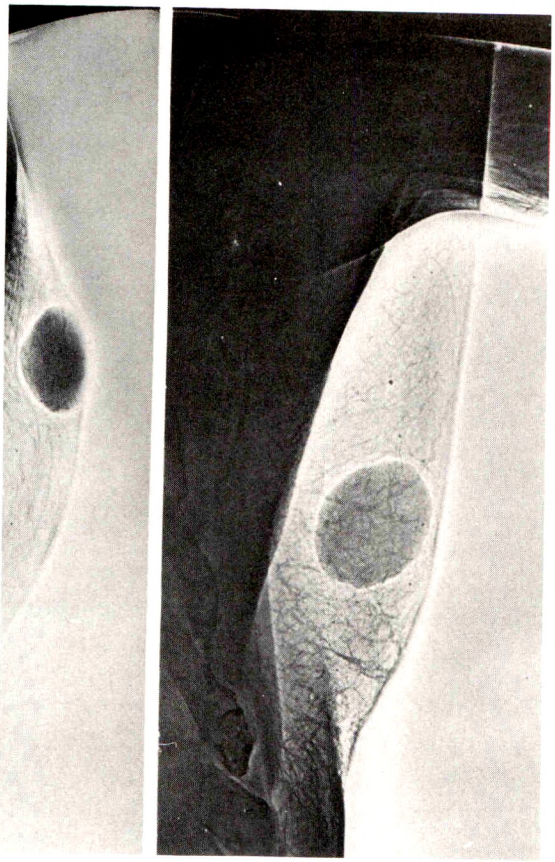

FIG. 8. A sharply marginated, smooth border mass with a halo surrounding it is definitely benign. It is an epithelial inclusion cyst.

The major differential diagnosis of gynecomastia is carcinoma of the male breast, representing 1.0 percent of all breast carcinomas and 0.7 percent of all cancers in men.[34] It is usually found in older patients and is unheard of under the age of 30. Donegan and Perez-Mesa[8] state that the mean age of these patients is 71.2 years.

Unlike gynecomastia, carcinoma usually presents clinically as a central, retroareolar, hard, painless mass. Occasionally skin or nipple ulceration, nipple bleeding or discoloration, and enlarged axillary lymph nodes may be noted.[6]

Roentgenographically, it is a dense retroareolar mass with irregular margins, mimicking in appearance scirrhous carcinoma of the female. It has not been associated with microcalcifications. Early skin changes are common, and many will display enlarged axillary lymph nodes (Fig. 7). Our two cases were subareolar but above the midplane of the nipple. It has been stated by some investigators[8] that carcinoma is usually central, but unlike gynecomastia it is rarely in the midplane of the nipple. Although it was previously felt that the over-all prognosis for male breast carcinoma was poorer than that for females, the sex difference is not statistically significant if correction is made for age and clinical stage.[8] Five year survival without lymph node involvement is 75 percent and with lymph node involvement 30 percent, with an over-all five year survival of 54 percent. The first five years are considered critical.[34]

Occasionally a subareolar cystic mass is discovered in the male breast. This displays the same benign roentgenographic characteristics as a female breast cyst. Figure 8 demonstrates an example of a sharply defined, benign-appearing mass which was found on biopsy to be an epithelial inclusion cyst. Sebaceous cysts may also have a similar appearance.

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REFERENCES

1. BANNAYAN, G. A., and HAJDU, S. I. Gynecomastia: clinicopathologic study of 351 cases. *Am. J. Clin. Path.*, 1972, *57*, 431–437.
2. BECKER, K. L., MATHEWS, M. J., HIGGINS, G. A. JR., and MOHAMADI, M. Histologic evidence of gynecomastia in hyperthyroidism. *A.M.A. Arch. Path.*, 1974, *98*, 257–260.
3. BERCOVICI, J. P., and MAUVAIS-JARVIS, P. Hyperthyroidism and gynecomastia: metabolic studies. *J. Clin. Endocrinol.*, 1972, *35*, 671–677.
4. CLARKE, E. Spironolactone therapy and gynecomastia. *J.A.M.A.*, 1965, *193*, 163–165.
5. COOKE, J. V. Chorioepithelioma of testicle. *Johns Hopkins Hosp. Bull.*, 1915, *26*, 215–221.
6. CRICHLOW, R. W., KAPLAN, E. L., and KEARNEY, W. H. Male mammary cancer: analysis of 32 cases. *Ann. Surg.*, 1972, *175*, 489–494.
7. DALEY, D. W., DOSSETT, J. A., and JULL, J. W. Estrogen secreting sex chromatin-positive teratoma of testis associated with gynecomastia. *Brit. J. Surg.*, 1963, *50*, 816–819.
8. DONEGAN, W. L., and PEREZ-MESA, C. A.

Carcinoma of male breast: 30-year review of 28 cases. *A.M.A. Arch. Surg.*, 1973, *106*, 273–279.

9. Gilder, H., and Hoagland, C. L. Urinary excretion of estrogens and 17-ketosteroids in young adult males with infectious hepatitis. *Proc. Soc. Ex. Biol. Med.*, 1946, *61*, 62–65.
10. Greenblatt, D. J., and Koch-Weser, J. Gynecomastia and impotence complications of spironolactone therapy. *J.A.M.A.*, 1973, *223*, 82.
11. Greenwood, S. M., Goodman, J. R., Schneider, G., Forman, B. H., Kress, S. C., and Gelb, A. F. Choriocarcinoma in man: relationship of gynecomastia to chorionic somatomammotropin and estrogens. *Am. J. Med.*, 1971, *51*, 416–422.
12. Haagenson, C. D. Diseases of the Breast. Second edition. W. B. Saunders Company, Philadelphia, 1971, pp. 76–84; 779–792.
13. Hardy, J. D. Gynecomastia associated with lung cancer. *J.A.M.A.*, 1960, *173*, 1462–1465.
14. Harmon, J., and Aliapoulios, M. A. Gynecomastia in marihuana users. *New England J. Med.*, 1972, *287*, 936.
15. Jacobs, E. C. Gynecomastia following severe starvation. *Ann. Int. Med.*, 1948, *28*, 792–797.
16. Latorre, H., and Kenny, F. M. Idiopathic gynecomastia in seven preadolescent boys: elevation of urinary estrogen excretion in two cases. *A.M.A. Am. J. Dis. Child.*, 1973, *126*, 771–773.
17. Leyson, J. F. Mediastinal seminoma: associated with exophthalmos and gynecomastia. *Urology*, 1974, *3* 366–369.
18. Lloyd, C. W., and Williams, R. H. Endocrine changes associated with Laennec's cirrhosis of liver. *Am. J. Med.*, 1948, *4*, 315–319.
19. McCullagh, E. P., Alivisatos, J. G., and Schaffenburg, C. A. Pituitary tumor of gynecomastia lactation. *J. Clin. Endocrinol. & Metabol.*, 1956, 397–405.
20. Molina, C., and Aberkane, B. Les gynecomasties des tuberculeux pulmonaires. *Semaine hôp. Paris*, 1960, *36*, 834–836.
21. Nagel, T. C., Freinkel, N., Bell, R. H., Friesen, H., Wilber, J. F., and Metzger, B. E. Gynecomastia, prolactin, and other peptide hormones in patients undergoing chronic hemodialysis. *J. Clin. Endocrinol. & Metabol.*, 1973, *36*, 428–432.
22. Nicolis, G. L., Modlinger, R. S., and Gabrilove, J. L. Study of histopathology of human gynecomastia. *J. Clin. Endocrinol. & Metabol.*, 1971, *32*, 173–178.
23. Nydick, M., Bustos, J., Dale, J. H., and Rawson, R. W. Gynecomastia in adolescent boys. *J.A.M.A.*, 1961, *178*, 449–454.
24. Rodstein, M. Gynecomastia: unusal manifestation of digitalis toxicity. *GP*, 1962, *26*, 95–97.
25. Sawin, C. T., Longcope, C., Schmitt, G. W., and Ryan, R. J. Blood levels of gonadotropins and gonadal hormones in gynecomastia associated with chronic hemodialysis. *J. Clin. Endocrinol. & Metabol.*, 1973, *36*, 988–990.
26. Sbar, S. Unilateral gynecomastia. *New England J. Med.*, 1972, *286*, 1367–1368.
27. Schwartz, I. S., and Wilens, S. L. Formation of acinar tissue in gynecomastia. *Am. J. Path.*, 1963, *43*, 797–803.
28. Stewart, W. K., Fleming, L. W., and Wotiz, H. W. Feminizing syndrome in male subjects with adrenal cortical neoplasms. *Am. J. Med.*, 1964, *37*, 455–472.
29. Summerskill, W. H., and Adson, M. A. Gynecomastia as sign of hepatoma. *Am. J. Digest. Dis.*, 1962, *7*, 250–254.
30. Treves, N. Gynecomastia. *Cancer*, 1958, *11*, 1083–1102.
31. Twombly, G. H. Relationship of hormones to testicular tumors. In: Endocrinology of Neoplastic Diseases. Edited by G. H. Twombly, and G. T. Pack, Oxford University Press, New York, 1947, pp. 228.
32. Watson, R. Lepromatous gynecomastia. *Internat. J. Leprosy*, 1971, *39*, 899.
33. Wolfe, J. N. Xeroradiography of the Breast. Charles C Thomas, Publisher, Springfield, Ill., 1972.
34. Wolloch, Y., and Dintsman, M. Primary carcinoma of male breast. *Am. J. Surg.*, 1973, *125*, 628–632.

THE USE OF XERORADIOGRAPHY IN THE INVESTIGATION OF DISEASES OCCURRING IN THE CENTRAL NERVOUS SYSTEM*

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ABSTRACT:

A total of 88 cases of normal and pathological conditions of the skull, brain and spinal cord was studied by skull series, pneumoencephalography, and air myelography. The roentgenographic images of these structures were recorded by conventional roentgenograms, xeroradiograms, and tomograms and compared with each other. In general the high resolution and wide recording latitude of xeroradiography produced a more pleasing, sharper image than that obtained by conventional roentgenography. However, the individual tomographic image was superior to xeroradiography.

THE neuroradiological application of xeroradiography is presented as a new modality in the analysis of disease of the brain and spinal cord. It is a method whereby roentgenographic images are recorded on a photoconductive surface and has enjoyed its greatest acceptance in mammography.[14] Although the Xerox process was developed by Carlson[3] in 1937 and applied in the study of various portions of the human anatomy by numerous investigators,[1,2,4,6,7,11,12] very little has been written on its application in the study of the central nervous system.[9]

The xeroradiographic system offers a number of distinct advantages in comparison to film roentgenography. These include a very wide exposure latitude, increased image resolution, reversibility of image polarity, no requirement for a dark room, and the convenience of using an opaque paper for recording of the image, thus requiring no back-lighting for viewing.[8,14]

This report is a comparison of 88 cases of skull series, pneumoencephalography, and air myelography studied by xeroradiography and conventional film roentgenography. Some of the advantages and disadvantages of xeroradiography are presented and discussed.

TABLE I

Type of Study by Xeroradiography	No. of Cases
Pneumoencephalography	35
Air myelography	10
Skull series	10
Paranasal sinuses and facial bones	13
Orbital region	15
Naso-oro-pharynx	5

TABLE II

Type of Condition	No. of Cases
Normal brain	5
Cerebellopontine angle lesion	7
Pituitary adenoma	5
Cerebral atrophy	4
Brain stem atrophy	3
Hydrocephalus	3
Cerebellar atrophy	2
Postsurgical change	3
Porencephaly	2
Arnold-Chiari malformation	2
Normal spinal cord	3
Tethered cord	2
Syringomyelia	2
Atrophy of the spinal cord	2
Spinal cord tumor	2
Craniopharyngioma	1
Hydrencephaly	1
Meningioma	1
Retinoblastoma	1

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Table III

AVERAGE EXPOSURES USED

	MAS	KV	Distance	Phase
A. Pneumoencephalography—Anteroposterior (AP) and lateral (lat.) views of the skull				
Xeroradiography (negative mode)				
Adult	120	120	100 cm	3
Child	50	80	100 cm	3
Conventional Roentgenography (grid, screen, high speed film)				
Adult (AP)	64	85	100 cm	3
(lat.)	64	80	100 cm	3
Child (AP)	32	85	100 cm	3
(lat.)	32	80	100 cm	3
B. Air Myelography (cervical spine)				
Xeroradiography (negative mode)				
Adult	100	120	100 cm	3
Child	40	80	100 cm	3
Conventional Roentgenography (grid, fast screen, high speed film)				
Adult	64	75	100 cm	3
Child	32	60	100 cm	3
C. Air Myelography (thoraco-lumbar)				
Xeroradiography (negative mode)				
Adult	100	110	100 cm	3
Child	40	65	100 cm	3
Conventional Roentgenography (grid, fast screen, high speed film)				
Adult	64	78	100 cm	3
Child	32	60	100 cm	3

Table IV

COMPARISON OF IMAGES

Regional Anatomy and Tissue Type	Conventional Roentgenogram	Conventional Tomogram	Xeroradiogram	Xerotomogram
Skin	o	o	++	+
Subcutaneous tissue	o	o	++	+
Bone—skull	+	+++	++	+++
facial	+	+++	++	+++
Paranasal sinuses	+	+++	++	+++
Mastoid area	+	+++	++	+++
Posterior nasopharynx	+	+++	++	+++
Falx cerebri	+		++	
Meninges	o	o	+	
Cerebral sulci	+	+	++	
Cerebellar folia	+	++	++	
Cisterns—pontine	+	+++	++	
cerebellopontine angle	+	+++	++	
suprasellar	+	+++	++	
interpeduncular	+	+++	++	
cisterna magna	+	+++	++	
Diaphragm of sella	+	+++	+++	
Optic nerve and chiasm	o	++	+	
Basilar artery	o	++	+	
Subarachnoid air	+	++	++	
Subdural air	+	++	+++	
Intracranial calcification	+	++	++	
Intracranial foreign body	+	++	++	
Spinal canal	+	+++	++	
Spinal cord	o	+++	++	

o = poor; + = fair; ++ = good; +++ = excellent.

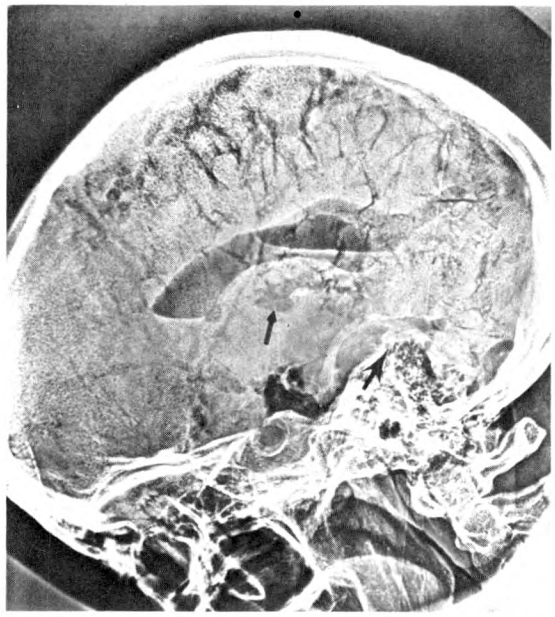

FIG. 1. *Cerebral Sulci and Basal Cisterns.* Xeroradiogram of skull shows gas in lateral, third (small arrow), and fourth (large arrow) ventricles. Gas in cerebral sulci and basal cisterns is well seen.

MATERIAL AND METHOD

A total of 45 cases with various neurological symptoms which were studied by pneumoencephalography, air myelography, or both was picked at random for this study over a period of two years. An additional 43 cases of skull roentgenograms including special views of the paranasal sinuses, orbits, and nasopharynx in patients with neurological and ophthalmological problems were also included in this study (Table I). The normal and pathological findings which were encountered have been listed in Table II. The age of the patients who were studied ranged from one month to 67 years. Prior to this study numerous preliminary "test-exposures" were made with single and three-phase generators varying the milliampere-seconds (MAS) and kilovoltage (KV) in order to obtain the best image for the demonstration of various anatomical structures outlined by air. The

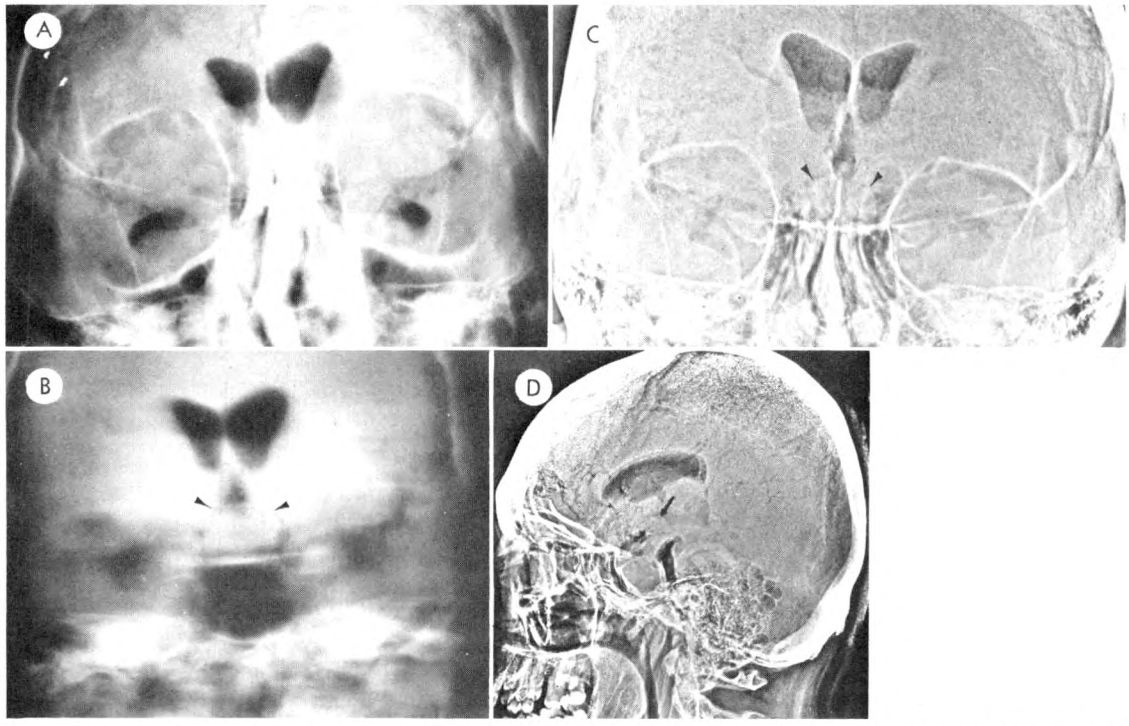

FIG. 2. *Chromophobe Adenoma* (40M). (*A*) Conventional roentgenogram. Suprasellar mass does not show in frontal view. (*B*) Tomography clearly demonstrates suprasellar mass (arrow head) deforming inferior portion of third ventricle. (*C*) Xeroradiogram. Suprasellar mass is shown (arrow head) in frontal view. (*D*) Lateral view also demonstrates large suprasellar mass deforming third ventricle.

average exposures used for xeroradiography and conventional roentgenography in pneumoencephalography and air myelography are listed in Table III.

Satisfactory exposures for xeroradiograms of air myelography varied more than for pneumoencephalography, depending on the different level of interest of the spinal canal in addition to the age and size of the patient. Similar exposures were used for xerotomograms. When a positive mode is used in xeroradiography, significantly greater exposure is necessary in contrast to a negative mode.

DISCUSSION

Xeroradiograms were compared with conventional roentgenograms, tomograms, and xerotomograms of specific tissue and anatomical regions. The comparison of each method has been listed in Table IV and the image of each was graded as poor (0), fair (+), good (++), and excellent (+++).

In general the images of the ventricular system, brain stem, basal cisterns, cerebral sulci and cerebellar folia were shown in greater detail in xeroradiograms (Fig. 1) than in conventional roentgenograms due to the higher resolution of this technique. Studies have shown that 15–50 lines per centimeter can be recorded in xeroradiography in contrast to 8–16 lines in film roentgenography (1 mm focal spot).[8,13] Although the images of normal anatomical structures such as the recesses of the third ventricle, the aqueduct, and the fourth ventricle were shown more clearly in routine xeroradiography than in conventional roentgenography, the images obtained by tomography were the best. However, the advantage of xeroradiography over tomography was the number of films used. Although several films were needed to cover a certain anatomical

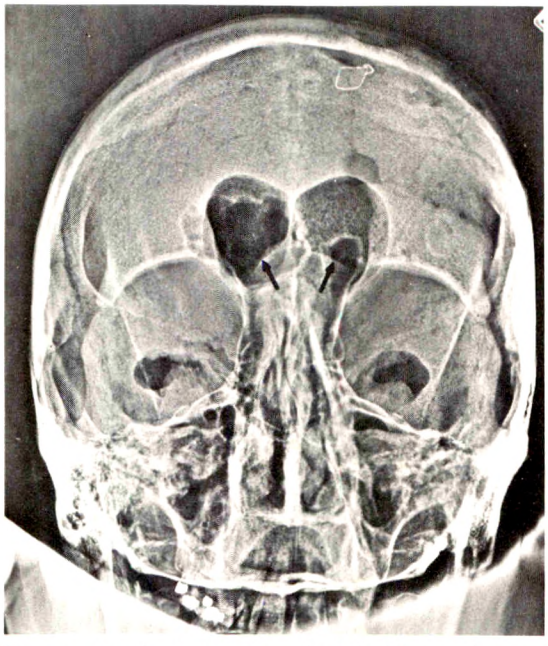

FIG. 3. *Craniopharyngioma* (30M). Xeroradiogram shows large suprasellar mass due to craniopharyngioma (arrow) which is not calcified.

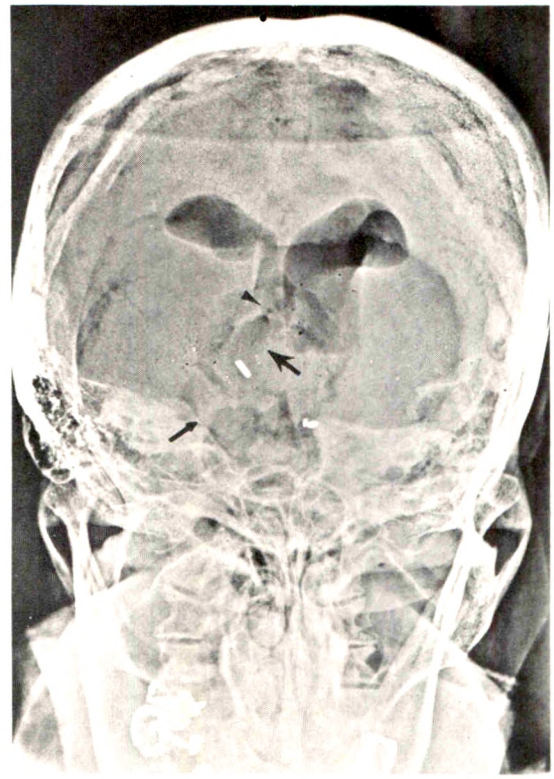

FIG. 4. *Recurrent Acoustic Neuroma* (42F). Xeroradiogram shows displacement of aqueduct (arrow head) and rotation of fourth ventricle to left due to large right cerebellopontine angle mass. Compressed left cerebellopontine angle cistern is due to displaced brain stem.

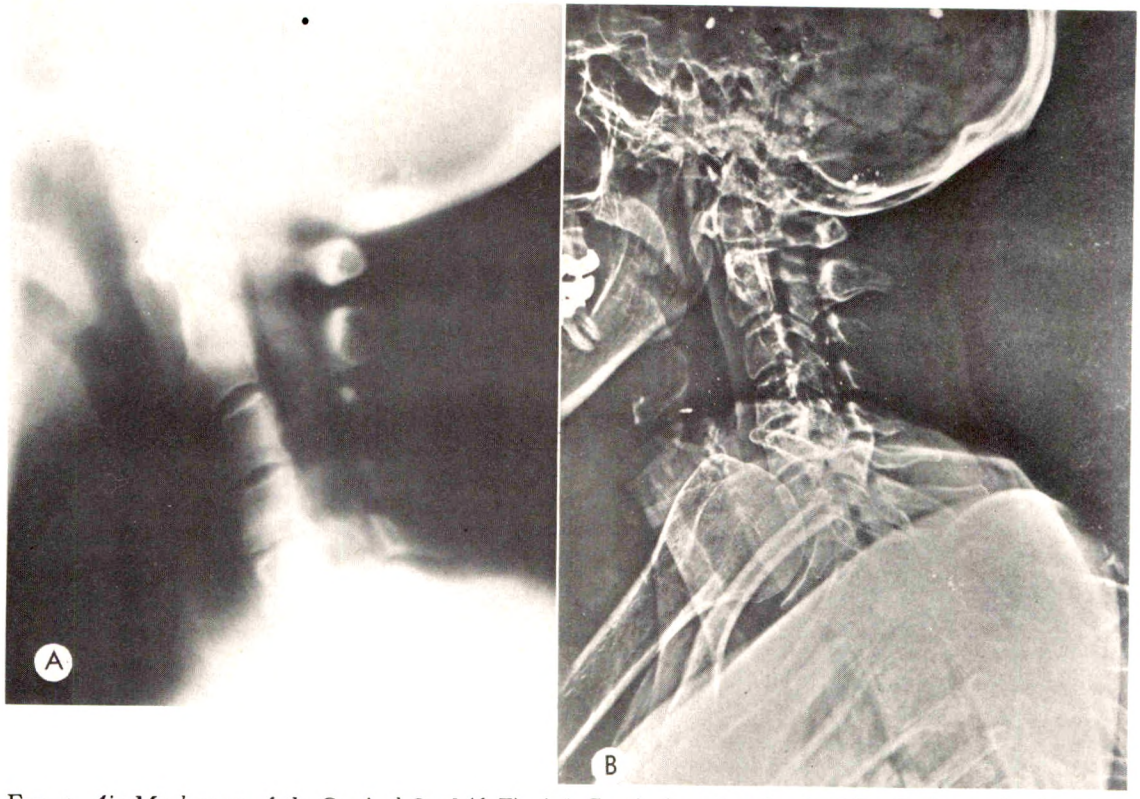

FIG. 5. *Air Myelogram of the Cervical Cord* (60F). (*A*) Cervical cord at level of C_3–C_5 is poorly shown on single conventional tomographic cut due to scoliosis of cervical spine. (*B*) Xeroradiogram outlines entire length of cervical cord as a single image.

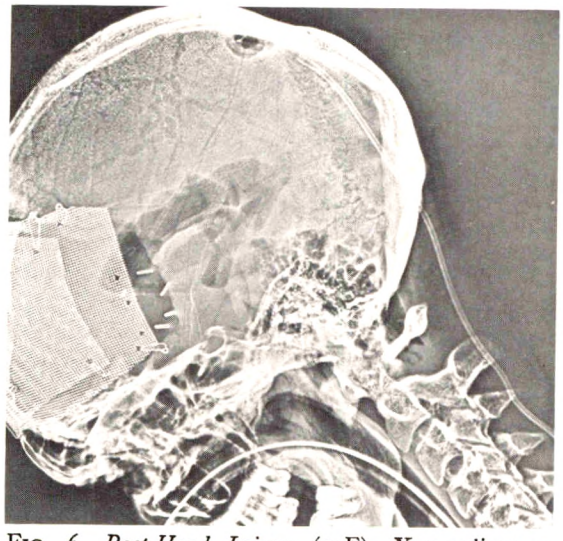

FIG. 6. *Post-Head Injury* (24F). Xeroradiogram clearly demonstrates wire-mesh covering bony defect, metal clips, and catheter within lateral ventricle.

area in tomography, xeroradiography covered it in a single exposure. Similarly, masses in the suprasellar (Fig. 2; and 3) or in the cerebellopontine angle cisterns (Fig. 4) were adequately shown with a single xeroradiogram in contrast to the need for several films in tomography. When the skull or cervical spine was deformed, midline structures such as the brain stem and spinal cord were not completely shown in a single tomogram but required several cuts. Xeroradiography, in contrast, encompassed the similar area with a single image (Fig. 5).

The property of edge enhancement in xeroradiography allows good delineation of structures whose roentgenographic density is similar to that of the surrounding tissue if the margins are sharp. This has allowed the demonstration of intracranial calcification, clips, pantopaque, and catheters to be

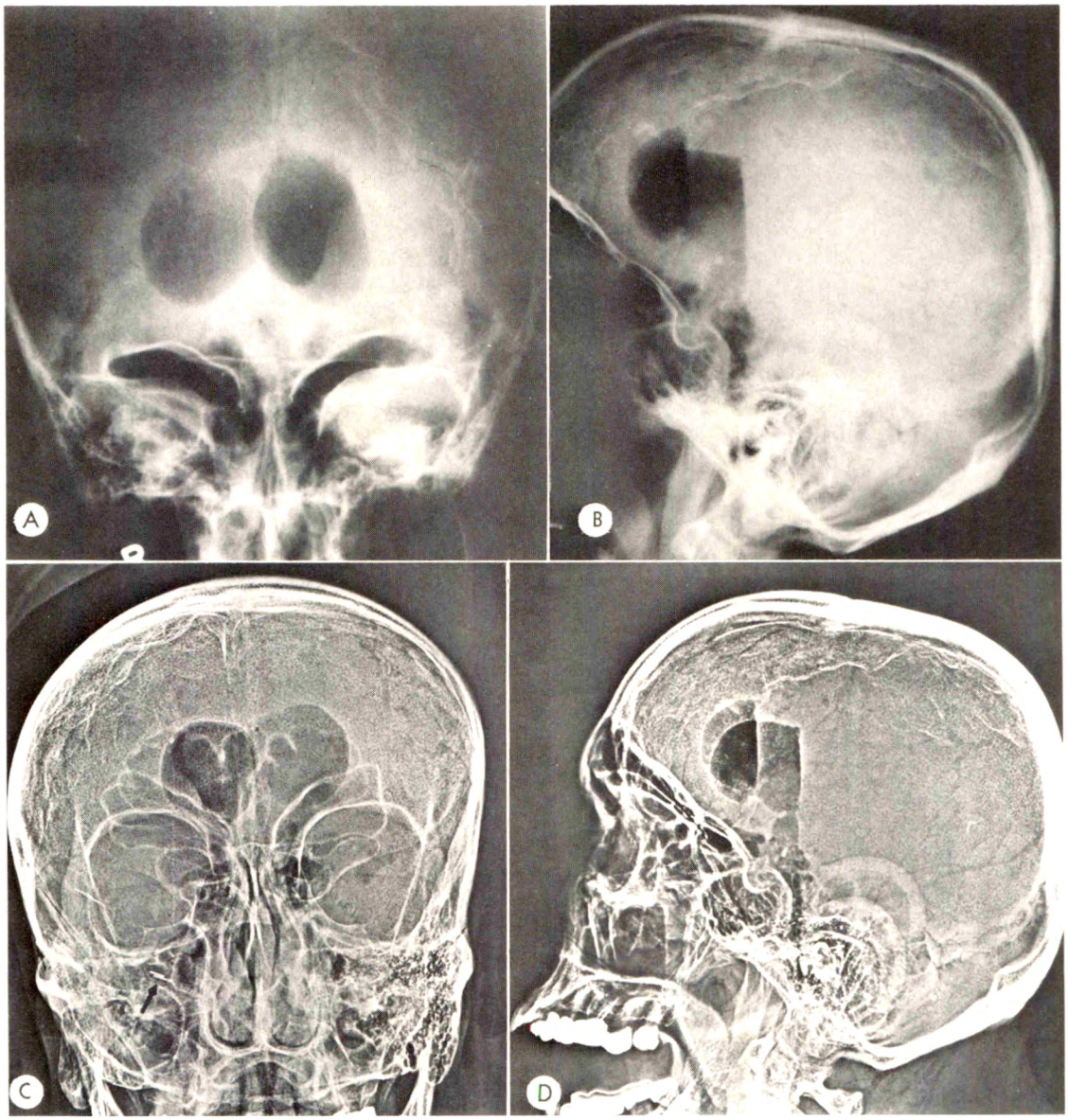

Fig. 7. *Meningeal Calcification* (68M). (*A*) Conventional roentgenogram. Anteroposterior view of skull shows air in frontal horns, but outline of the skull is poorly seen. (*B*) Lateral view shows skull outline along with lateral and third ventricles but meningeal calcification is poorly seen. (*C*) Xeroradiogram. Skull, brain, and meningeal calcification are shown well in frontal view. Metal wire in left internal auditory canal (arrow) is also shown. (*D*) Meningeal calcification is better seen in relation to skull than in (*A*).

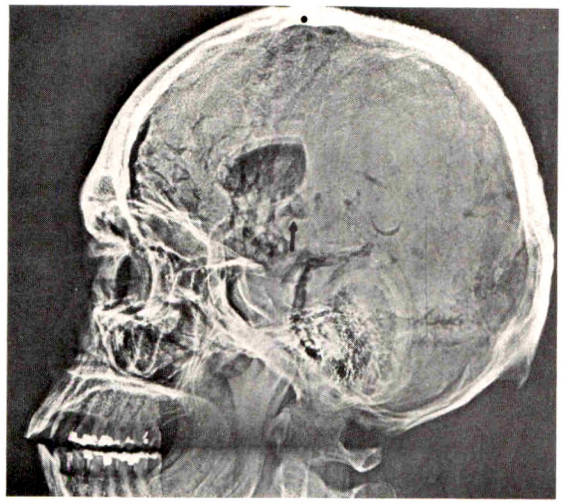

FIG. 8. *Acromegaly* (40M). Xeroradiogram shows large pituitary adenoma which bulges into sphenoid sinus and extends into suprasellar cistern to deform third ventricle. Subdural gas anterior to frontal lobe and subarachnoid gas in basal cisterns are clearly seen.

shown in greater detail than by conventional roentgenograms (Fig. 6).[5,10] Thin meningeal calcifications (Fig. 7) or gas in the subdural or subarachnoid spaces was more sharply delineated in xeroradiograms than in conventional roentgenograms (Fig. 8).

Another important property of xeroradiography is its wide recording latitude.[13] This has allowed xeroradiography to demonstrate all radiodensities on a single exposure in contrast to the necessity of several films with different exposure factors when conventional films are used (Fig. 9).

Facial bones and soft tissue of the posterior nasopharynx were shown with a sharp, etched quality in xeroradiograms in many of the skull series. Xeroradiography has allowed good demonstration of various pathological conditions in the naso-oropharynx in addition to the ocular (Fig. 10)

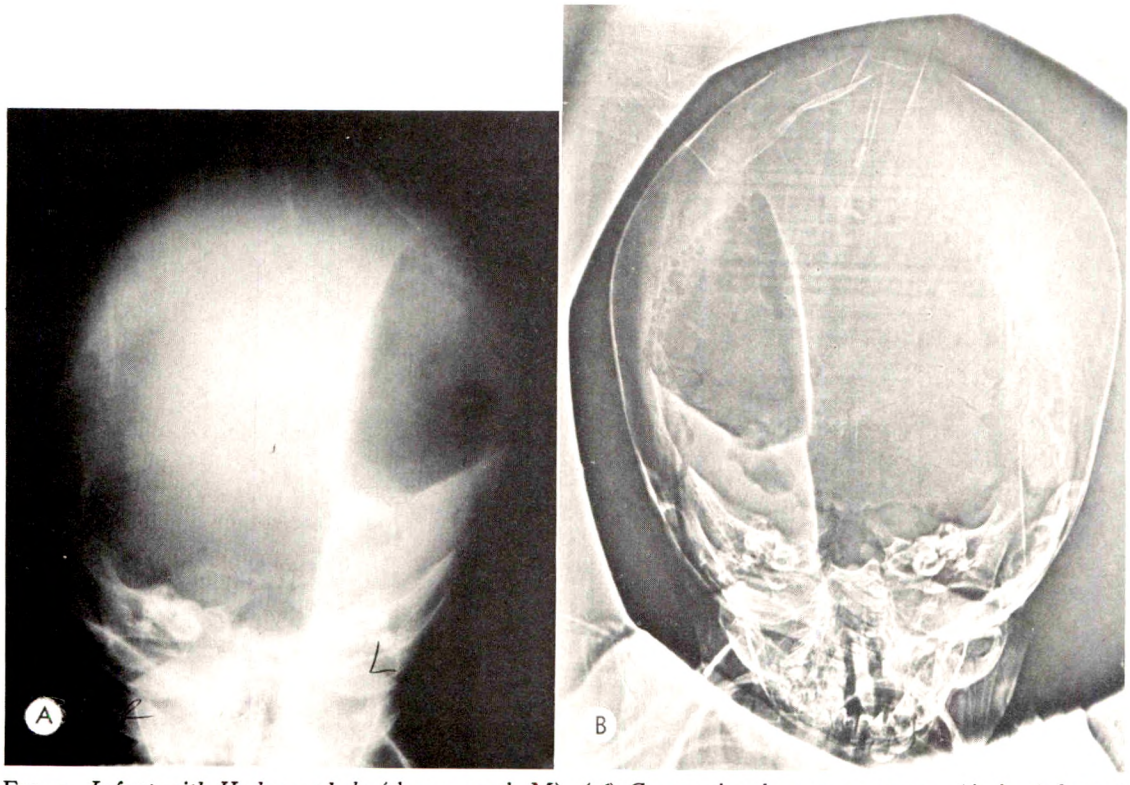

FIG. 9. *Infant with Hydrencephaly* (three month M). (*A*) Conventional roentgenogram. Air in deformed lateral ventricles is seen, but skull outline is not. (*B*) Xeroradiogram. Enlarged skull with thinned bone is shown with air in deformed lateral ventricles. Cochlea and semicircular canals in petrous bones are clearly shown.

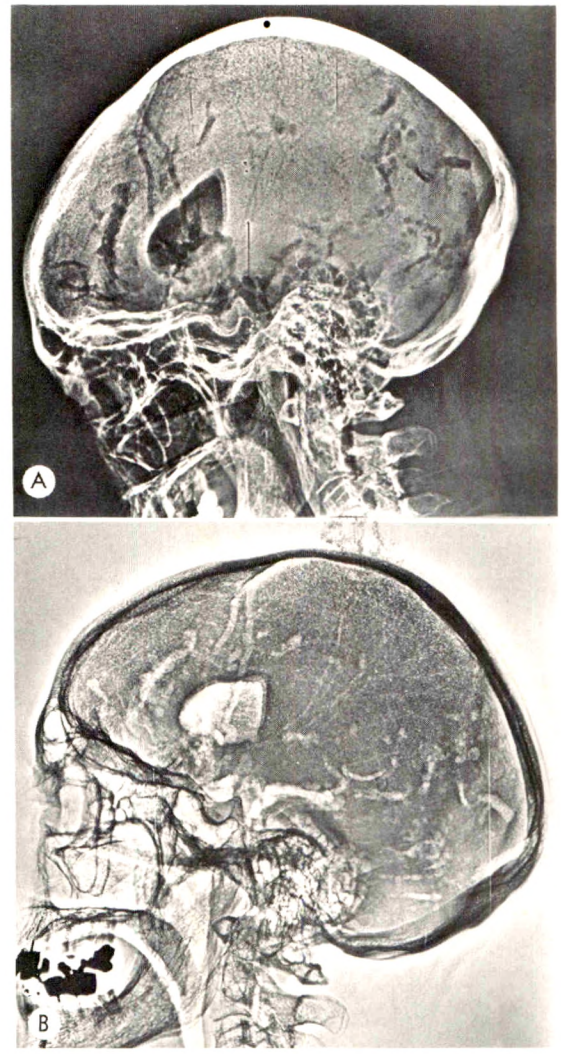

Fig. 10. *Comparison of Negative and Positive Modes in Xeroradiography.* (*A*) Negative image shows gas in the cerebral sulci, basal cisterns, and lateral ventricles in detail. (*B*) Positive image shows bony details more sharply than above. Vascular groove in skull is more difficult to separate from gas in cerebral sulci.

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and mastoid regions. Tomography was often unnecessary in many of these cases.

The use of positive and negative modes in xeroradiography has delineated certain lesions in the basal cisterns in greater detail (Fig. 11). The negative mode was used more often in routine pneumoencephalographic studies. The positive mode was useful in the detailed analysis of bone trabeculation when cases of meningioma or acromegaly with osseous involvement were studied by pneumoencephalography. Positive images were used more frequently for routine and special views of the skull requiring bony detail.

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REFERENCES

1. Bedrossian, C. W., and Martin, J. E. Xeroradiography of lung. *Radiology*, 1973, *107*, 217–218.

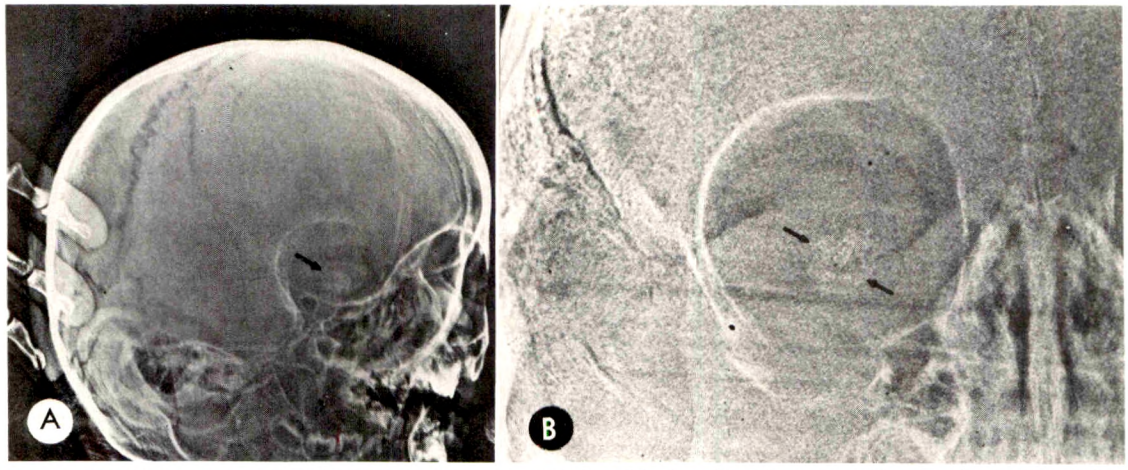

Fig. 11. *Retinoblastoma* (6F). (*A*) Xeroradiogram of left orbital region shows calcification in ocular globe (arrow) which was less clearly seen on conventional roentgenograms. (*B*) Oblique magnification of orbit pinpointing calcium (arrows).

2. Campbell, C. J., Roach, F. J., and Jabbur, F. Xeroroentgenography: evaluation of its use in diseases of bones and joints of extremities. *J. Bone & Joint Surg.*, 1959, *41-A*, 271–277.
3. Carlson, C. F. Electrophotography. U.S. Patent No. 2,297,691, 1937.
4. Doust, B. D., and Ting, V. M. Xeroradiography of larynx. *Radiology*, 1974, *110*, 727–730.
5. Doust, B. D., Ting, V. N., and Chuang, V. P. Detection of aspirated foreign bodies with xeroradiography. *Radiology*, 1974, *111*, 725–727.
6. Griffiths, H. J., and D'Orsi, C. J. Use of xeroradiography in cruciate ligament injuries. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *121*, 94–96.
7. Guibor, P. Xeroradiography and ophthalmology. *Tr. Acad. Ophth.*, 1973, *77*, 751–759.
8. Hills, T. H., Stanford, R. W., and Moore, R. D. Xeroradiography: present medical application. *Brit. J. Radiol.*, 1955, *28*, 545–551.
9. Schertel, L., Winkel, K., Motzkus, F., and Kraska, H. Die Xeroradiographie des Schädels. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1974, *121*, 541–549.
10. Woesner, M. E., and Sanders, I. Xeroradiography: significant modality in detection of nonmetallic foreign bodies in soft tissues. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 636–640.
11. Woesner, M. E., Braun, E. J., and Sander, I. Xeroradiographic zonography of larynx and hypopharynx. *Ann. Otol., Rhin. & Laryn.*, 1974, *83*, 42–48.
12. Wolfe, J. N. Xeroradiography of bones, joints, and soft tissues. *Radiology*, 1969, *93*, 583–587.
13. Wolfe, J. N. Xeroradiography: image content and comparison with film roentgenograms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 690–695.
14. Wolfe, J. N. Mammography. *Radiol. Clin. North America*, 1974, *12*, 189–203.

Vol. 125, No. 3

THORIUM INDUCED HEPATIC HEMANGIOENDOTHELIOMA*

ROENTGEN-ANGIOGRAPHIC FINDINGS IN TWO ADDITIONAL CASES WITH CLINICAL "INFORM AND CONSENT" PROBLEMS

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ABSTRACT:

1. Two additional cases of presumed, thorium-induced hemangioendothelioma of the liver are reported.
2. The angiographic pattern of large venous lakes conforms closely to the pathologic descriptions of this malignancy.
3. To our knowledge, angiography of the thorotrast (and vinyl chloride) liver, before the clinical evolution of malignancy, has not been recorded and may provide information of prognostic and therapeutic value.
4. Incidental identification of thorium deposits in the tissues of clinically healthy patients creates perplexing "inform and consent" problems. Presumably, these must be solved on an individual basis.
5. Because the use of thorotrast has been discontinued since the early 1950s, all physicians reviewing chest and abdominal roentgenograms should be aware of the characteristic appearance of human thorotrast deposits, especially in the liver, spleen and celiac lymph nodes.
6. The number of living thorotrast "carriers" and previous deaths from thorotrast related malignancy seems highly uncertain at this time.

THE late oncogenic effects of intravascular thorium dioxide have been recognized for many years.[9,11,19,21] This communication concerns two additional cases of thorotrast induced hepatic malignancy which occurred 26 and 28 years after thorotrast cerebral angiography. The incidental discovery of thorium deposits during chest roentgenography in one of these patients, who was then clinically well, raised perplexing "inform and consent" problems which are discussed below (Case 1).

In 1972, Janower *et al.*[10] stressed the importance of recognition and study of thorotrast injected patients, since this is a rapidly disappearing group of individuals. The remaining thorotrast "carriers" are a group of persons who will probably disappear from our population within the next 25 years. More important, their characteristic abdominal roentgenograms may escape accurate recognition, especially by recently trained radiologists and other physicians[14,15] (Fig. 1, *A* and *B*; and 2*A*).

The actual number of humans injected with thorotrast is unknown but has been estimated to be between 50,000 and 100,000.[8] Reports also suggest a substantial difference of opinion with regards to the cancer liability involved.[2,5,10,14] Although most thorium related malignancies recorded are hepatic and have followed intravascular injection of the thorium colloid, occasionally malignancy has been reported following thorium use as a contrast medium for intracavitary roentgenographic studies, such as retrograde pyelography,[13,23] bronchography,[4] and visualization of the paranasal sinuses.[7] Sarcomas appearing in the edge of thorotrast granulomas of the neck, due to accidental extravascular deposits of thorotrast, have also been recorded.[5]

Thorotrast, the purified form of colloidal

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thorium dioxide, has been used as a contrast medium in roentgenology since 1930. Because of its deposition in the reticuloendothelial cells, intravascular thorotrast was employed for many years to opacify the spleen and liver.[14] Its dense radiopacity also made it a popular agent for cerebral angiography. Thorotrast use was largely abandoned in the early 1950s due to the growing awareness of its potential oncogenic effects and the risks of severe interstitial fibrosis with accidental extravascular injection.[1,2,16]

After intravascular injection, the thorium colloid is deposited in the reticuloendothelial system; approximately 70 percent in the liver and 20 percent in the spleen. Most of the remaining ten percent is deposited in lymph nodes and bone marrow.[17] Accumulation in the lymph nodes is most striking in the celiac area due to deposition in efferent hepatic lymph nodes.[5]

Radioactive disintegration is complex and, in biologic material, its late distribution is subject to variation. There is an overwhelming preponderance of alpha energy (90 percent), with a beta particle energy contribution of about nine percent and gamma radiation approximately one percent.[17] From the human clinical viewpoint, the biological and physical half-lives of thorium are virtually "forever," the former being 400 years; the latter 1.4×10^{10} years.[9]

Although oncogenic risks were predicted as early as 1932,[6] the first recorded case of thorium-induced malignancy, a liver sarcoma, was not reported until 1947.[18] By 1972, an additional 123 cases of thorotrast liver malignancy had been documented, but only 18 of these were reported from the United States.[20] Although the latent period between thorium injection and liver malignancy has varied from three years to 35 years, all of these 123 cases occurred in patients between 49 and 55 years of age.[20] Isolated reports of thorium related malignancy continue to appear in the literature.[12,23,24]

REPORT OF CASES

CASE 1. This 52 year old white female had been in good clinical health for many years until December, 1970, when she noted a subjective feeling of generalized "puffiness." Physical examination was entirely normal as were hematologic studies and urinalysis. A chest roentgenogram showed lacelike, reticular, and punctate opacities in the convexity of the upper liver and splenic margins (Fig. 1*A*). This discovery led to additional survey roentgenograms which confirmed the presence of opacities, suggestive of thorotrast deposits, in the liver, spleen, and regional lymph nodes (Fig. 1*B*). Laboratory studies showed a marked increase in the alkaline phosphatase and the SGOT levels, confirmed by bench methods. Discreet inquiry disclosed that this patient had undergone thorotrast cerebral angiography 26 years previously, following a transient left hemiparesis.

Serious problems arose concerning the wisdom of informing this patient that she was a "carrier" of radioactivity with a risk of subsequent cancer death. Because this patient could have lived in reasonably normal clinical health for an undetermined number of years, it was deemed advisable to withhold this information from her and to refrain from further investigative studies. This decision was made after consultation with her husband, who agreed that such information would probably devastate the patient's morale and should be withheld until such time as she might become clinically ill.

Twelve months later, in December, 1971, because of a history of acute anxiety, and a weight loss of 18 pounds, the patient was re-evaluated. At this time, the liver was slightly enlarged but non-tender. Her clinical laboratory studies were similar to those one year earlier. The patient was ambulatory and able to pursue limited activities. She maintained this clinical status until two years later, when, in February, 1974, she was hospitalized because of sudden pain in the right flank. Re-examination showed a markedly enlarged, firm liver. Roentgenographic examinations of the gastrointestinal tract, gallbladder, and urinary tract were normal, except for the enlarged liver and the previously noted thorium deposits. Hepatomegaly was the only abnormality noted on physical examination. Radioactive liver scan showed diffuse, widespread perfusion defects of varying size, and the alkaline phosphatase and SGOT levels were markedly elevated.

The serum electrophoretic pattern, alpha fetoprotein, serum electrolytes, VDRL, prothrombin, partial thromboplastin levels, and urinalysis were normal. Surprisingly, the blood

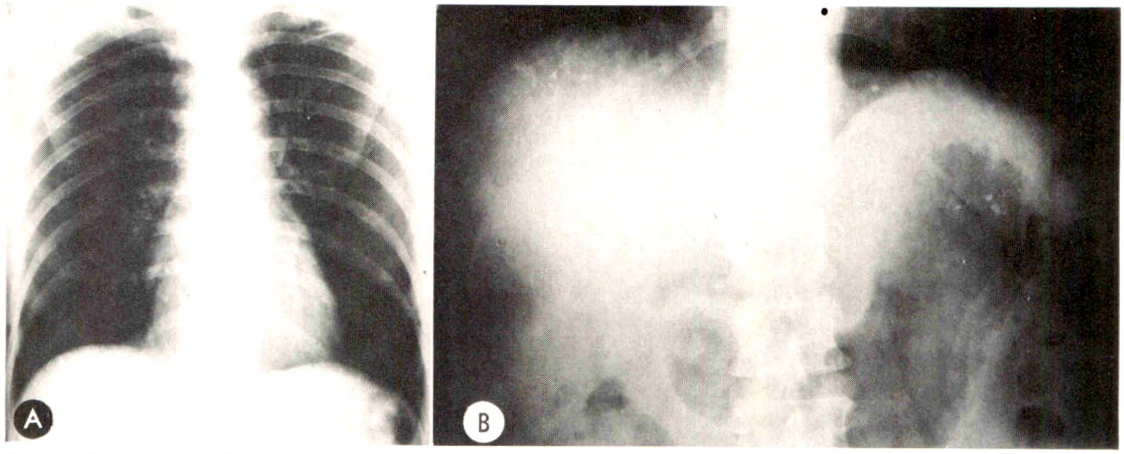


Fig. 1. Case 1. (*A*) Chest roentgenogram procured as part of routine physical examination, showing linear hepatic and punctate splenic opacities. (*B*) Reticular hepatic, punctate splenic, and solid celiac nodal opacities shown on abdominal roentgenogram.

cell count, hemoglobin and hematocrit were also normal. Liver biopsy was not performed for fear of possible hemorrhage in an enlarged, fibrotic, and possibly malignant tumor.

Celiac angiography showed hepatic replacement by large vascular "lakes" which filled early in the arterial phase and remained opacified for a period exceeding 30 seconds (Fig. 1, *C* and *D*). The portal circulation appeared normal with hepatopedal flow and no evidence of varices. Because of the history, the angiographic findings suggested the large, vascular pools of hemangioendothelioma.[5]

The patient's pain subsided and she returned home, but she became intensely jaundiced and continued to lose weight and was rehospitalized several weeks later. On readmission to the hospital, laboratory studies indicated marked elevation of alkaline phosphatase, elevated prothrombin time and profound anemia. The Coombs' test was negative and serum folate was decreased. The patient was dyspneic with a massive right pleural effusion. Palliative measures kept her pain-free with progressive de deterioration, and she expired on July 8, 1974, seven weeks after her final hospital admission.

Necropsy showed diffuse angiosarcomatosis, macroscopic in the liver and spleen and microscopic in the left adrenal, left renal and bone marrow tissues.

Grossly, the liver surface was firm and fibrotic with gray and whitish-brown surface discoloration and dense, linear fibrotic strands. The cut surface showed replacement by nodules of varying size, many of which were necrotic and hemorrhagic. Diffuse, ovoid vascular lesions containing blood clots were visible. Microscopically, the tumors were composed of anastomosing vascular channels that were dilated to varying degrees and which were lined by

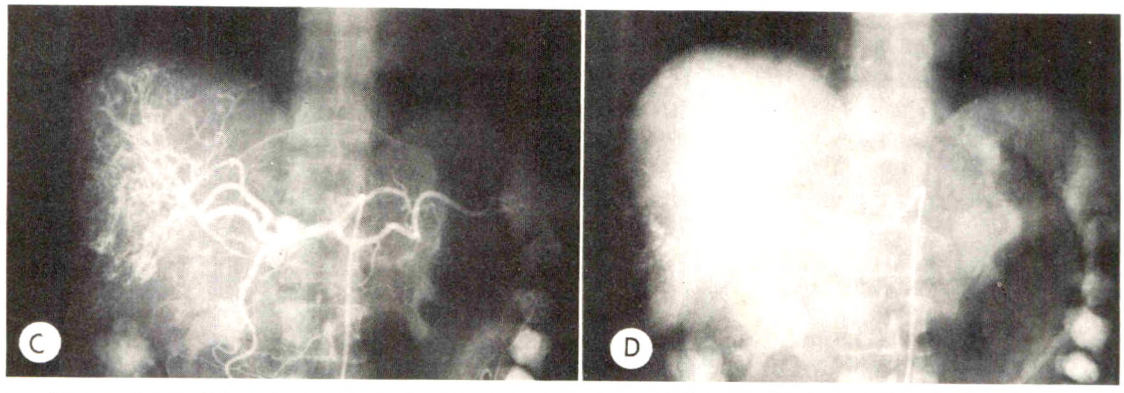


Fig. 1. (*C*) Celiac angiography, arterial phase, with early filling of diffuse, mottled vascular pools. (*D*) Celiac angiography, 30 seconds later, showing persistence of large vascular lakes.

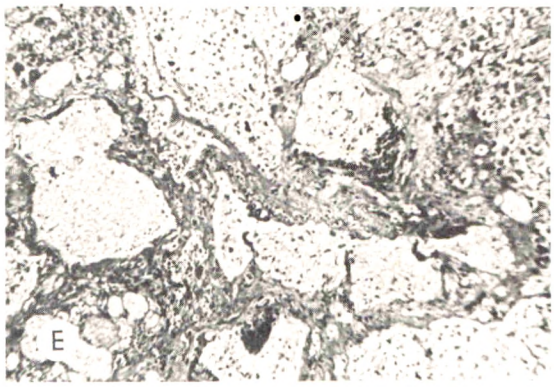


FIG. 1. (*E*) Photomicrograph, liver, showing large vascular lakes, malignant tumor cells and darkly stained thorotrast granules (Hematoxylin phloxine Safranin, ×100).

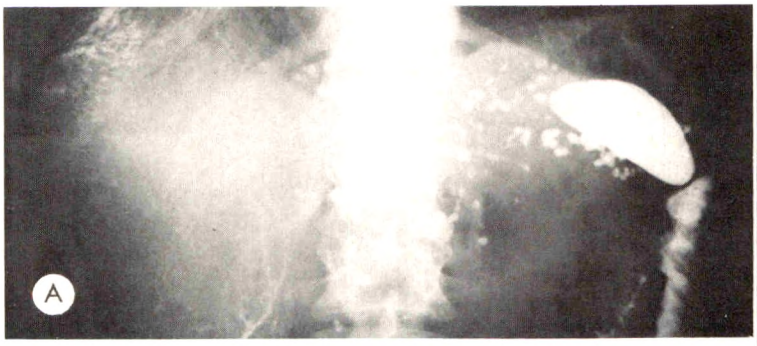
anaplastic, endothelial cells which also infiltrated supporting stroma (Fig. 1*E*). In many areas, the large blood pools were identifiable as markedly dilated sinusoids.

The spleen was grossly involved by hemorrhagic and necrotic tumor but lacked the gross, vascular pools seen in the liver. Microscopically, the splenic and the adrenal, kidney and bone marrow tumors all showed hemangioendothelioma. Refractile, yellowish-brown crystalline material consistent with thorotrast was found regional to neoplastic areas in the liver and bone marrow, but none was found within tumor cells.

CASE II. This 54 year old acutely ill, white, male patient was hospitalized with abdominal distention, hepatomegaly and weight loss. Roentgenologic studies showed lacey and reticular deposits of thorium in the liver with solid opacities in the spleen and regional lymph nodes, as well as interstitial densities in the area of the lower carotid sheath (Fig. 2, *A* and *B*). It was established that a thorotrast cerebral angiogram had been performed 28 years previously. The patient had been extensively studied elsewhere, three years previously for radiation activity in his tissues, and these findings were recorded. However, the patient seemed unaware of the potential, harmful effects of these thorotrast deposits, and this particular matter was not discussed with him.

Pertinent laboratory data included a hemoglobin of 10.6 milligrams percent, hematocrit 33 percent, white blood cell count, 6,900 per cubic millimeter with a normal differential white count, normal prothrombin time, alkaline phosphatase 650 units, LDH 320 units, and SGOT 150 units.

Celiac angiography supported the clinical suspicion of hepatic malignancy, showing large, diffuse vascular "lakes" which perfused early in the arterial phase and remained opacified for a period of 45 seconds (Fig. 2, *C* and *D*). The angiographic abnormalities were similar to those noted in Case 1, and suggested a diagnosis of hemangioendothelioma. Because of a falling hematocrit level and increasing abdominal fluid, hemoperitoneum was diagnosed and emergency laparotomy was performed in an effort to control the patient's bleeding. At surgery, 2,000 cc of bloody ascitic fluid was aspirated from the



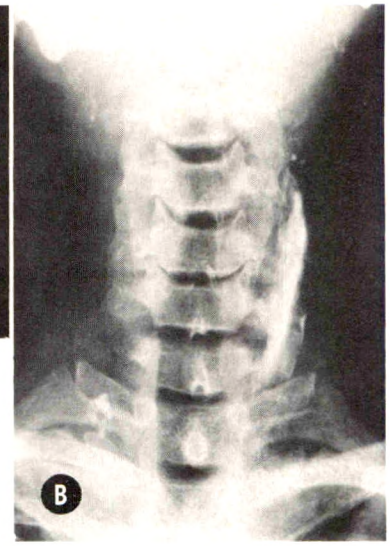


FIG. 2. Case II. (*A*) Abdominal roentgenogram showing reticular and linear thorium deposits in an enlarged liver with nodular densities in regional lymph nodes. The spleen is replaced by massive, solid thorium opacity. (*B*) Extravascular deposits of thorotrast in the carotid sheath related to percutaneous cerebral angiography, 28 years previously.

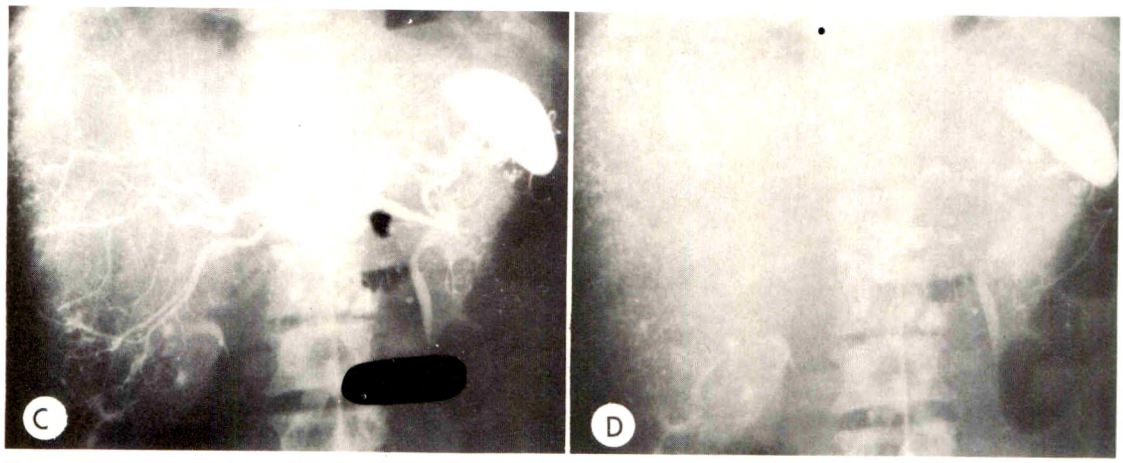


Fig. 2. (*C*) Celiac angiography showing early arterial filling of large vascular pools. (*D*) Celiac angiography showing persistent opaque medium in large vascular lakes 35 seconds later.

peritoneal cavity and an enlarged, firm lobular liver was seen. A large blood clot was adherent to the inferior aspect of the left lobe of the liver and marked the site of recent hemorrhage. A single biopsy was obtained from the anterior convexity of the central portion of the right lobe, and considerable difficulty was encountered in controlling bleeding. Postoperatively, the patient became progressively weaker as the ascites reaccumulated. The hemoglobin gradually fell to 5 grams, and the patient expired on the eighth postoperative day.

Histologically, the biopsy specimen showed extensive interstitial fibrosis with many macrophages containing granular material consistent with thorotrast, none of which was present within malignant cells. There was proliferation of elongated and spindled cells which outlined atypical vascular channels of varying size, lined by predominantly neoplastic endothelial cells, considered characteristic of hemangioendothelial sarcoma (Fig. 2*E*).

Permission for autopsy was not granted.

DISCUSSION

Over 120 thorium-related liver malignancies have been recorded, and almost 50 of these have been classified as hemangioendothelioma; the remainder being represented by hepatomas and cholangio-carcinomas.[10,20] In 1967, J. da Silva Horta,[5] in an extensive study of late thorotrast effects, emphasized the frequency of hemangioendothelioma in the thorotrast liver and its rarity in individuals who have not been injected with thorotrast. Recent epidemiologic studies by United States Public Health Agencies cite the potential, oncogenic hepatic effects of industrial polyvinyl chloride exposure, and also stress the rarity of spontaneous hepatic angiosarcoma.[22]

Although most recorded cases of intravascular, thorium-induced malignancy are hepatic, it has been noted that more detailed autopsy evaluation of bone marrow and other extrahepatic thorium deposition sites might uncover additional evidence of malignancy.[5] This impression is supported by autopsy findings in Case 1 of this report where microscopic hemangioendothelioma was found in bone marrow, adrenal and

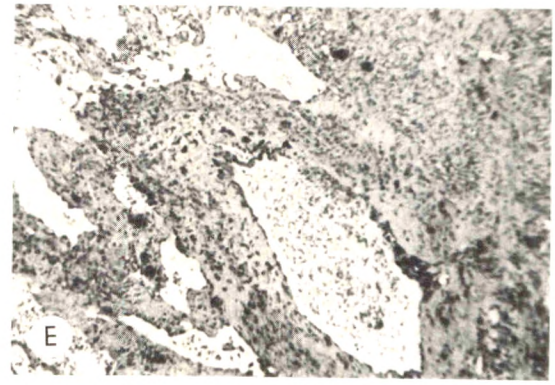


Fig. 2. (*E*) Photomicrograph of liver biopsy showing vascular lakes, malignant cells and darkly staining thorotrast granules (Hematoxylin phloxine Safranin, ×100).

kidney. In the bone marrow specimens, angiosarcomas were accompanied by regional thorium deposits which were not present within the malignant cells. This finding has raised speculation as to whether the extrahepatic malignancies represent metastases or tumors of multicentric origin.[5]

Large "blood lakes" have been described as a lacunar feature of hepatic hemangioendothelioma which confers a particular aspect to this tumor and may be seen macroscopically.[5] The large and persistent vascular pools shown in the angiograms of these two recorded cases would seem to reflect the roentgenologic counterpart of the pathologic descriptions (Fig. 1, *C–E*; and 2, *C–E*).

To our knowledge, angiography of the thorotrast liver before and after the clinical evolution of malignancy has not been recorded and may provide information of therapeutic and prognostic value. Since isolated cases of juvenile angiosarcoma of the liver have been cured by lobectomy, Smoron and Battifora[20] advocate a search for premalignant and early malignant changes in thorotrast livers. Others have also advocated a search for early diagnosis in the hope that local resection might offer a chance for surgical cure.[12,13,19] The possible value of intensive chemotherapy has also been suggested, before the occurrence of gross hepatomegaly and diffuse liver malignancy.[20]

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Appreciation is expressed to Wilfredo G. Salcedo, M.D., of the Department of Pathology, Easton Hospital, Easton, Pennsylvania, for valuable suggestions and advice.

Addendum: Since the completion of this report, we have identified two additional "thorotrast livers" on a survey of our roentgenographic files during the past 20 years. One of these was a male patient who was subjected to roentgen studies in 1964 and at that time was 56 years of age. His abdominal roentgenograms are highly compatible with previous thorotrast injection but he was an itinerant patient and has been lost to follow-up. The other patient, a 53 year old female, was first examined at Easton Hospital several months ago because of minor gastrointestinal symptoms. She was exposed to a thorotrast cerebral angiogram at age 20 years and now seems to be in good clinical health. This patient has not yet been informed of her thorium deposits for reasons already discussed in Case 1 of this report, and this matter is now under consideration.

REFERENCES

1. Barry, W. J., and Rominger, C. J. Thorotrast granulomas. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1954, *92*, 584–590.
2. Blomberg, R., Larson, I. E., Lindell, B., and Lindgren, F. Late effects of thorotrast in cerebral angiography. *Acta radiol.* (Diag.), 1963, *1*, 995–1006.
3. Boyd, J. T., Langlands, A. O., and MacCabe, J. J. Long term hazards of thorotrast. *Brit. M. J.*, 1968, *2*, 517–521.
4. Dahlgren, S. Thorotrast tumours: review of literature and report of two cases. *Acta path. et microbiol. scandinav.*, 1961, *53*, 147–161.
5. da Silva Horta, J. Late effects of thorotrast on liver and spleen and their efferent lymph nodes. *Ann. New York Acad. Sc.*, 1967, *145*, 676–699.
6. Editorial. Council on pharmacy and chemistry: thorotrast. *J.A.M.A.*, 1932, *99*, 2183–2185.
7. Feldman, F., Seaman, W. B., and Wells, J. S. Residual thorotrast in paranasal sinuses: study of its roentgen appearance and implications. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *89*, 1147–1154.
8. Heger, N., and Bayindir, S. Thorotrast: primary liver sarcoma, 22 years after administration. *Minnesota Med.*, 1970, *53*, 1137–1139.
9. Hursh, J. B., Stedman, I. T., Looney, W. G., and Colodzin, M. Excretion of thorium and thorium daughters after thorotrast administration in human subjects. *Acta radiol.*, 1957, *47*, 481–498.
10. Janower, L. J., Miettinen, O. S., and Flynn, M. J. Effects of long-term thorotrast exposure. *Radiology*, 1972, *103*, 13–20.
11. Jahn, D. Thorotrast induced hemangioendothelioma of liver (case with thirty year latent period.) *Connecticut M. J.*, 1967, *31*, 557–560.
12. Kiely, J. M., Titus, J. L., and Orvis, A. J. Thorotrast induced hepatoma presenting as hyperparathyroidism. *Cancer*, 1973, *34*, 1312–1314.
13. Kruckemeyer, H. D., Lessman, H. D., and Pudwitz, K. R. Renal carcinoma due to injury by thorotrast. *Fortschr. a. d. Geb. d. Röntgenstrahlen. u. d. Nuklearmedizin*, 1960, *93*, 313–321.
14. Kuisk, H., Sanchez, J. A., and Mizumo, N. S. Colloid thorium dioxide (thorotrast) in radiology with emphasis on hepatic cancerogenesis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 463–475.
15. Latourette, H. B. Editor's comment. Yearbook

of Radiology. Year Book Medical Publishers, Inc., Chicago, 1973, p. 435.
16. Levowitz, B. S., Hughes, R. E., and Alford, T. C. Treatment of thorium dioxide granulomas of neck. *New England J. Med.*, 1963, *268*, 340–342.
17. Looney, W. B. Investigation of late clinical findings following thorotrast (thorium dioxide) administration. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *83*, 163–185.
18. MacMahon, H. F., Murphy, A. S., and Bates, M. I. Endothelial cell sarcoma of liver following thorotrast injections. *Am. J. Path.*, 1947, *23*, 585–611.
19. Person, D. A., and Sargent, I. Thorotrast induced carcinoma of liver. *A.M.A. Arch. Surg.*, 1964, *88*, 503–510.
20. Smoron, G. L., and Battifora, H. A. Thorotrast induced hepatoma. *Cancer*, 1972, *30*, 1252–1259.
21. Suchow, E. E., Henegar, G. C., and Boserga, B. Tumors of liver following administration of thorotrast. *Am. J. Path.*, 1961, *38*, 663–664.
22. Thomas, L. B., Popper, H., Berk, P. D., Jelikoff, I., and Falk, H. Vinyl chloride induced liver disease. *New England J. Med.*, 1975, *292*, 17–22.
23. Verhaak, R. L. O. M., Harmsen, A. E., and van Unnik, A. J. M. On frequency of tumor induction in thorotrast kidney. *Cancer*, 1974, *34*, 2061–2068.
24. Westin, J., Lanner, O., Weinfeld, A., and Zettergren, L. Renal pelvic carcinoma after thorotrast pyelography. *Acta med. scandinav.*, 1973, *194*, 141–143.

INTRATHORACIC KIDNEY*

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ABSTRACT:

Two cases of ectopic, partly intrathoracic kidneys are discussed. One case represents bilateral ectopy, an extremely rare condition. This anomaly deserves consideration because a correct diagnosis can save the patient extensive examinations and possibly thoracic surgery.[1]

FULLY or partly ectopic intrathoracic kidney is a very rare developmental anomaly. In 15,919 autopsies of children Campbell[3] found 22 cases of ectopic kidney, and only one was an intrathoracic kidney. Burke *et al.*[2] compiled a list of 31 described cases of intrathoracic kidney. The left kidney was most commonly ectopically situated, and no case of bilateral ectopic kidneys was described. The sex of the patients was known in 17 of these cases: 15 were male. The intrathoracic kidneys were asymptomatic and in all cases their function was unimpaired. Wolfromm,[10] in 1940, was the first to describe an intrathoracic kidney in a living patient. On a bilateral retrograde pyelogram he found an asymptomatic intrathoracic kidney on the right side.

Spillane and Prather[8] postulated three criteria of congenital intrathoracic kidney: (1) rotation anomaly; (2) long ureter; and (3) abnormally high derivation of the vessels of the kidney. Gondos[6] remarked that in addition to these three criteria there will be a medial deviation of the lower pole of the kidney.

Differentiation must be made between congenital ectopy and cranial displacement of the kidney combined with eventration of the diaphragm. In congenital ectopy the ascent of the kidney, which in early fetal life develops in the pelvis, has continued cranially and passed the diaphragm which closes below or around the kidney. In cranial displacement there is a possibility of a developmental anomaly of the diaphragm adjacent to the adrenal gland and the upper pole of the kidney, so that the diaphragm closes around these organs, or there may be a weakening resulting in eventration of the diaphragm. Both conditions can be simulated by a traumatic diaphragmatic rupture.

REPORT OF CASES

CASE I. A male, aged 62 years, with a history of duodenal ulcers in 1932 and 1941; thereafter no abdominal discomfort was noted. In 1965, he had had a fracture of the cervical spine in a traffic accident, in which there was no known trauma to the chest or the abdomen. In 1972 he had attacks of cardiac arrhythmias for which he was treated in the Cardiology Department. A chest roentgenogram showed a tumor situated in the base of the right thorax (Fig. 1, *A* and *B*). On an intravenous pyelogram, this tumor was shown to be a partly intrathoracic kidney (Fig. 1*C*). On the same study an additional sign was found indicating a left-sided suprarenal tumor. Renal angiography confirmed the right-sided finding and showed a partly intrathoracic kidney supplied by two arteries on the left (Fig. 1, *D* and *E*). On both sides the renal arteries were shown to leave the aorta at the normal level. On the left side the abnormal location of the kidney was best seen during deep inspiration. The renal pelves on both sides showed no pathological changes. The ureters were displaced distally and bilaterally by an enlarged prostate which made an impression in the urinary bladder.

CASE II. A boy, aged five years, was examined in the Pediatric Clinic because of a cardiac murmur, first noticed at the age of one year.

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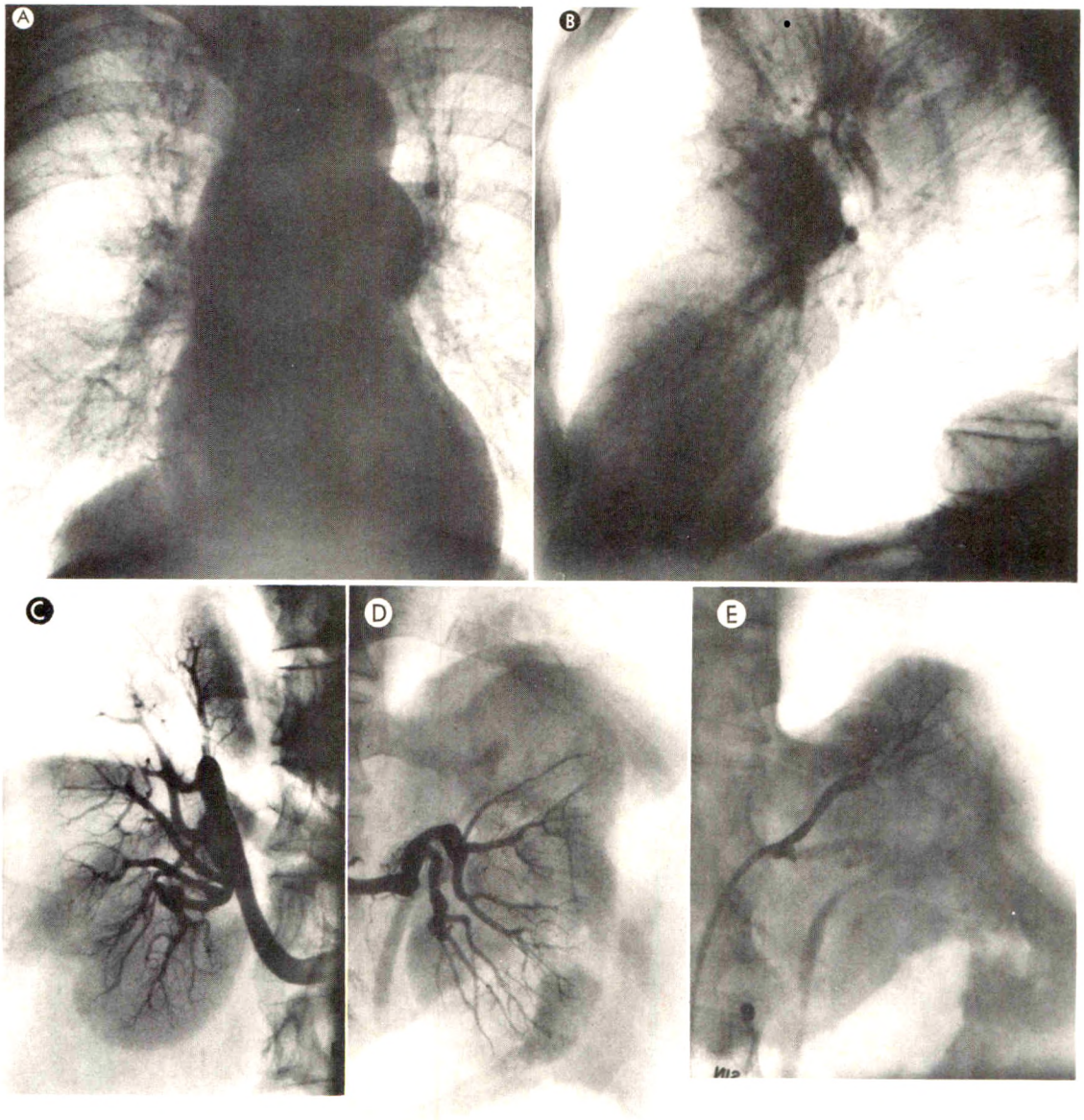

Fig. 1. Case 1. Sixty-two year old male. (*A*) Posteroanterior and (*B*) lateral chest roentgenograms show bilateral, well defined medial mass lesions posteriorly at the diaphragms. (*C*) Selective right renal angiography demonstrates the kidney to extend above the right hemidiaphragm. (*D* and *E*) Selective angiography of the two arteries to the left kidney show the upper pole extending above the left hemidiaphragm.

The examination showed an acyanotic tetralogy of Fallot.

A roentgenogram of the chest showed a tumor at the base of the left lung and a small herniation of the stomach (Fig. 2, *A* and *B*). The tumor was shown on intravenous pyelography to be an ectopic left kidney, the greater part of which was displaced intrathoracically (Fig. 2, *C* and *D*). The morphology of the pelvis was normal. There were a long ureter and a medial deviation of the lower pole but no malrotation of the kidney. The patient had no symptoms from his left ectopic kidney.

DISCUSSION

Two cases of ectopic, partly intrathoracic kidneys are described. One case is a left-sided ectopy in a five year old boy, the other a bilateral ectopy in a 63 year old

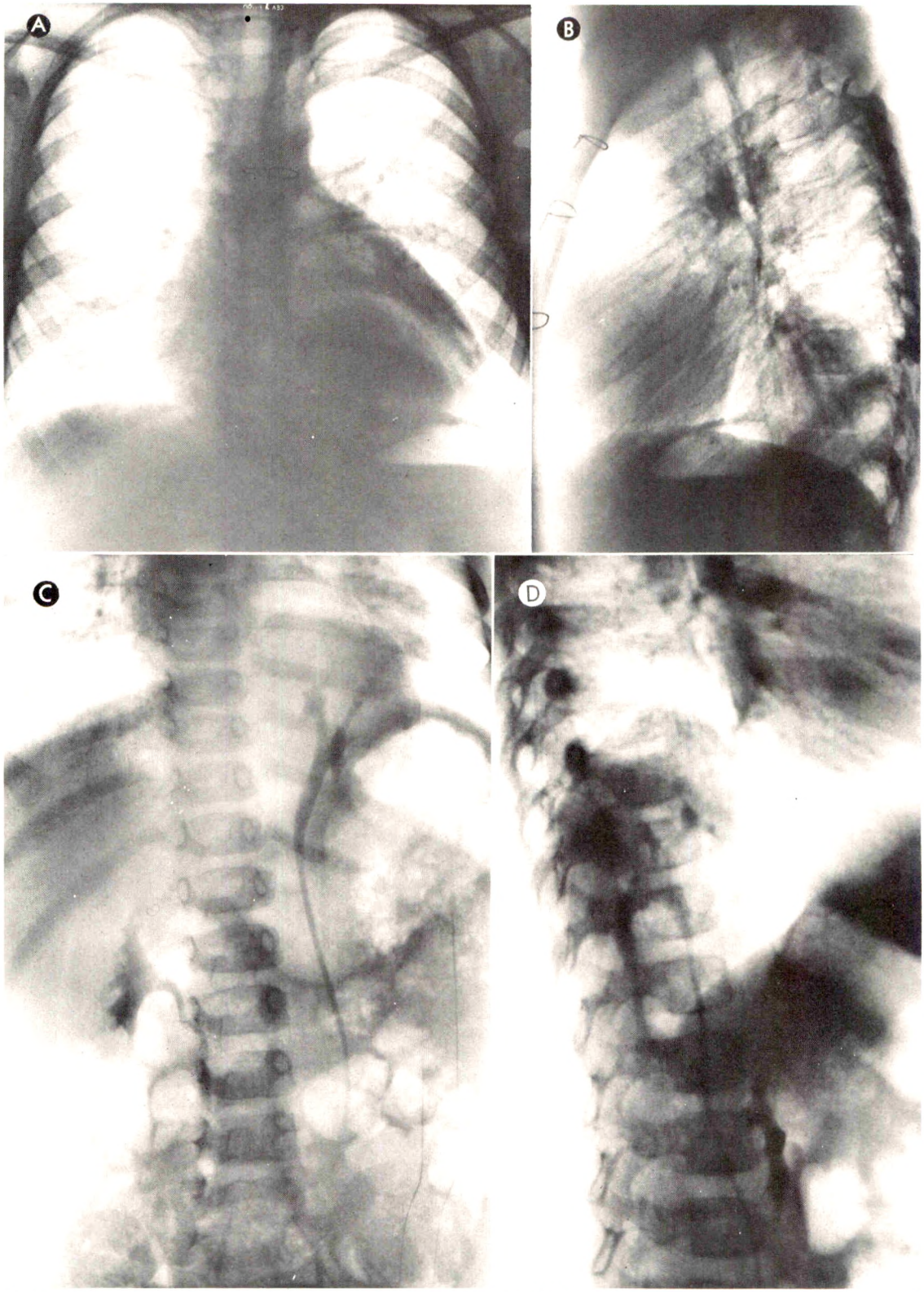

FIG. 2. Case II. Five year old boy. (*A*) Posteroanterior and (*B*) lateral chest roentgenograms show a well defined medial mass lesion posteriorly on the left side. Intravenous pyelogram, (*C*) anteroposterior and (*D*) lateral views, demonstrate the high position of the left kidney and the long ureter.

man. In neither of the cases did trauma cause rupture of the diaphragm.

The application of diagnostic pneumothorax has been described in the literature several times as a method for establishing differential diagnosis between diaphragmatic hernia and eventration. We have considered it inadvisable to use this method in these patients because the ectopic kidneys were asymptomatic.

In one of the cases an aortogram showed the renal arteries leaving the aorta at a normal level. The arteries were duplicated on the left side. However, there was no malrotation of the kidneys. Ectopic, partly intrathoracic kidneys can be very difficult to differentiate by chest roentgenography from diaphragmatic tumors, usually lipomas,[9] whereas intravenous pyelography can establish the differential diagnosis.

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REFERENCES

1. Ang, A. H., and Chan, W. F. Ectopic thoracic kidney. *J. Urol.*, 1972, *108*, 211–212.
2. Burke, E. C., Wenzel, J. E., and Utz, D. C. Intrathoracic kidney: report of case. *A.M.A. Am. J. Dis. Child.*, 1967, *113*, 487–490.
3. Campbell, M. F. Renal ectopy. *J. Urol.*, 1930, *24*, 187–189.
4. DeCastro, F. J., and Schumacher, H. Asymptomatic thoracic kidney. *Clin. Pediat.*, 1969, *8*, 279–280.
5. Fleischner, F., Robins, S., and Abrams, M. High renal ectopia and congenital diaphragmatic hernia. *Radiology*, 1950, *55*, 24–26.
6. Gondos, B. High ectopy of left kidney. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1955, *74*, 295–298.
7. Spillane, R. J., and Prather, G. C. High renal ectopy: case report. *J. Urol.*, 1949, *62*, 441–445.
8. Spillane, R. J., and Prather, G. C. Right diaphragmatic eventration with renal displacement. *J. Urol.*, 1952, *68*, 804–806.
9. Weilgoni, M. Zwerchfell Lipom. *Der Radiologe*, 1963, *3*, 401–404.
10. Wolfromm, G. Position of kidney in right diaphragmatic eventration. *Mém. Acad. chir.*, 1940, *66*, 41–47.

CAVERNOUS HEMANGIOMA OF THE KIDNEY*

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ABSTRACT:

Three patients with cavernous hemangiomas of the kidney all presented with hematuria. In each there was found a poorly defined renal medullary mass with displacement of the calyces and renal vessels and paradoxical hypovascularity of the mass. This rare, benign tumor of the kidney should be considered in the presence of this characteristic clinical picture.

IN September, 1973, two of the authors (E.S. and R.J.S.) reported the first known case of cavernous hemangioma of the kidney which presented angiographically as a hypovascular, poorly defined mass in the medulla.[4] Since then two similar cases of this tumor have come to our attention. The striking angiographic similarities of these three cavernous hemangiomas of the kidney suggest that the combination of clinical and roentgenographic features which were found may be characteristic of this lesion and prompt us to describe this entity in further detail.

REPORT OF CASES

CASE I. The clinical features of Case 1 are described in detail in the previous report.[4] In summary, a 23 year old female experienced the sudden onset of right flank pain two weeks prior to admission and severe hematuria during the seven days prior to admission. Initial evaluation by excretory urography and nephrotomography showed deformity of the upper calyx and filling defects within the renal pelvis presumed to be blood clots. A selective renal arteriogram (Fig. 1, *A* and *B*) demonstrated some separation of the upper pole intrarenal arteries, diminished arterial branching and an ill-defined hypovascular area in the medullary portion which produced slight overall increase in the size of the upper pole. Due to continued bleeding a nephrectomy was performed.

CASE II. An eleven year old girl was admitted to St. Louis Children's Hospital in November, 1974, with a one week history, of gross, painless hematuria unaccompanied by fever or other significant signs or symptoms. There was no prior history of urinary tract disease. An excretory urogram demonstrated a normal appearing left kidney and left collecting system. On the early roentgenograms (Fig. 2*A*) the contour of the enlarged right upper pole was poorly defined due to diminished contrast density, while the lower pole was clearly defined. The upper pole calyces were not visualized and a filling defect, presumably a blood clot, was present within the renal pelvis. Delayed roentgenograms (Fig. 2*B*) revealed an "obstructive nephrogram" appearance of the upper and mid portion of the right kidney and a poorly defined area of central lucency. A renal sonogram (Fig. 3, *A* and *B*) demonstrated enlargement of the right kidney with multiple echoes arising from the central or medullary portion of the kidney as well as from the area of the collecting system. A selective renal arteriogram (Fig. 4, *A* and *B*) showed separation and diminished branching of the intralobar arteries very similar to the pattern seen in the earlier case. A few minute tortuous and irregular vessels were visible in the central area of the upper pole which otherwise remained hypovascular. The appearance suggested a centrally expanding process, a pattern sometimes seen in hydronephrosis. In the nephrogram phase, the corticomedullary junction was poorly defined and the somewhat lucent medullary area appeared to be enlarged. No abnormal venous drainage was seen.

The clinical presentation and the roentgenographic features were considered to be sufficiently similar to the previous case that a pre-

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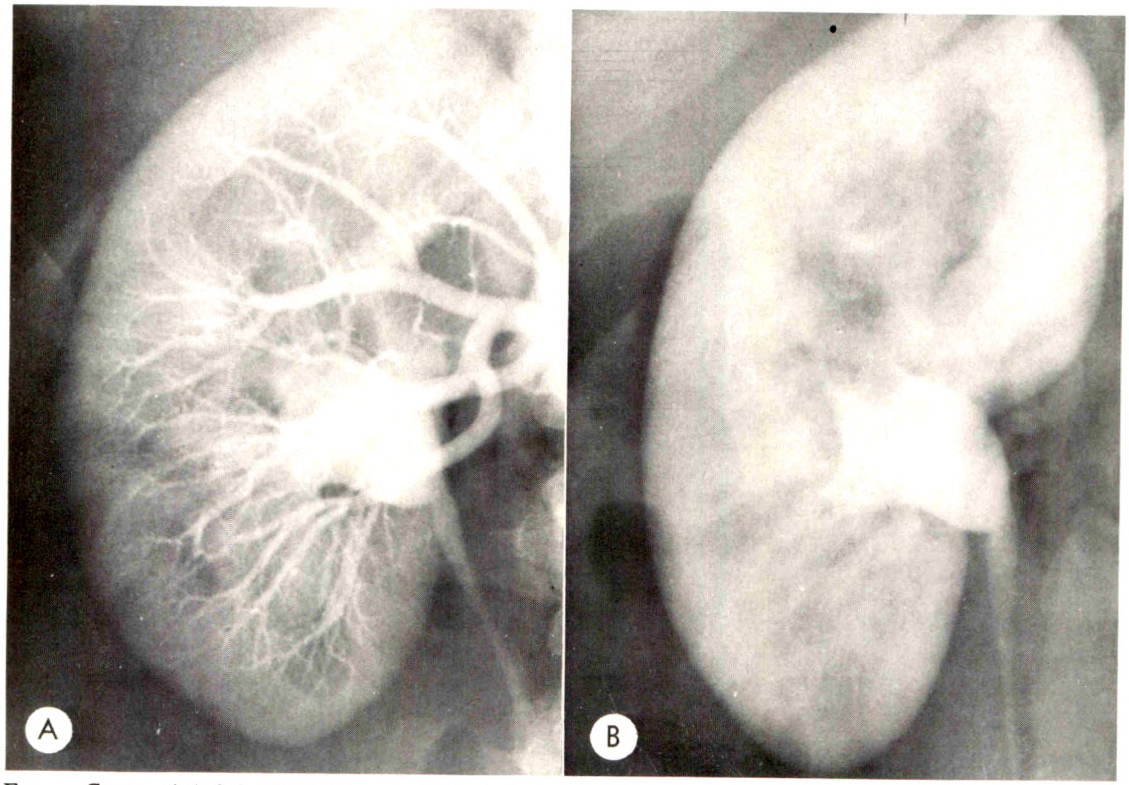

Fig. 1. Case 1. (*A*) Selective right renal arteriogram, anteroposterior, arterial phase. Segmental branches to the upper pole are separated and slightly bowed. Branching of these vessels in the medulla is diminished compared to the lower pole arteries. (*B*) Selective right renal arteriogram, right posterior oblique, capillary phase. Poorly marginated, hypovascular area is noted in center of uniformly enlarged upper pole. A distorted calyx included in the area is faintly visible. The corticomedullary junction is not visible in the upper pole. (Reproduced with permission from the *Journal of the Canadian Association of Radiologists*.)

operative diagnosis of cavernous hemangioma was offered. A nephrectomy was performed.

Case III. A 32 year old white female was admitted to the Harrisburg Polyclinic Hospital in September, 1970, for evaluation of intermittent total gross, painless hematuria since the age of 16. During the 16 year interval, numerous uroroentgenologic examinations had failed to define a specific lesion. On each endoscopy, blood was seen to emanate from the left ureteral orifice.

A left retrograde pyelogram (Fig. 5) demonstrated extrinsic pressure on the infundibulum and calyces of the upper pole. In addition, a "calyceal diverticulum" was also noted. A left selective renal arteriogram (Fig. 6) showed stretching and diminished branching of the arteries in the upper half of the kidney similar to Case I, and II. The significance of these findings was not initially appreciated and the discovery of a large cavernous hemangioma at the time of nephrectomy was unexpected.

Pathologic Findings

Gross examination of the three nephrectomy specimens revealed spongy hemorrhagic lesions primarily located in the medullary regions. In Case I the tumor measured 4×3 cm and surrounded the upper pole calyces and infundibulum. The lesions in Case II (Fig. 7) and Case III were very extensive, involving almost the entire medullary portions of the kidneys. There was also focal involvement of the cortex in Case II. Microscopically, the three tumors were composed of large, tortuous anastomosing vascular channels characteristic of cavernous hemangiomas. In some areas of the tumor in Case II, it appeared that the hemangioma was actually arising from the

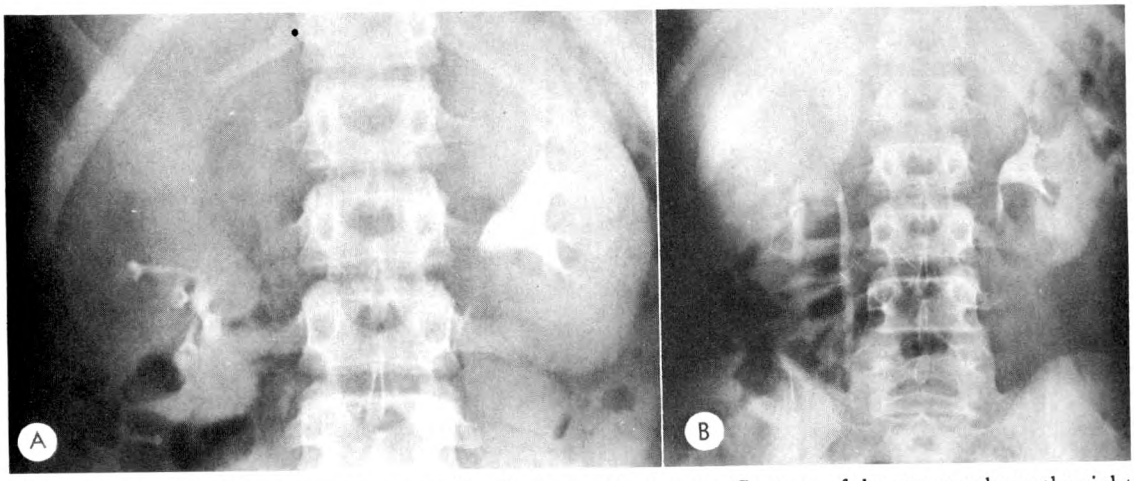

Fig. 2. Case II. (*A*) Excretory urogram, eight minute roentgenogram. Contour of the upper pole on the right is poorly defined due to over-all diminished contrast medium density. Right renal pelvis is deformed and the calyces to the upper pole are not visualized. Left renal contour and collecting system appear normal. (*B*) Thirty minute roentgenogram from same excretory urogram. The contour of the enlarged right upper pole is clearly defined. Upper and mid portion of right kidney now have an "obstructive nephrogram" appearance. Large filling defect within renal pelvis is produced by blood clot. A poorly defined area of lucency is visible in the center of the expanded upper pole.

arcuate arteries (Fig. 8) and, in addition, organized thrombi were frequently seen.

ROENTGENOLOGIC FINDINGS

The roentgenographic features common to these three cases include: (1) the presence of an intramedullary mass effect producing varying degrees of calyceal deformity; (2) slight bowing and separation as well as diminished branching of the intrarenal arteries throughout the lesion; (3) hypovascularity and over-all diminished contrast medium density compared to the surrounding normal parenchyma; and (4)

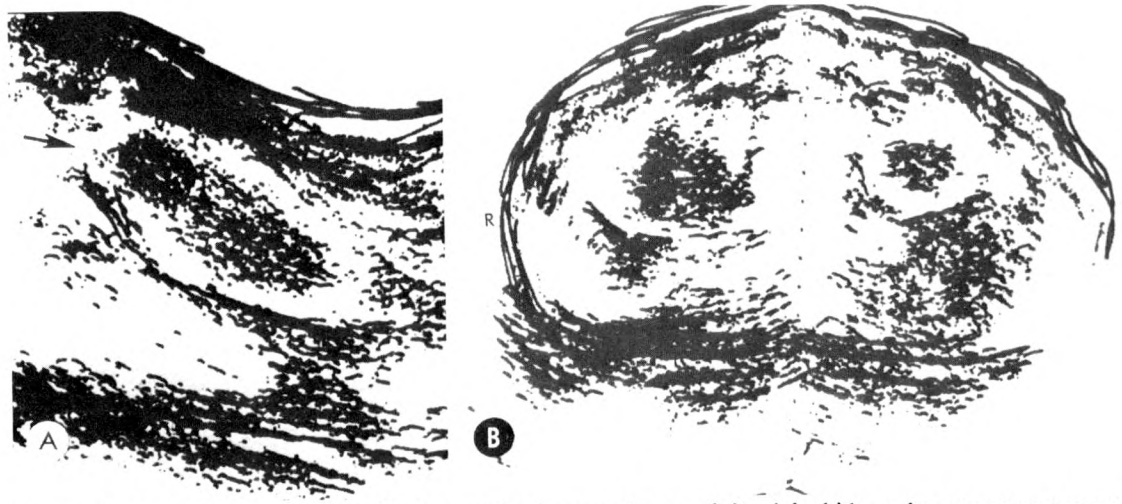

Fig. 3. Case II. (*A*) Prone, longitudinal B mode ultrasound scan of the right kidney demonstrates a prominent sonographic appearance of the collecting system and a solid mass lesion in the upper pole (arrow). Head is to the left. (*B*) Prone, transverse B mode ultrasound scan at the level of the right upper pole. Enlargement of right upper pole is apparent. Solid mass lesion produces dense echo pattern in the area of the medulla and collecting system.

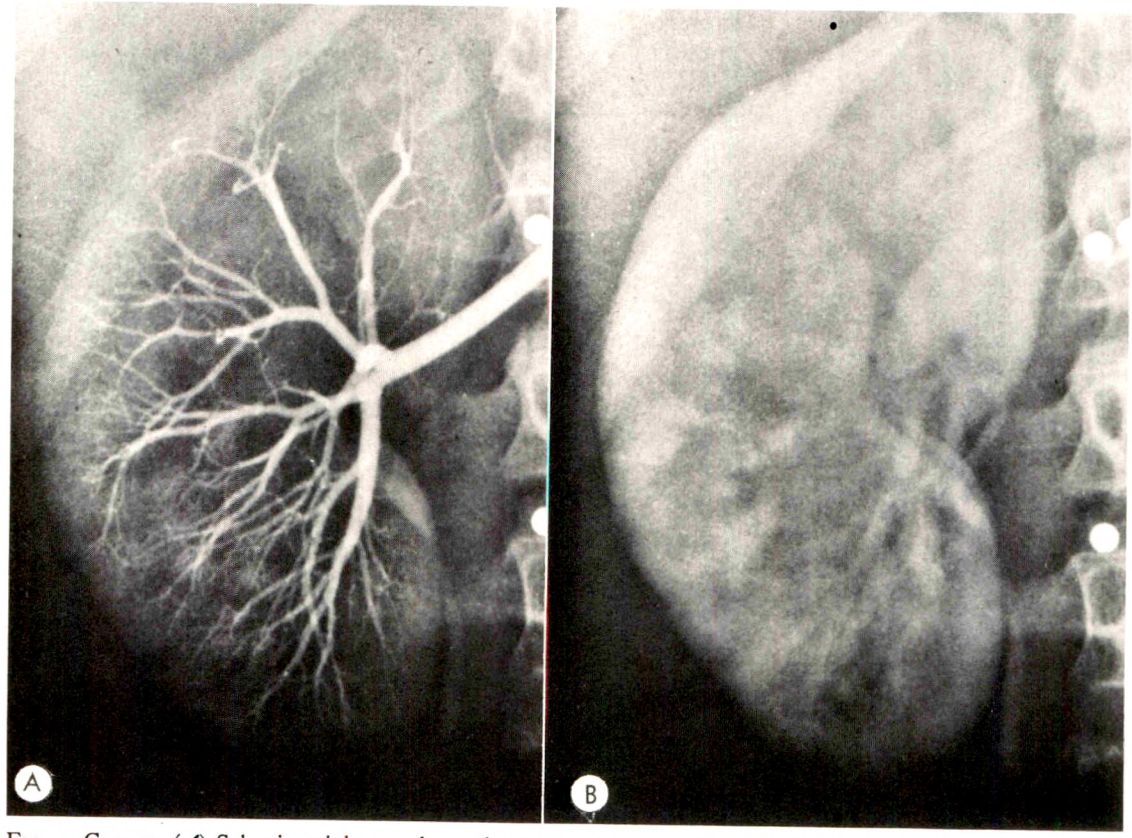

Fig. 4. Case II. (*A*) Selective right renal arteriogram, anteroposterior, arterial phase. Segmental arteries to upper half of kidney are separated and distorted. Diminished and abnormal branching is apparent. (*B*) Selective right renal arteriogram, anteroposterior, capillary phase. Poorly defined area of central lucency is present in the upper and mid zone of the expanded right kidney. No abnormal collections of contrast agent or anomalous draining veins are evident.

poor definition of the margins of the mass. Not seen in any of the three cases were focal collections of contrast medium within the mass, appearing early and persisting well beyond the venous phase of the angiogram. This latter appearance has been described in cavernous hemangiomas of the liver,[1,6,7] as well as in one of the two cases of renal cavernous hemangiomas illustrated by Evans and Bosniak.[5] In addition, Case II exhibited an "obstructive nephrogram" in the upper half of the affected kidney and more obvious over-all enlargement of the upper and mid portion of the kidney. It is presumed that the expanding mass itself produced the partial obstruction resulting in the unusual obstructive nephrogram and the lack of calyceal visualization. The renal sonogram further established the solid nature of the lesion in Case II and eliminated the possibility that this was simply a fluid filled cyst or isolated hydrocalyx.

DISCUSSION

The renal hemangioma is a rarely occurring benign tumor. Only two renal hemangiomas have been evaluated and diagnosed by the Surgical Pathology Department of Washington University Medical Center. The autopsy studies of Bell[3] and Riley and Swann[8] found one renal hemangioma in a combined total of over 43,000 autopsies.

In 1964 Andersen and Rasmussen[2] reported a case of a hemangioma of the renal pelvis which was highly vascular on selective arteriography. The lesion consisted of numerous, closely adjacent, coiled blood

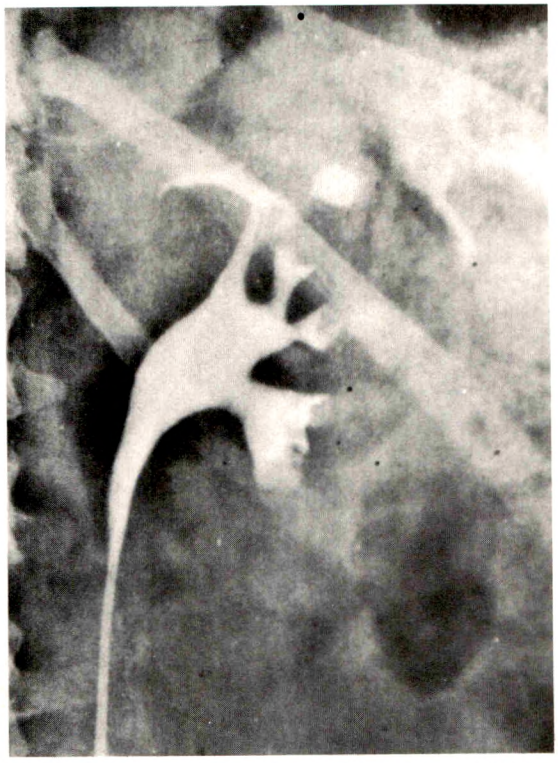

Fig. 5. Case III. Left retrograde pyelogram. Calyces in the upper pole are distorted. Margins of kidney are not well seen but upper pole appears enlarged. An incidental finding is a "calyceal diverticulum."

vessels causing a dense accumulation of the contrast medium as well as delayed emptying compared with the surrounding normal intrarenal vessels. Their preoperative diagnosis was renal hemangioma. Evans and Bosniak[5] illustrate two renal hemangiomas, one being the capillary form and very similar to the case of Andersen and Rasmussen.[2] The other, presenting as an unevenly hypovascular mass, was described as a venous hemangioma. While bearing striking similarities to the three cases reported here, it differed in that there were focal collections of contrast medium within the radiolucent mass representing contrast within the cyst-like venous lakes of the hemangioma. A large surface vein was seen to drain the hemangioma as well.

It is not clear why this histologically vascular tumor presented as a hypovascular mass in these three cases. Thrombosed channels, noted grossly and histologically within the tumor in Case II, could account for some of this appearance. A large, slowly flowing blood pool within the tumor might be poorly opacified by a bolus injection. Conceivably, a prolonged slow arterial injection and selective venography could augment the findings obtained by the standard technique.

While the above described roentgenographic and ultrasound features are not pathognomonic of a cavernous hemangioma of the kidney, the unusual findings are sufficiently different from other benign and malignant renal neoplasms, renal cysts, and other less common inflammatory processes that the demonstration of these findings

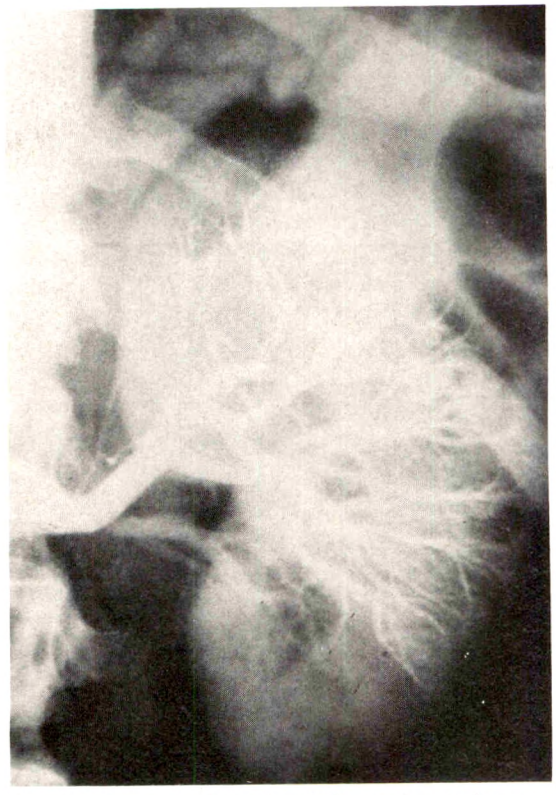

Fig. 6. Case III. Selective left renal arteriogram, arterial phase. Segmental arteries to the upper pole appear separated and diminished branching pattern, similar to Cases I and II is present. Incomplete filling of early branching artery to lower pole accounts for lack of arterial opacification. Center of upper half of kidney appeared relatively lucent during capillary phase (not shown).

should cause the examiner to strongly consider the possibility of a cavernous hemangioma and to certainly include it in the differential diagnosis. Hypovascular cystic malignancies as well as other hypovascular infiltrating renal tumors, including metastatic lesions could present in the same fashion. Of course, a history of intermittent hematuria over many years in a young person would be less consistent with a malignant process.

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The authors wish to express their thanks to Howard M. Pollack, M.D., Episcopal Hospital, Philadelphia, Pennsylvania for calling our attention to Case III and to Howard C. Beane, M.D., of the Harrisburg Polyclinic Hospital, Harrisburg, Pennsylvania for permission to report Case III.

Fig. 7. Gross specimen of Case II. Bi-valved kidney showing an extensive spongy and hemorrhagic tumor located in the entire medulla and encroaching upon the collecting system. Focal extensions into the cortex are also seen.

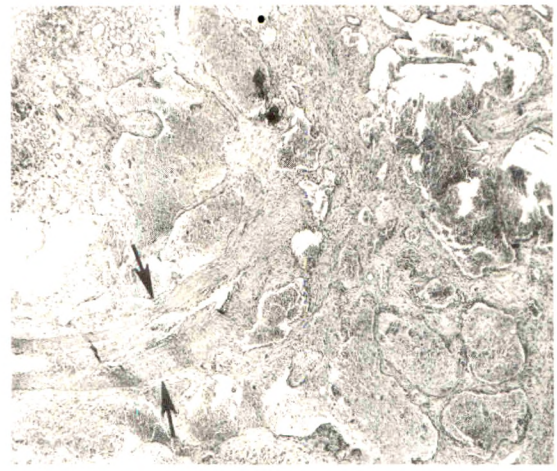

Fig. 8. Case II. Photomicrograph illustrating replacement of the renal medulla by large anastomosing vascular channels filled with blood. At the left (arrows) there is an arcuate artery in intimate relationship with the abnormal blood vessels (H and E ×40).

REFERENCES

1. Abrams, R. M., Beranbaum, E. R., Santos, J. S., Lipson, J. Angiographic features of cavernous hemangioma of liver. *Radiology*, 1969, *92*, 308–312.
2. Andersen, J. B., and Rasmussen, T. Renal hemangioma diagnosed preoperatively by selective renal angiography. *Acta radiol. (Diag.)*, 1964, *2*, 201–204.
3. Bell, E. T. Classification of renal tumors, with observations of frequency of various types. *J. Urol.*, 1938, *39*, 238–243.
4. Cubillo, E., Hesker, A. E., and Stanley, R. J. Cavernous hemangioma of kidney: angiographic-pathologic correlation. *J. Canad. A. Radiologists*, 1973, *24*, 254–256.
5. Evens, J. A., and Bosniak, M. A. Atlas of Tumor Radiology; the Kidney. Year Book Medical Publishers, Inc., Chicago, 1971, pp. 278–289.
6. Pantoja, E. Angiography in liver hemangioma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 874–879.
7. Pollard, J. J., Nebesar, R. A., and Mattoso, L. F. Angiographic diagnosis of benign diseases of liver. *Radiology*, 1966, *86*, 276–283.
8. Riley, A., and Swann, W. J. Angioma of kidney. *Urol. & Cutan. Review*, 1941, *45*, 377–382.

RADIOLOGIC FEATURES OF THE INTERURETERIC RIDGE*

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ABSTRACT:

Careful attention to the dimensions and symmetry of the interureteric ridge, when it is visible, aids in interpretation of abnormalities in the bladder and ureters. It is important in evaluating renal agenesis, low ureteral calculi, trauma, and neoplasm.

THE ureteral orifices of the urinary bladder are separated by a gently curved bundle of non-striated muscle, the *torus uretericus*. This muscle bundle actually extends slightly lateral to each ureteric orifice; this lateral extension is named the *plica uretericae*. Commonly, and for purposes of this report, both the *torus uretericus* and *plica uretericae* are collectively referred to as the "interureteric ridge." The interureteric ridge thus encompasses the distal ends of the ureters and forms the boundary of the widest portion (base) of the trigone of the urinary bladder.

Recent uroradiological texts have published roentgenograms of the interureteric ridge visible in its normal state on routine urograms or cystograms.[2,3] However, it is not widely appreciated that roentgenologically detectable abnormalities in this structure may provide useful diagnostic information particularly with reference to acutely obstructing ureteral calculi. Several cases are presented in this report as illustrative examples.

REPORT OF CASES

CASE I. Figure 1 is a roentgenogram exposed with dilute contrast medium in the urinary bladder immediately following bilateral retrograde pyelography. The normal left side of the interureteric ridge can be seen as a radiolucent bar extending from the midline to the left lateral, superior aspect of the bladder. On the right, this bar is widened and ends in a large, stellate, filling defect. The right lateral aspect of the bladder is irregular compared with the smoothly and sharply marginated left side. The right ureter is obstructed.

In this instance these changes are due to marked edema of the ureteric orifice and adjacent interureteric ridge secondary to ureteral catheterization. A similar picture might be seen with an acutely obstructing ureteral stone or a tumor in the area of the right side of the trigone.

CASE II. A middle aged male presented with acute left-sided flank pain and hematuria. A supine roentgenogram made 30 minutes after injection of 100 mls of contrast medium shows an obvious horseshoe kidney (Fig. 2*A*). The nephrogram of the left kidney is intense and the calyces are dilated. The normal right interureteric ridge can be seen as a well defined lu-

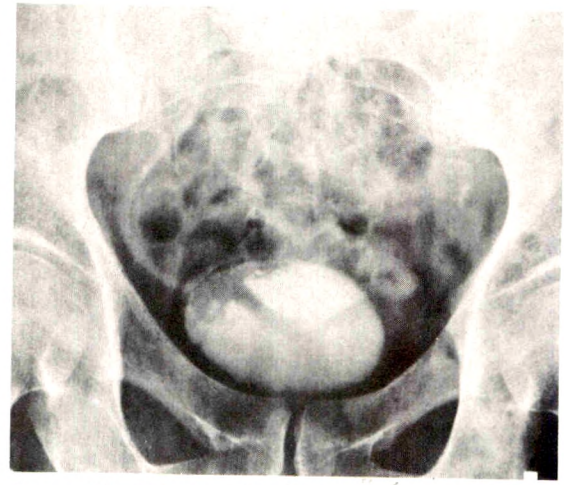

FIG. 1. The left side of the interureteric ridge is normal. The right lateral aspect of the ridge is effaced by a large irregular area of edema.

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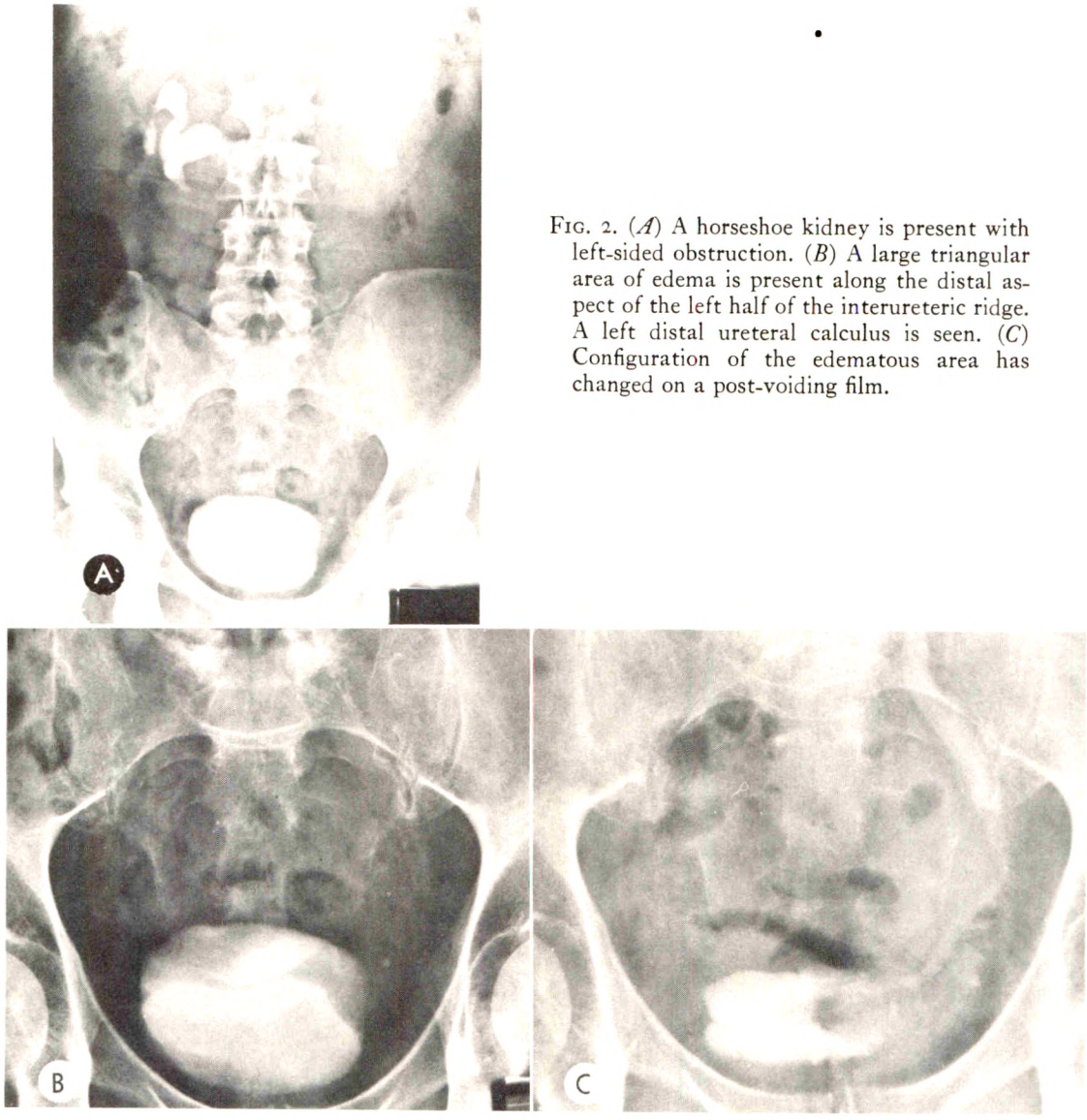

Fig. 2. (*A*) A horseshoe kidney is present with left-sided obstruction. (*B*) A large triangular area of edema is present along the distal aspect of the left half of the interureteric ridge. A left distal ureteral calculus is seen. (*C*) Configuration of the edematous area has changed on a post-voiding film.

cent bar. The medial portion of the left interureteric ridge is normal but the more lateral aspect is obscured by a large, well-marginated, triangular filling defect in the region of the left ureteral orifice and distal portion of the interureteric ridge representing marked edema in this area. These findings in the urinary bladder are better appreciated on a coned view of the pelvis, where a small opaque calculus can also be seen just outside the left lateral aspect of the bladder in the distal left ureter (Fig. 2*B*).

Figure 2*C* is a supine coned view of the urinary bladder in the same patient after voiding. In this state, the changes in the left interureteric ridge are manifest by a rounded, poorly-marginated filling defect in the left side of the bladder. Again, the normal right side is seen as a lucent bar extending to the right side of the bladder.

Case III. A young male presented himself to the emergency room with acute onset of right flank pain and microhematuria. A post-voiding supine roentgenogram performed one hour after injection of contrast medium shows an intense nephrogram on the right and a slightly dilated right ureter visible to the lumbosacral junction (Fig. 3*A*). A coned view of the bladder shows conspicuous nodularity and irregularity of the right side of the bladder representing

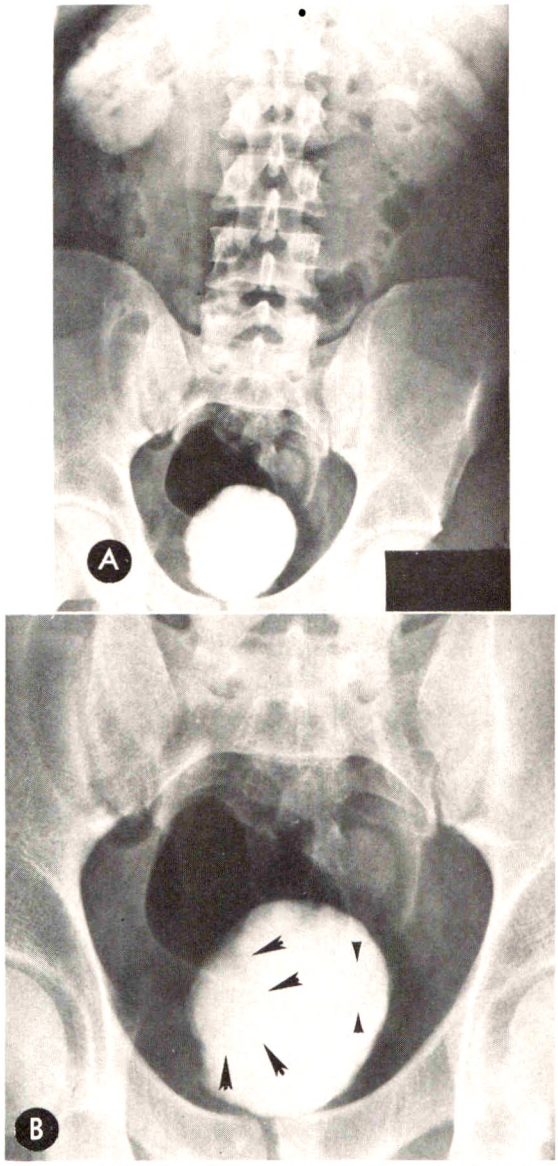

FIG. 3. (*A*) The right ureter is obstructed and a dense right nephrogram is present. (*B*) The right lateral aspect of the bladder is irregular. The right half of the interureteric ridge is markedly widened (large black arrows) with respect to the left (small, vertical arrows).

marked edema. The associated changes in the interureteric ridge (Fig. 3*B*) present as a vague triangular area of radiolucency (large black arrows) which is quite wide in comparison to the normal left side (small vertical black arrows). A tiny opaque stone could be seen on the right on a preliminary roentgenogram but the right distal ureter was never filled with contrast medium even on delayed studies.

CASE IV. A middle aged male presented with acute right lower abdominal pain and dysuria. Urinalysis revealed scanty hematuria. No calculus was visible on a plain film of the abdomen. Excretory urography showed no abnormality of either kidney or ureter except for a roentgenogram exposed in the erect position after voiding (Fig. 4). A large irregular filling defect was present in the right side of the bladder. Notably, the right ureter has drained completely. At cystoscopy, the right ureteral orifice and adjacent interureteric ridge were edematous. A small ureteral stone was extracted by closed manipulation.

DISCUSSION

The frequency of visualization of the interureteric ridge on routine urographic studies is low. This structure is most often seen when the urinary bladder is partly distended with dilute opaque medium and the patient in a supine position.

Nearly 30 years ago, Edling[1] pointed out that the interureteric ridge was visible in about 135 urographic studies he performed when multiple views of the bladder were obtained using different roentgen-ray tube

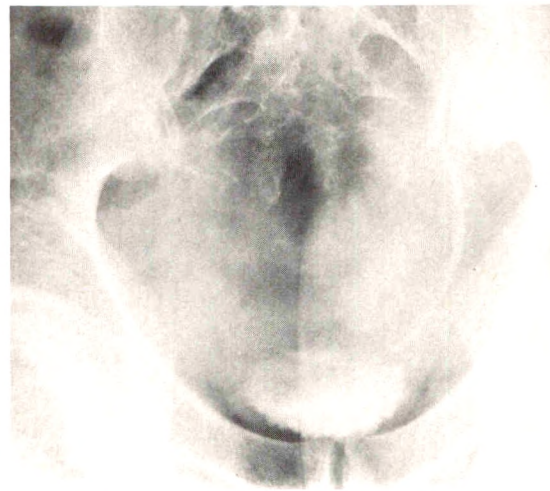

FIG. 4. A large, irregular filling defect is present in the right side of the bladder. The right ureter has drained completely (erect, post-voiding roentgenogram).

angulations. In that paper were published five roentgenograms showing changes in the ridge due to ureteral calculi similar to the preceding cases, although the findings are somewhat difficult to appreciate from the illustrations presented. We concur with Edling's[1] observation that the ridge is very difficult to demonstrate in females. Conversely, we have noted a high frequency of visualization of the ridge in males with anomalies of renal rotation such as horseshoe kidney.

The illustrative cases have shown that roentgenographic changes due to edema and inflammation about the ureteral orifice and adjacent interureteric ridge are useful in the diagnosis of ureterovesical junction calculus or operative trauma. This is true even when no other urographic abnormalities are present as in Case IV. In the absence of acute symptoms, effacement and distortion of the ridge should suggest a bladder cancer.

If the entire interureteric ridge can be clearly seen in an individual with a unilateral non-functioning kidney, the diagnosis of unilateral renal agenesis can be confidently excluded; in unilateral renal agenesis, the ipsilateral half of the trigone is also absent.

Differentiation of intrinsic changes in the interureteric ridge from other filling defects occurring in the urinary bladder such as blood clots, calculi, and ureteroceles should not be difficult when clinical information and standard diagnostic criteria have been applied.[4]

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The author wishes to express his appreciation to Dr. I. Meschan for his advice and assistance.

REFERENCES

1. Edling, N. P. Further studies of interureteric ridge of bladder. *Acta radiol.*, 1948, *30*, 69–75.
2. Emmett, J. L., and Witten, D. M. Clinical Urography. W. B. Saunders Company, Philadelphia, 1971, pp. 354–355.
3. Friedland, G. W., and Cunningham, J. Elusive ectopic ureterocoels. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 792–811.
4. Hertz, M. Cystourethrography. *Excerpta Med.*, 1973, 7.

SCHWANNOMAS OF THE CENTRAL NERVOUS SYSTEM*

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ABSTRACT:

Isolated schwannomas may occur in many different situations within the central nervous system. Most neurilemmomas are benign, and enucleation after longitudinal dissection of the nerve will usually affect a cure. It is for this reason that the roentgenological appearances should be appreciated and this diagnosis considered so as to attempt total surgical removal.

ISOLATED schwannomas form about eight percent of all primary intracranial tumors, usually occur during the middle years of life, and are twice as common in females as in males.[9,13] They are extremely rare in childhood[8] and, as in an adult, the acoustic nerve is the site of predilection.[3] Neurinomas of the spinal cord are relatively common, accounting for about 30 percent of all spinal tumors.[7]

The purpose of this communication is to report the roentgenographic appearances of schwannomas occurring in the central nervous system.

CRANIAL NERVE INVOLVEMENT

This tumor has a tendency to selectively affect sensory nerves and arises on a distal segment of the root after it has penetrated the pia mater. The acoustic nerve is most frequently involved, although on occasions the tumor has been observed arising from the trigeminal nerve. The vagus, glossopharyngeal and other motor nerves are usually involved in the multiple manifestation of von Recklinghausen's disease, but as sites for solitary schwannomas are extremely rare.

(A) ACOUSTIC NEURINOMAS

Acoustic neurinomas account for approximately eight percent of all intracranial tumors and for 80 percent of tumors of the cerebello-pontine angle.[13] Most, but not all, acoustic neurinomas originate within the internal auditory canals, presumably at the junction between the neurilemmal sheath arising from the peripheral ganglia and the neuroglial fibers extending peripherally from the brain stem. The patient usually presents with unilateral sensorineural hearing loss, followed by slight dizziness or imbalance and less frequently by true vertigo.[17]

Roentgenographic evaluation of the internal auditory canal is best performed by

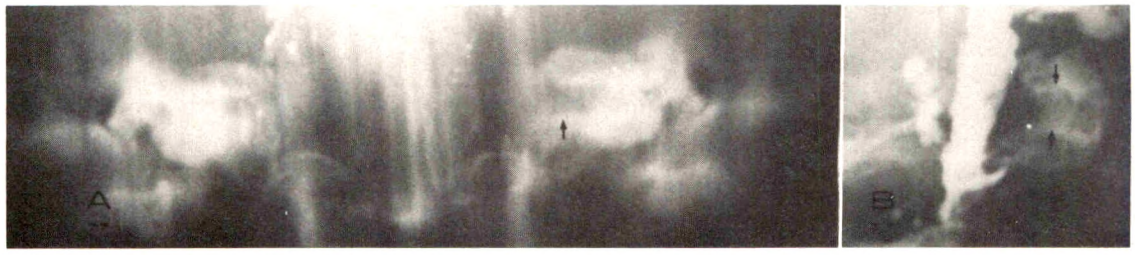

FIG. 1. (*A*) Tomography of the internal auditory meatus demonstrating widening of the left internal auditory canal (↑) with shortening of its posterior wall. (*B*) Posterior fossa cisternogram, shoot through prone/oblique projection, demonstrating non-filling of the left internal auditory canal (↑).

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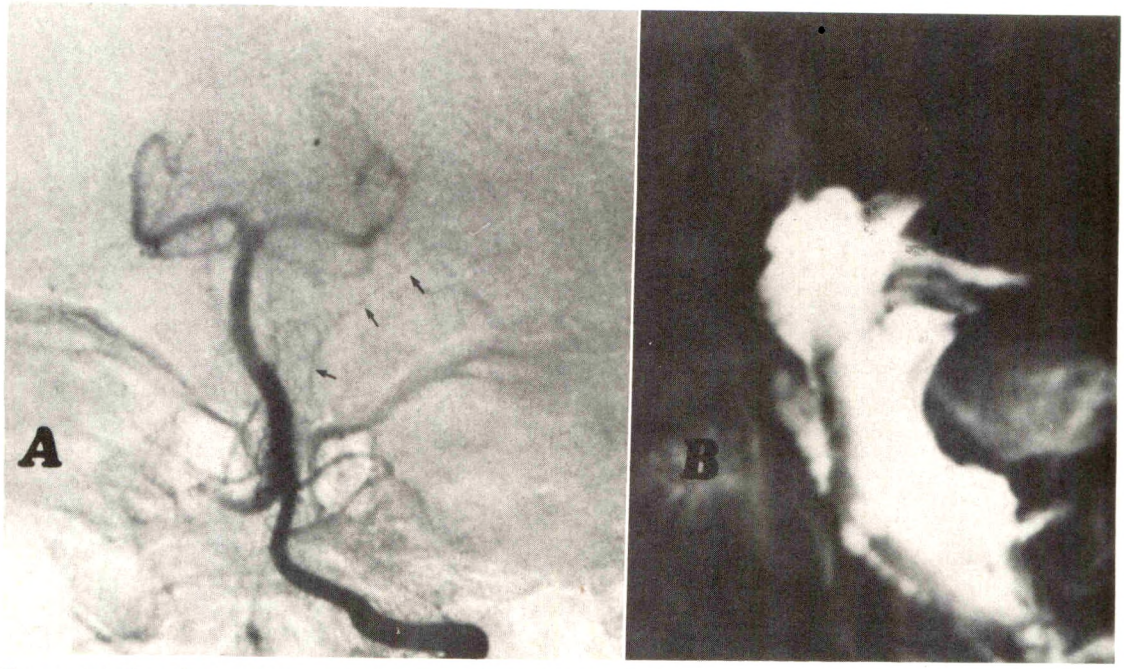

Fig. 2. (*A*) Vertebral angiogram, anteroposterior projection with subtraction, demonstrating marked elevation of the left anterior inferior cerebellar artery (↑) around the mass. (*B*) Posterior fossa cisternogram, demonstrating a mass superolaterally in the cerebello-pontine angle.

tomography where: (a) erosion of the cortical line surrounding the lumen of the canal; (b) widening by 1 to 2 mm of any portion of the internal auditory canal in comparison to the corresponding segment of the opposite side; (c) shortening of the posterior wall by at least 2 to 3 mm; and (d) shortening of the crista falciformis by 1 mm or more when compared with the normal side (Fig. 1*A*) may be observed.

Study of the cerebello-pontine cistern and of the subarachnoid space within the internal auditory canal is performed by vertebral angiography (Fig. 2*A*), air studies,

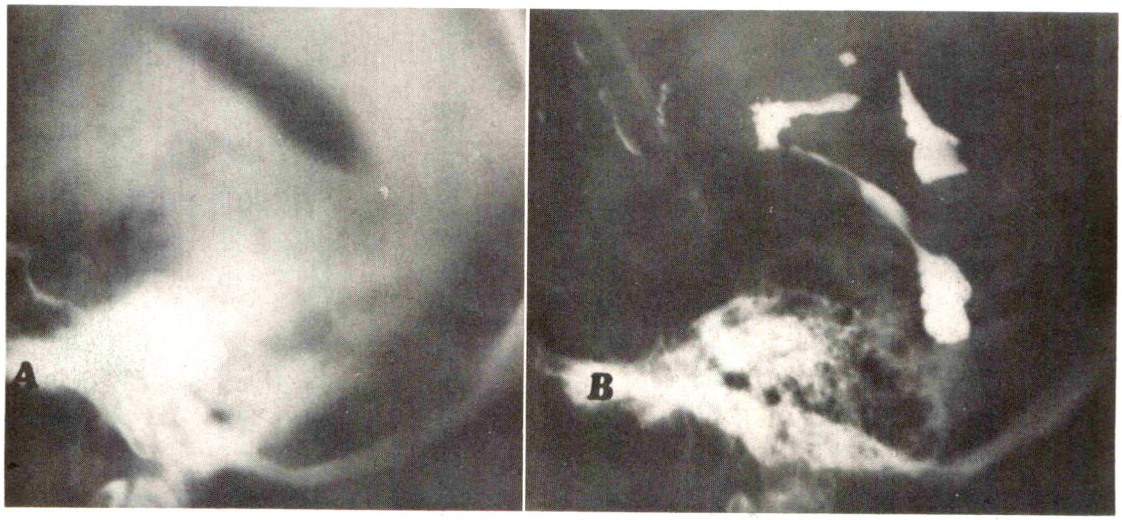

Fig. 3. (*A*) Pneumoencephalogram, erect lateral projection, showing posterior displacement of the fourth ventricle and aqueduct. (*B*) Myodil ventriculogram, demonstrating similar appearances in another patient.

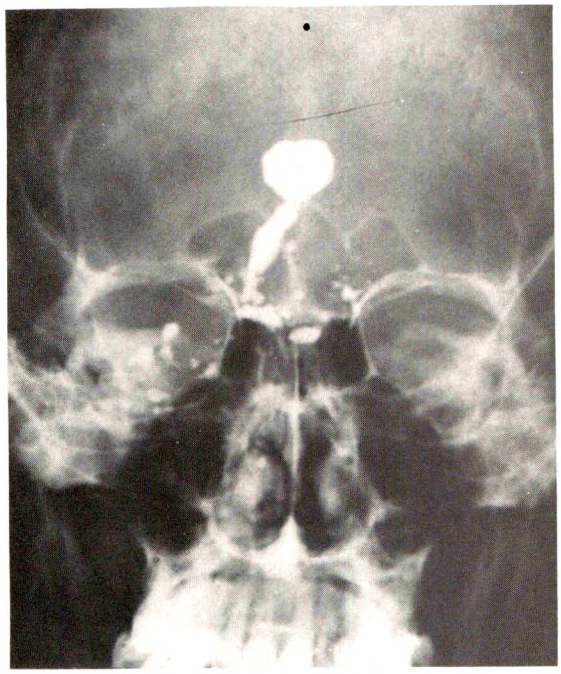

FIG. 4. Myodil ventriculogram, brow up anteroposterior projection, showing displacement of the aqueduct and partially filled fourth ventricle to the right.

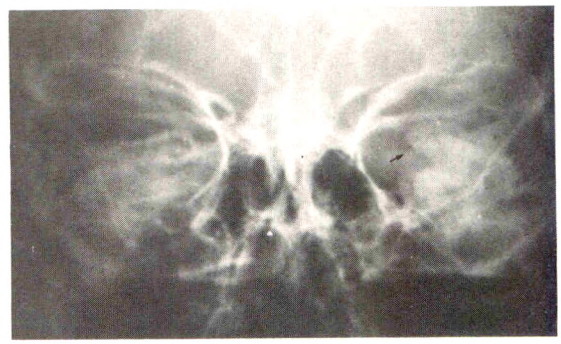

FIG. 5. Skull, anteroposterior projection, demonstrating destruction of the apex of the left petrous bone (↑).

and posterior fossa cisternography (Fig. 2*B*). When a mass is present, its vertical diameter and extension along the posteromedial aspect of the petrous bone can be evaluated as well as the extent of involvement in the internal auditory meatus (Fig. 1*B*). In the study of the internal auditory canal with positive contrast medium, incomplete filling may occur due to anatomic variants or conditions other than a tumor. These include the size of the canal, which varies from 2 to 10 mm with an average of 4.5 to 5 mm, shortening of the meningeal sleeve cul-de-sac, and viscosity of the myodil, when the canal is relatively small, since the size of the neurovascular bundle remains constant.[16]

If pneumoencephalography or ventriculography is performed, this will demonstrate the size of the tumor within the cerebello-

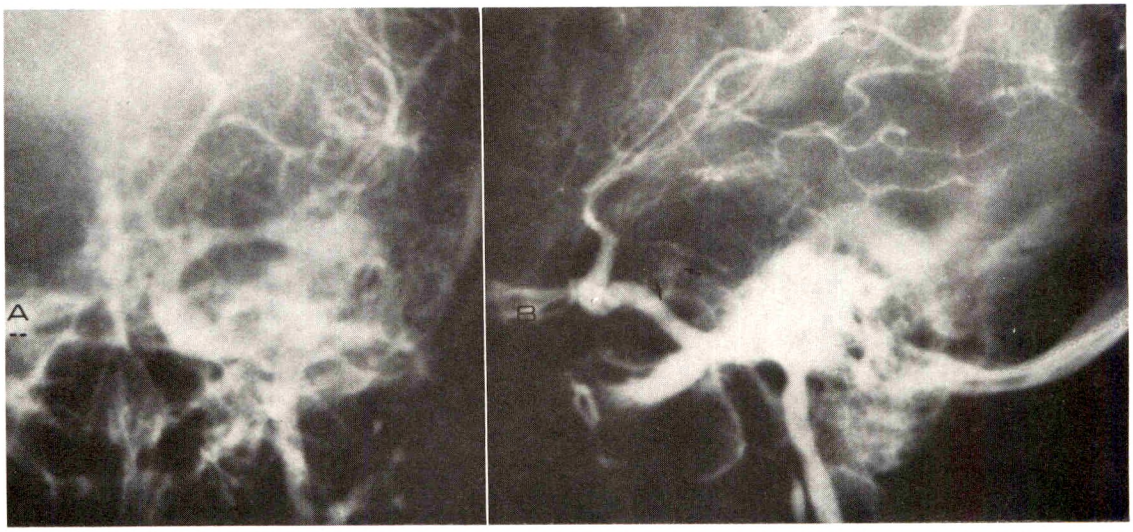

FIG. 6. Left carotid angiogram, (*A*) Anteroposterior and (*B*) lateral projections, demonstrating downward, forward and medial displacement of the ganglion portion of the carotid siphon. Note the meningohypophyseal artery (↑).

pontine angle, by the extent that the brain stem is displaced with related rotation (Fig. 3, *A* and *B*; and 4).

(B) TRIGEMINAL TUMORS

These are rare and comprise only 0.2 percent of intracranial tumors and occur most commonly in the fourth decade.[14] They originate in the gasserian ganglion or trigeminal nerve root and therefore will be located in Meckels' cave or in the cerebellopontine angle. When the tumor increases in size, it may become dumbell in shape and will then be located in both the middle and posterior cranial fossae. These tumors are well circumscribed, grow very slowly and consequently reach a large size before causing sufficient symptoms to require investigation.

Their symptomatology is fairly typical although the manifestations may be varied depending upon the site of origin and direction of spread of the tumor.[12]

Plain roentgenograms of the skull may show destruction of the anteromedial portion of the petrous apex which is smooth and well delineated (Fig. 5).

With extension of the tumor into the middle cranial fossa, carotid angiography will demonstrate that the ganglial part of the siphon (which is normally located extradurally) is displaced forward, downward and medially (Fig. 6, *A* and *B*). There may also be elevation of the middle cerebral group of arteries, stretching of the anterior choroidal artery, and elevation of the basal vein of Rosenthal. A tumor circulation may be evident, usually deep within the middle fossa supplied by the tentorial branch of the meningo-hypophyseal artery (Fig. 7).

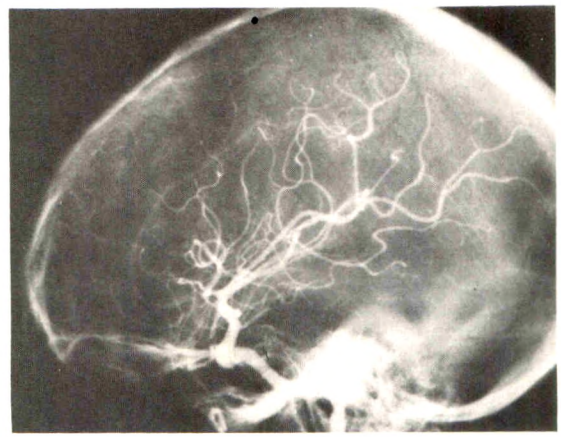

Fig. 7. Carotid angiogram, lateral arterial phase, showing elevation and posterior displacement of the proximal portion of the middle cerebral artery by a mass. Note the meningohypophyseal artery (↑) supplying the tumor.

Spread of tumor into the posterior cranial fossa will be shown by elevation of the posterior cerebral artery, and destruction of the anteromedial portion of the petrous apex.

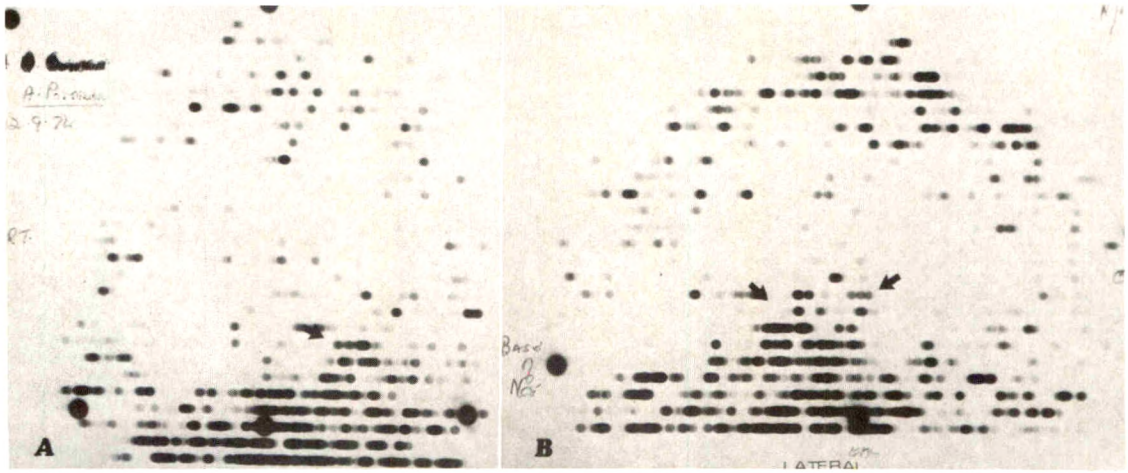

Fig. 8. Brain scan, (*A*) anteroposterior and (*B*) lateral projections, demonstrating an area of increased uptake within the anterior part of the middle cranial fossa (↑).

FIG. 9. Tomography of middle cranial fossa, lateral projection, demonstrating an area of irregular calcification in the right temporal region (↑).

Radioactive brain scanning may demonstrate the situation of the tumor by an area of increased uptake lying deep within the middle cranial fossa (Fig. 8, *A* and *B*).

INTRACEREBRAL SCHWANNOMA

Parenchymatous schwannomas of the brain are rare.[18] As far as we are aware, only two other documented reports of intracerebral schwannomas have been published.[6,11] Our patient complained of episodes of uncontrollable behaviour of which he had no recollection. Roentgenograms of the skull showed an area of calcification in the right temporal region and slight enlargement of the right middle cranial fossa. Tomography confirmed the presence of a well defined area or irregular calcification in the right middle fossa (Fig. 9). Pneumoencephalography showed a cap of air surrounding the area of calcification suggesting that the latter lay in the pes hippocampi (Fig. 10). Right carotid angiography demonstrated slight asymmetry of the middle cerebral arteries, the knee of the right middle cerebral artery being displaced medially. No tumor circulation was demonstrated. The mass was removed surgically from the temporal lobe and histologically proved to be a schwannoma.

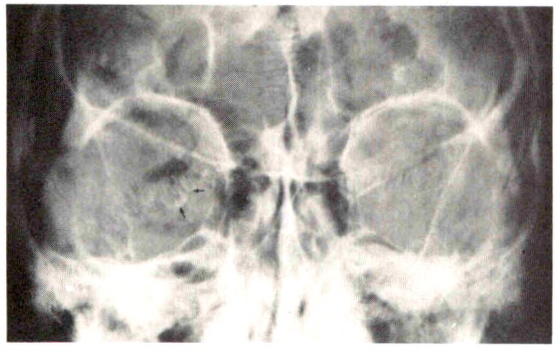

FIG. 10. Pneumoencephalogram, showing air in the right temporal horn above an area of calcification (↑) located in the pes hippocampi.

SCHWANNOMA ARISING WITHIN THE PITUITARY FOSSA

To the best of our knowledge, a schwannoma arising within the pituitary fossa has not been previously reported. This patient presented with a history of headaches and progressive blindness.

This tumor produced destruction of the pituitary fossa, extending upward to elevate and encroach onto the optic nerves and downwards into the sphenoid sinus and nasopharynx. Lateral extension of the tumor produced occlusion of both carotid arteries by direct involvement as well as destruction of the right petrous temporal bone (Fig. 11; and 12, *A*). The tumor was surgically visualized and histologically proved.

ORBITAL SCHWANNOMAS

A schwannoma occurring within the orbit is considered to be a rare tumor.[5] This

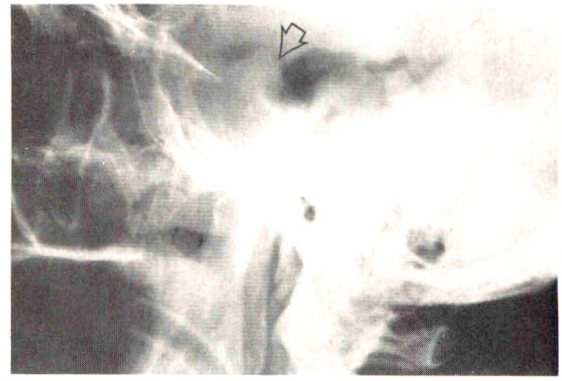

FIG. 11. Pneumoencephalogram, demonstrating an intrasellar mass extending superiorly (open arrow). Note the destruction of the pituitary fossa, obliteration of the sphenoid sinus and extension into the nasopharynx (↑).

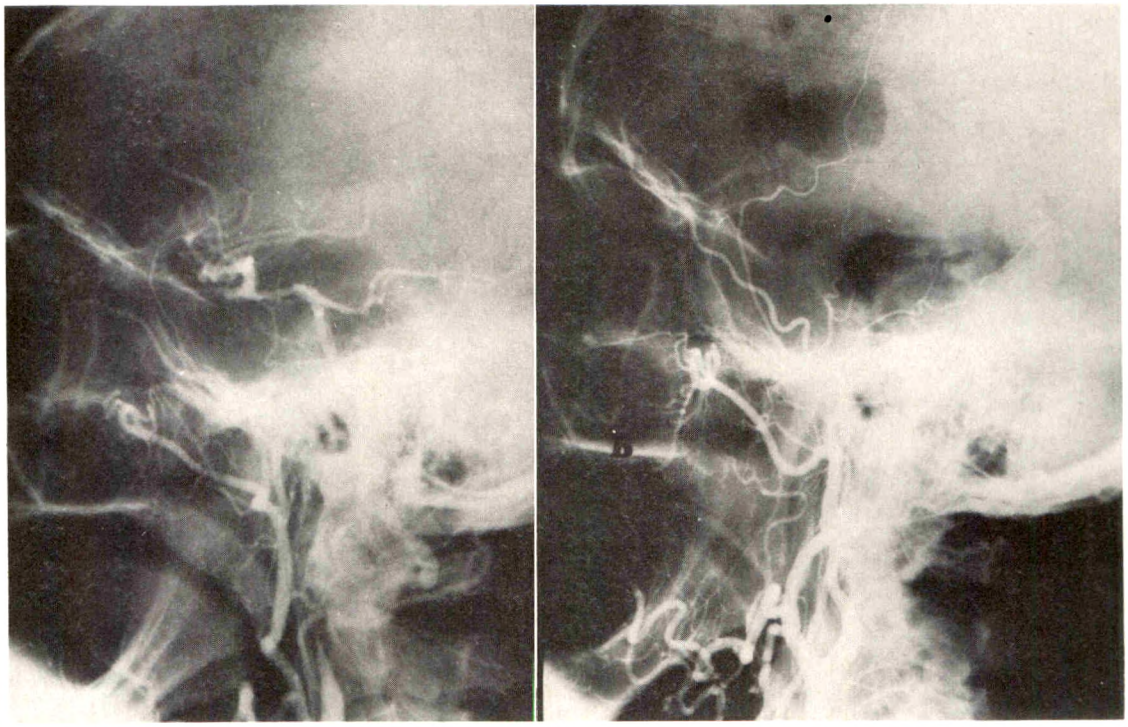

Fig. 12. Bilateral carotid angiogram, lateral arterial phases, demonstrating occlusion of both internal carotid arteries as far as the carotid siphon. The intracranial circulation on the left is filling *via* the vertebrobasilar system by means of collateral circulation in the neck.

is a localized, encapsulated, usually benign nerve sheath tumor growing slowly without distinctive clinical features. It can cause exophthalmos and blurring of vision by pressure on the optic nerve.[10] This tumor may be very vascular, supplied by an enlarged ophthalmic artery, and well demonstrated on carotid angiography (Fig. 13, *A*

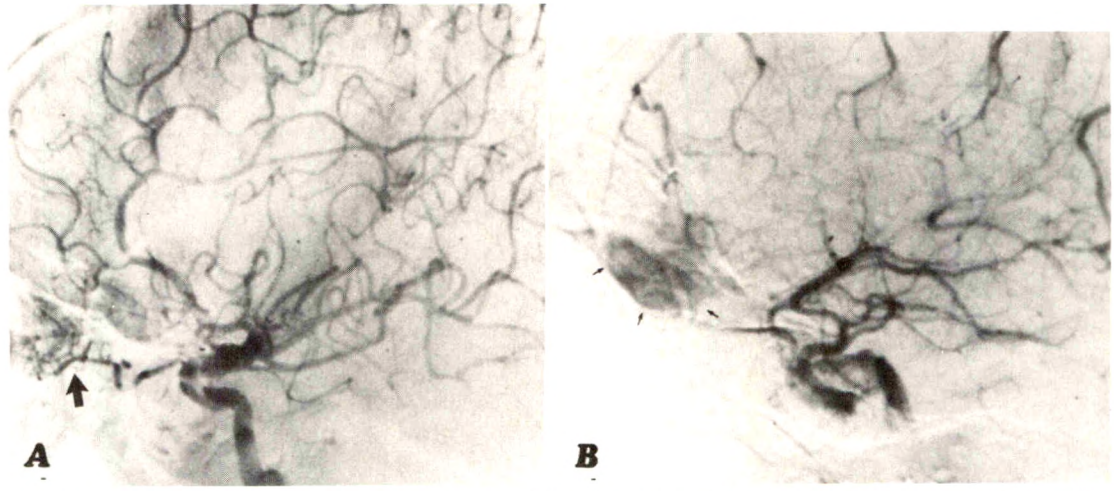

Fig. 13. Selective left internal carotid angiogram with subtraction, (*A*) arterial and (*B*) venous phases, demonstrating an enlarged ophthalmic artery supplying a vascular tumor within the superior part of the orbit (↑).

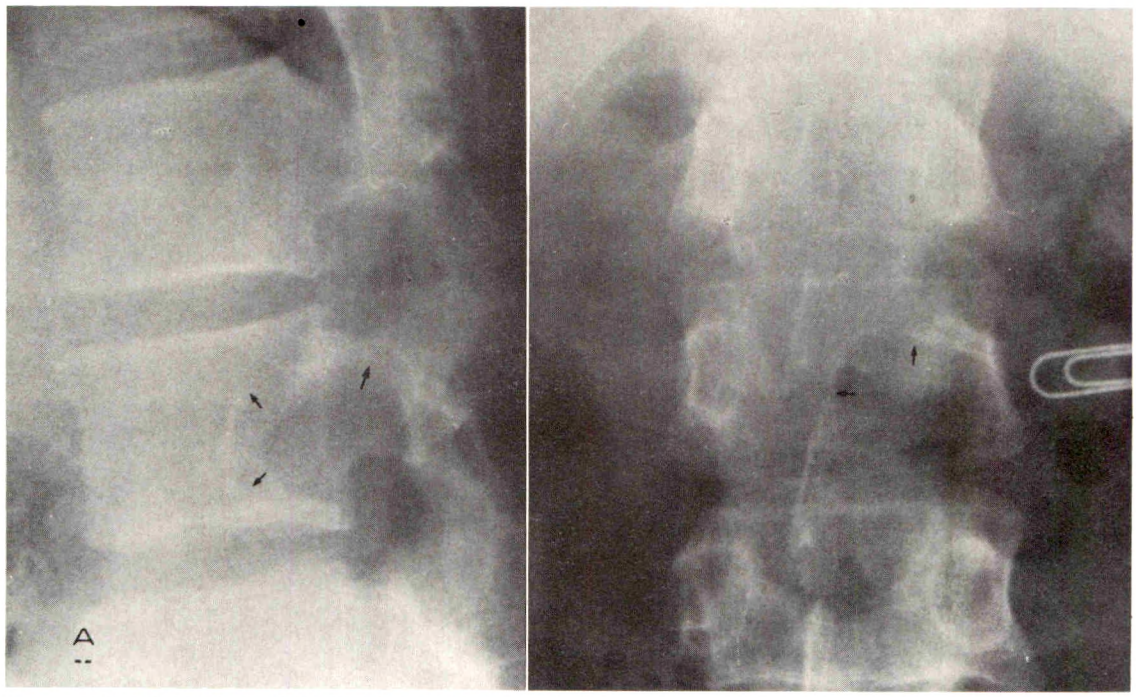

FIG. 14. Lumbar spine, (*A*) lateral and (*B*) anteroposterior projections, demonstrating scalloping of the posterior margin of a lumbar vertebra produced by a schwannoma originating within the spinal canal (↑). Note that the adjacent pedicle is thinned inferiorly and medially.

and *B*). Venography may aid in defining the situation of the mass lesion within the orbit, but gives no indication as to its pathology. If the intra-orbital pressure is significantly elevated, there may be non-visualization of the superior ophthalmic vein on the involved side.

Recurrence of the tumor after total excision is uncommon but does occur. This is considered to be due to residual elements of tumor remaining after surgery rather than due to malignant change.

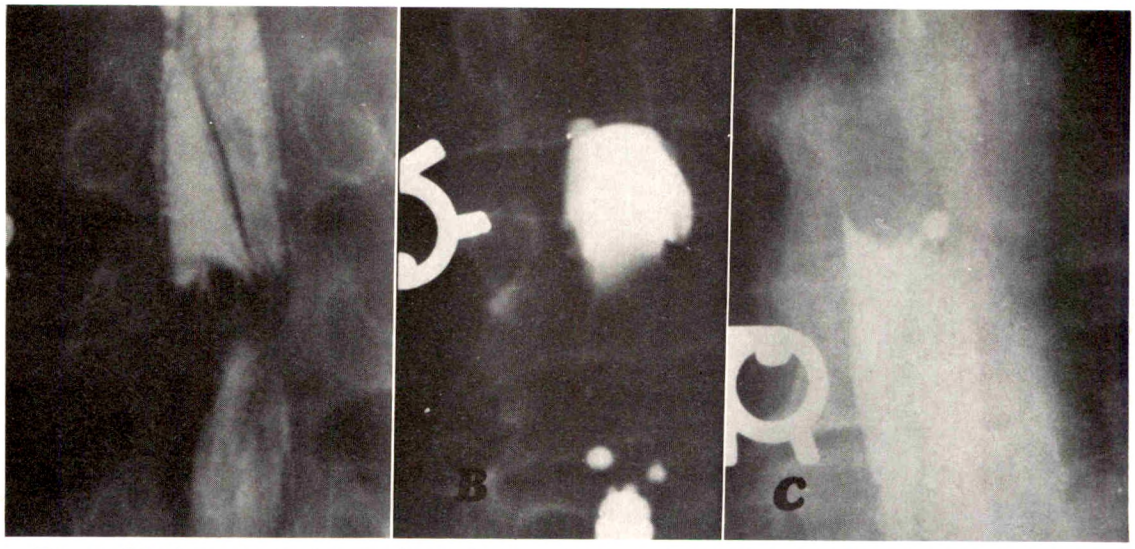

FIG. 15. Myelography in three patients, showing the typical cap deformity of intradural neurinomas.

SPINAL NEURINOMAS

These tumors may be situated at any point along the long axis of the spinal cord or cauda equina. Since these tumors originate from the nerve sheath, they may be situated extradurally, intradurally, or have an element which is situated both extra and intradurally.

(A) INTRADURAL NEURINOMAS

These are in direct contact with the contrast medium. Hence, they are sharply outlined by the dye as clear cut cap defects (Fig. 15). This is their most common characteristic feature.[15] As they arise from the nerve roots, they tend to lie to one side of the midline and displace the cord to the contralateral side. Those that occupy a more central position during their growth, either in front of or behind the spinal cord, displace the cord in a sagittal direction. This results in an anteroposterior compression of the cord, causing an increase in its coronal diameter. The block produced by such a tumor will be indistinguishable from that produced by an intramedullary tumor in the anteroposterior projection. A lateral projection will, however, reveal the relationship of the tumor to the spinal cord. The symptomatology varies according to

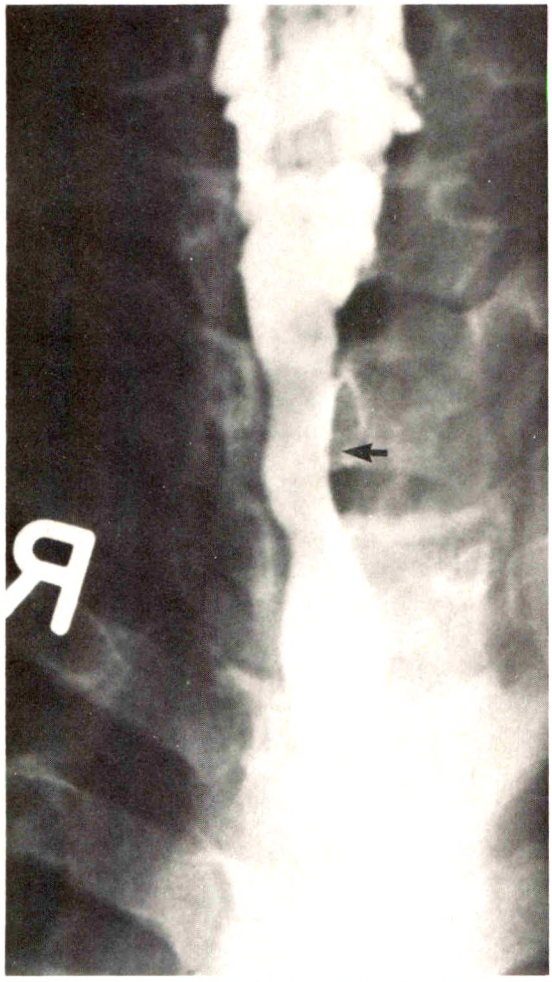

Fig. 16. Thoracic myelogram, anteroposterior projection, demonstrating an extradural displacement of the contrast column at T2 (↑).

Fig. 17. Cervical myelogram, oblique projection, demonstrating the intradural component of a dumbbell schwannoma at C_3/C_4. Note the enlarged intervertebral foramen (↑).

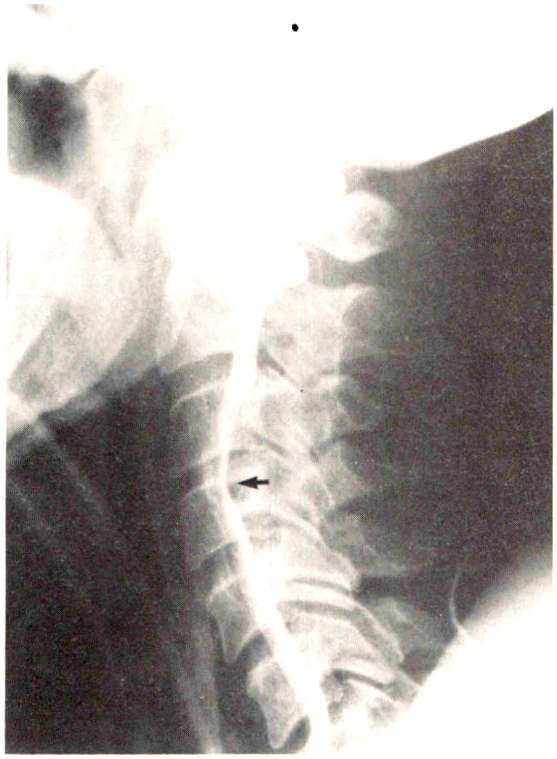

FIG. 18. Vertebral angiogram, lateral projection, demonstrating anterior displacement of the vertebral artery by a schwannoma.

the situation of the tumor in the spinal canal.[1]

Plain roentgenograms are helpful only in approximately 30 percent of cases.[2] The most frequent primary roentgenographic finding is enlargement of the adjacent intervertebral foramen due to pressure erosion produced by growth of the tumor along the axis of the root through the foramen. This area of constriction at the foramen causes the tumor to be dumbbell in shape, and thus produces pressure erosion of the adjacent bone. Widening of the spinal canal with indentation into the dorsal aspect of the related vertebral body may also be observed (Fig. 14, *A* and *B*). Diagnostic accuracy is in the region of 98 percent with myelography.[2] The myelographic appearances will vary depending upon whether the tumor is situated extra- or intradurally: intradural tumors situated below the level of the spinal cord may cause displacement of the nerve roots of the cauda equina if eccentrically situated. If the tumor is centrally located a central cap-shaped defect is produced. Neurinomas in this region attain a large size and produce featureless total blocks.[4]

(B) EXTRADURAL NEURINOMAS

Like other extradural tumors, these displace the arachnoid from the pedicles, producing a lateral indentation on the contrast column with or without complete obstruction, depending on the size of the tumor (Fig. 16).

(C) INTRA AND EXTRADURAL NEURINOMAS

The myelographic pattern is influenced by the size of each component of the dumbbell tumor (Fig. 17). The intradural portion generally produces the typical cap deformity with displacement of the spinal cord. The extradural part can be discerned only if there is a lateral indentation on the side of the tumor in addition to the cap defect. This will be evident if the extradural part is of sufficient size and is intraspinally located.

When the schwannoma is situated in the cervical region, vertebral angiography is valuable in defining the extent of the anterior and medial displacement of the vertebral artery by the tumor, since this may not be evident from the size of the enlarged intervertebral foramen (Fig. 18).

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REFERENCES

1. BALAPARAMESWARA RAO, M. S., DINAKAR, I., and SUJATHAMMA, Y. Spinal neurinoma: study of fifty surgical cases. *Internat. Surg.*, 1970, *54*, 410–414.
2. CAMP, J. D. Roentgenologic localization of tumors affecting spinal cord. AM. J. ROENTGENOL., RAD. THERAPY, 1938, *40*, 540–544.
3. CRAIG, W. M., DODGE, H. W., and ROSS, P. J. Acoustic neuromas in children: report of two cases. *J. Neurosurg.*, 1954, *11*, 505–508.
4. DINAKAR, I., and BALAPARAMESWARA RAO, M. S.

Myelographic features of spinal neurinomas. *Internat. Surg.*, 1972, *57*, 730–734.
5. Duke-Elder, S. Textbook of Ophthalmology. Henry Kimpton, London, 1952, pp. 5580.
6. Gibson, A. A. M., Hendrick, E. G., and Conen, P. E. Intracerebral schwannoma: report of case. *J. Neurosurg.*, 1966, *24*, 552–554.
7. Jacobs, R. L., and Barmada, R. Neurilemoma: review of literature with six case reports. *A.M.A. Arch. Surg.*, 1971, *102*, 181–186.
8. Matson, D. D. Neurosurgery of Infancy and Childhood. Second edition. Charles C Thomas, Publisher, Springfield, Ill., 1969.
9. Minckler, J. Supporting cell tumors of peripheral nerves. In: Pathology of the Nervous System. Edited by J. Minckler. McGraw-Hill Book Company, Inc., New York, 1971, pp. 2093–2114.
10. Mohan, H., and Sen, D. K. Orbital neurilemmoma: presenting as retrobulbar neuritis. *Brit. J. Ophth.*, 1970, *54*, 206–207.
11. New, P. F. J. Intracerebral schwannoma: case report. *J. Neurosurg.*, 1972, *36*, 795–797.
12. Olive, I., and Svien, H. J. Neurofibromas of fifth cranial nerve. *J. Neurosurg.*, 1957, *15*, 484–505.
13. Russell, D. S., and Rubinstein, L. J. Pathology of Tumours of the Nervous System. Third edition. Edward Arnold & Company, London, 1971.
14. Schisono, G., and Olivecrona, H. Neurinomas of gasserian ganglion and trigeminal root. *J. Neurosurg.*, 1960, *17*, 306–322.
15. Shapiro, J. H., Och, M., and Jacobson, H. G. Differential diagnosis of intradural (extramedullary) and extradural spinal canal tumors. *Radiology*, 1961, *76*, 718–732.
16. Valvasorri, G. E. Abnormal internal auditory canal: diagnosis of acoustic neuromas. *Radiology*, 1969, *92*, 499–459.
17. Valvasorri, G. E. Diagnosis of acoustic neuromas. *Otolaryng. Clin. North America*, 1973, *2*, 391–400.
18. Van Rensburg, M. J., Proctor, M. S. F., Danziger, J., and Orelowitz, M. S. Case of temporal lobe epilepsy due to an intracerebral schwannoma. *J. Neurol., Neurosurg. & Psychiat.*, 1975, *38*, 703–709.

THE NEURORADIOLOGIC EVALUATION OF "OPTIC NEURITIS"*

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ABSTRACT:

Optic neuritis, even when clinically typical, cannot be diagnosed with certainty because mass lesions compressing the anterior visual pathways may mimic it. The radiologist is in a position to assist in identifying such lesions or in ruling them out and thus preventing surgical exploration. While every patient in whom the diagnosis of optic neuritis is entertained should have plain roentgenograms of skull, orbits and optic canals, we have developed a schema for detailed neuroradiologic investigation based on clinical and plain roentgenographic findings. A classification of such lesions by location is illustrated.

THE term optic neuritis should be reserved for the designation of acute inflammatory or demyelinating disorders of the optic nerve. Many cases are due to multiple sclerosis, but most are cryptogenic. The characteristic features are listed in Table 1. The patient is usually young, between 15 and 50 years. Typically, the condition is heralded by retro-ocular pain or pain on eye movement. After an interval of hours to days, the patient notices impaired visual acuity in one eye. The vision then fails progressively over a period of days and usually plateaus at a level of 20/200 or worse. Examination generally shows a central scotoma, impaired ability to perceive colors, tenderness on palpation of the globe, and either a normal optic disk (retrobulbar neuritis, the lesion being at a distance from the globe) or mild disk swelling (papillitis, the lesion being close to the globe). A pale disk may be present if the disorder is recurrent. There is impairment of pupillary constriction to direct light stimulus; the pupil of the involved eye may constrict when stimulated directly, but dilates again if the stimulus is continued (Marcus Gunn's pupil sign). In red-free light it may be possible to identify gaps in the nerve fiber layer of the retina adjacent to the disk.[6] Even without treatment, most patients recover completely, or nearly so, over a period of weeks or months. Pallor of the nerve may be the only souvenir of the disorder. A hereditary form of optic neuritis (Leber's hereditary optic neuropathy) may cause confusion; patients with this disease tend not to recover, there is usually a family history, and there are ophthalmoscopic and fluorescein angiographic findings that may assist in arriving at the correct

TABLE I

CHARACTERISTIC FEATURES OF OPTIC NEURITIS[1,16,17,19]

1. Patient aged 15 to 50
2. Rapid progression, maximal visual loss within 2 weeks
3. Severe visual loss
4. Pain on eye movement or tenderness of the globe
5. Marked improvement commencing within weeks without therapy
6. Central, paracentral, or arcuate scotoma
7. Afferent defects in pupillary motility
8. Impaired color perception
9. Normal fundus (retrobulbar neuritis) or disk edema (papillitis)

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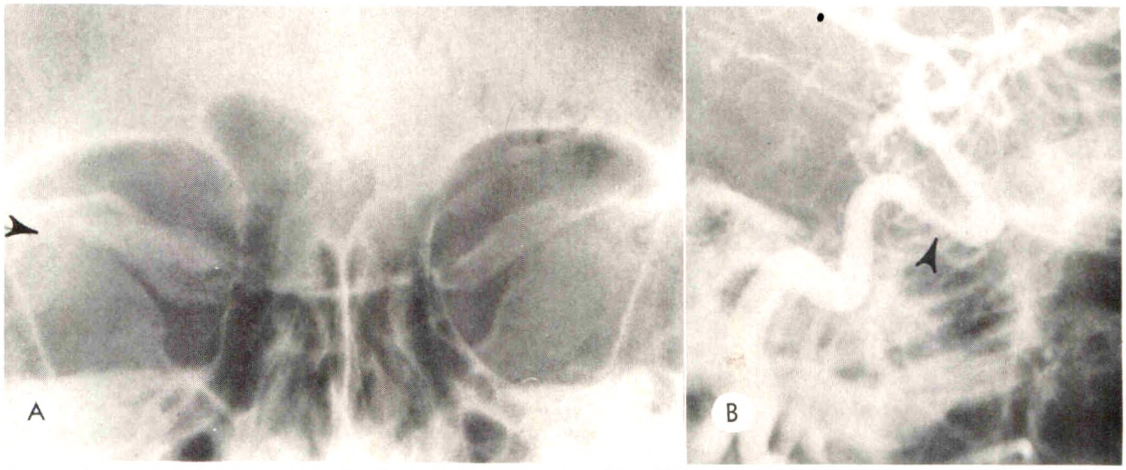


Fig. 2. Case II. (*A*) Hyperostosis of greater and lesser sphenoid wings on the right (arrow). (*B*) Irregular narrowing of internal carotid artery siphon due to encasement by sphenoid wing meningioma.

ence of simultaneous bilateral involvement, was marked by prompt and complete recovery.

Case IV. R.L. (Hospital #497441), a 26 year old man, consulted an ophthalmologist because of painless visual failure in the right eye—"things look darker." He had normal visual acuity bilaterally, but the right optic disk was swollen and there was a right Marcus Gunn pupil sign. Vision failed slowly, and serial tests for visual acuity showed a drop to 20/25 in the right eye. He was referred to our institution for evaluation of right "optic neuropathy" a year and a half after initial symptoms. At that time visual acuity was 20/25 on the right and 20/15 on the left. Impaired color vision was detected on the right, and he had disk edema in the right eye. There was no exophthalmos, but a suggestion of increased resistance to retrodisplacement of the globe ("tight orbit") was noted. He had an irregular concentric field loss. An iris abnormality sometimes seen in neurofibromatosis was present. Neurologic evaluation was normal.

Plain skull, orbit, and optic canal roentgenograms, optic canal tomograms, and orbital venography were normal. Carotid angiography, using subtraction and magnification techniques, disclosed tumor vessels derived from branches of the ophthalmic artery and a homogeneous tumor stain along the course of the optic nerve persisting into the venous phase (Fig. 4, *A–C*). The height of the lateral segment of the second portion of the ophthalmic artery was 8 mm (upper limits of normal 7 mm),[18] suggesting

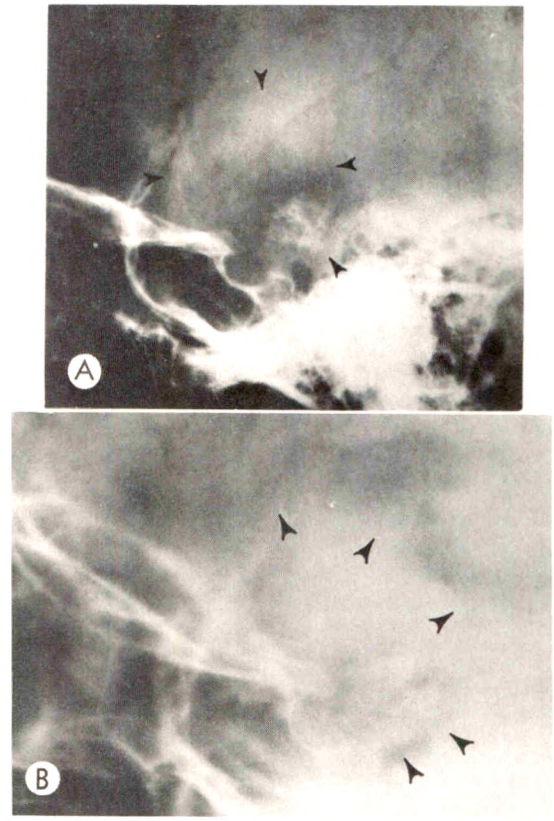


Fig. 3. Case III. (*A*) The arrows outline this suprasellar calcified craniopharyngioma delineated by peripheral curvilinear calcification. Clumps of calcium are also present and the dorsum sellae is markedly truncated. (*B*) At pneumoencephalography a large lobulated mass is demonstrated (arrows) indenting the floors of the anterior horns of both lateral ventricles and obliterating the anterior portion of the third ventricle.

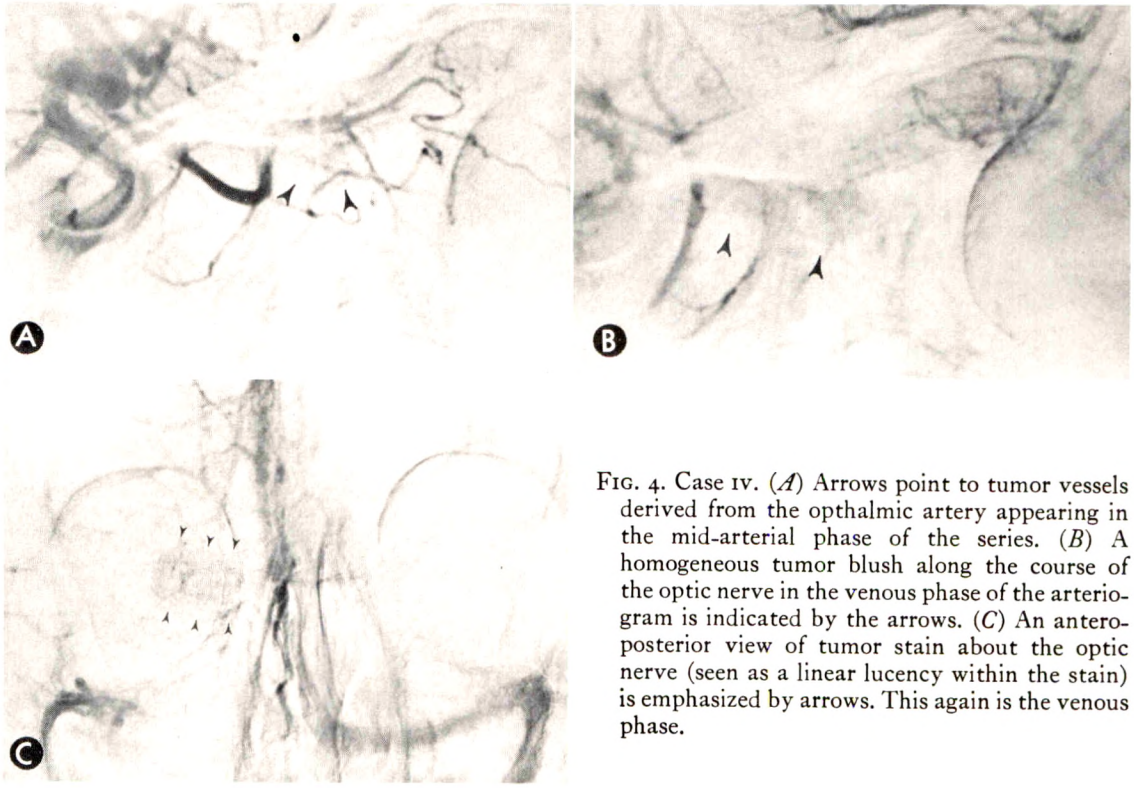


FIG. 4. Case IV. (*A*) Arrows point to tumor vessels derived from the opthalmic artery appearing in the mid-arterial phase of the series. (*B*) A homogeneous tumor blush along the course of the optic nerve in the venous phase of the arteriogram is indicated by the arrows. (*C*) An anteroposterior view of tumor stain about the optic nerve (seen as a linear lucency within the stain) is emphasized by arrows. This again is the venous phase.

enlargement of the optic nerve or its sheath. The findings were characteristic of an optic nerve sheath meningioma. No evidence of intracranial tumor was present. Ultrasonography corroborated the presence of a mass around the nerve. Currently, the patient is being observed because of his relatively intact visual acuity and the possibility of blindness resulting from removal of such a tumor.

CASE V. L.W. (Hospital #493909), a 64 year old woman was found on routine ophthalmological examination to have impaired visual acuity in the right eye. On subsequent examinations, further reduction in acuity and a centrocecal scotoma (a scotoma extending from fixation to the blind spot) was found. Upon referral to our institution, it was noted that the visual acuity was 20/40 in the right eye and 20/25 on the left. A right Marcus Gunn pupil sign and impaired color vision on the right were present as well as right disk pallor on funduscopic examination. A centrocecal scotoma was present in the right visual field, the left being normal. No neurologic abnormalities were detected.

Plain roentgenograms of the skull and optic canals were normal as was tomography of the optic canals axially. Brain scan was normal. Angiography (Fig. 5) demonstrated an aneurysm of the supraclinoid portion of the right internal carotid artery with no other abnormalities. Because of the location of the aneurysm it was felt that it led to compression of the optic nerve by displacing the carotid artery which, in turn, compressed the nerve.

Comment. Slow progression was the major feature of this patient's presentation which led to further investigation. The absence of clinical or roentgenologic findings pointing to the orbit, and the normal tomographic evaluation of the optic canals led to an intracranial search for a suspected compressive lesion.

CASE VI. B.Z. (X-ray #127839), a 68 year old woman who had been followed by an ophthalmologist for five years for a refractive problem, was found to have disk swelling and subnormal vision in the left eye; she was asymptomatic. The ophthalmologist recorded slow progres-

sion of the visual deficit and recommended additional evaluation with the patient refused. Borderline diabetes was later found and her optic nerve impairment was attributed to diabetic neuropathy. Two years later, the patient first noted subjective visual impairment on the left and was referred to our institution. Visual acuity was normal on the right and 20/200 on the left. There was a left Marcus Gunn pupil sign. Funduscopic examination showed a pale, elevated left disk. Orbital resiliency was normal and proptosis absent.

Plain roentgenograms and optic canal tomograms were obtained initially. These demonstrated enlargement of the left optic canal with slightly thickened cortical margins (Fig. 6*A*) and hyperostosis of the anterior clinoid as well as faint curvilinear calcification about the optic nerve within the canal (Fig. 6*B*), findings typical of meningioma. The patient has refused further evaluation.

Comment. This patient demonstrated gradual progression and a pale elevated disk, features indicating strongly the need for evaluation for a compressive lesion. The presence of disk swelling unassociated with evidence of an orbital mass suggested a strategic location, the optic canal. A meningioma was considered a strong possibility on clinical grounds both because of the slow progression and because meningiomas of the sphenoid wing or optic canal can seed along the optic nerve closer to the globe, producing disk swelling without a mass effect.[3,12]

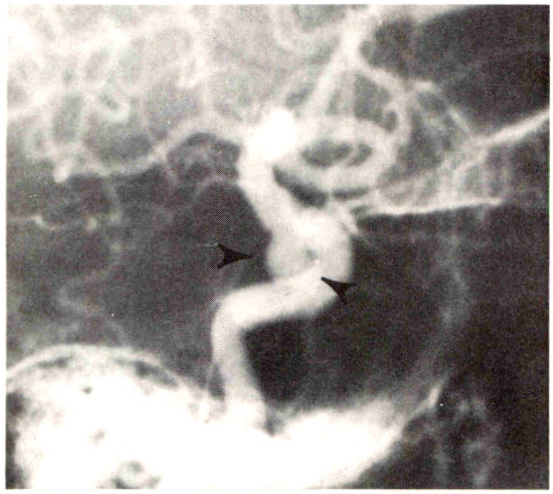


Fig. 5. Case v. Arrows point to an aneurysm arising from the supraclinoid portion of the internal carotid artery.

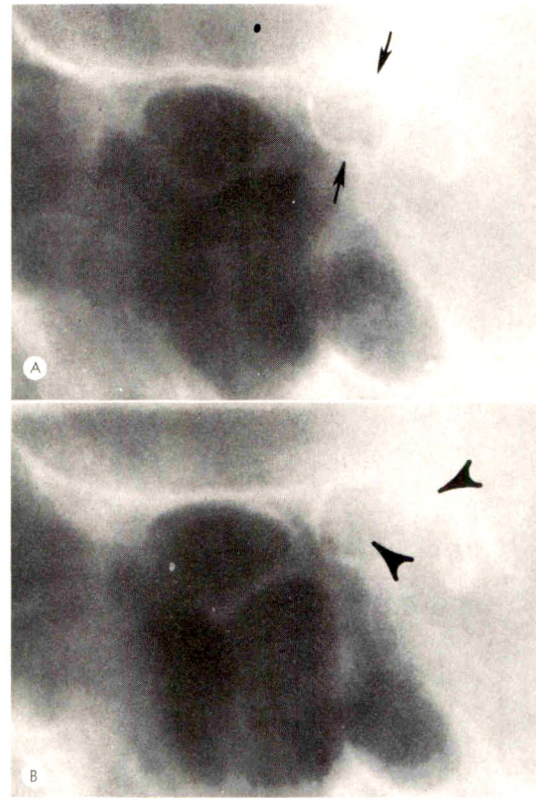


Fig. 6. Case vi. (*A*) Hypocycloidal tomogram perpendicular to the axis of the optic canal demonstrating enlargement of the posterior portion of the canal with increased thickness and density of its walls (arrows). (*B*) The cranial end of the optic canal, just posterior to *A*, showing faint calcification about the optic nerve within the canal (lower arrow) and hyperostosis of the anterior clinoid process (upper arrow).

Case vii. P.K. (Hospital #480195), a 16 year old boy, was found on a routine school visual test to have impaired vision in the left eye; previous examinations had been normal. Just prior to the examination, he had become aware of impaired vision in that eye. He had no other physical or neurological symptoms. He was referred to our institution. His visual acuity was 20/20 on the right and 20/50 on the left. There was a Marcus Gunn pupil sign on the left. Visual field testing showed a defect involving mainly the superior temporal quadrant of

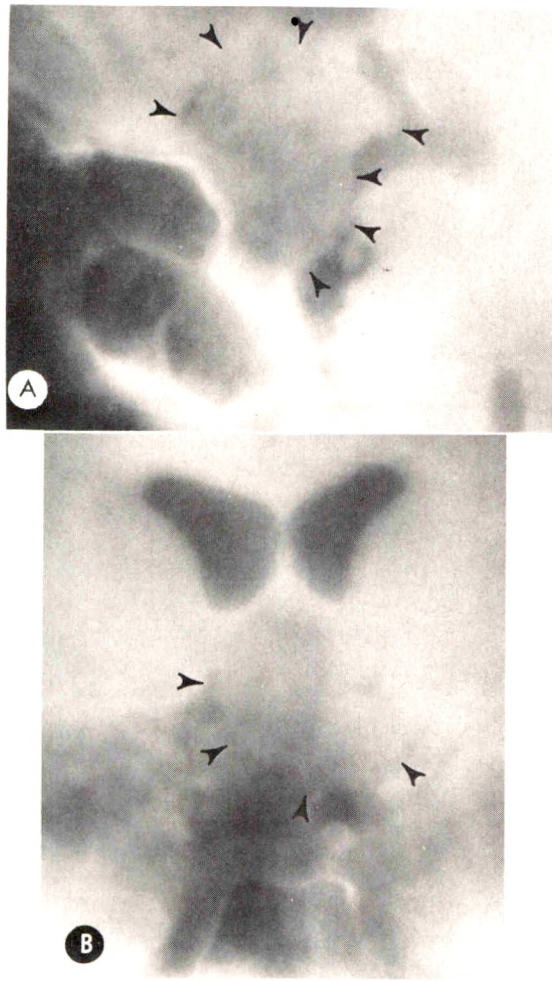


Fig. 7. Case VII. (*A*) Midline hypocycloidal pneumotomogram in the brow-up position and lateral projection demonstrating a suprasellar lobulated mass (arrows) outlined by air in the basal cisterns and by the indented anterior aspect of the third ventricle. The uppermost posterior arrow indicates a nodular excrescence of this tumor. (*B*) On this anteroposterior brow-up hypocycloidal pneumotomogram, the arrows outline the suprasellar mass which is seen to extend more to the left of the midline than to the right.

the left eye but with extension into the inferior quadrant. Testing with smaller test objects detected an inconstant area of impaired vision in the superior temporal quadrant of the right eye. The funduscopic examination showed questionable pallor of the left optic disk compared with the right. Plain roentgenograms of the skull and optic canals and an isotope brain scan were normal. Clinical examination one month later showed no changes.

Pneumoencephalography demonstrated a large, somewhat lobulated suprasellar mass indenting the anterior aspect of the third ventricle, extending more to the left of the midline than the right, and separated from the soft tissue contents of the sella by air outlining the undersurface of the mass (Fig. 7, *A* and *B*). Carotid angiography was normal except for displacement of the anterior choroidal arteries laterally. The patient refused treatment and has been lost to follow-up.

Comment. The findings prompting evaluation for a compressive lesion in this boy were the presence of a temporal field loss in the symptomatic eye and the suggestion of a second temporal field loss in the opposite eye together with the lack of improvement. Bitemporal field defects indicated a lesion affecting the chiasm and directed the investigation to this region.

Case VIII. L.L. (Hospital #492786), a 9 year old girl, was found on a routine school visual examination to be blind in the left eye. Her ophthalmologist noted disk pallor in that eye and had her evaluated at a local hospital for a possible compressive lesion; routine roentgenograms and a pneumoencephalogram were normal. Follow-up examination three years later showed that the pale left optic disk was now elevated. The patient was re-evaluated, and calcification was noted in the region of the left optic canal; brain scan, pneumoencephalogram, and carotid arteriogram were interpreted as normal. She was subsequently referred to our institution for evaluation. Ophthalmological examination disclosed no light perception in the left eye and a pale elevated left optic disk. No exophthalmos was present, but the orbit was tight when compared with the right. Neurological examination was normal. Plain roentgenograms and optic canal axial tomograms showed enlargement of the left optic canal throughout, with a dense circle of calcification about the optic nerve (Fig. 8, *A* and *B*); the calcification extended beyond the orbital end of the canal. Carotid arteriography demonstrated tumor vasculature in the orbit, derived from the ophthalmic artery, demonstrating the orbital extension of the tumor (Fig. 8*C*). A pneumoencephalogram, including pneumotomographic films, showed no intracranial extention (Fig. 8*D*). The plain roentgenograms and tomographic findings were typical of optic

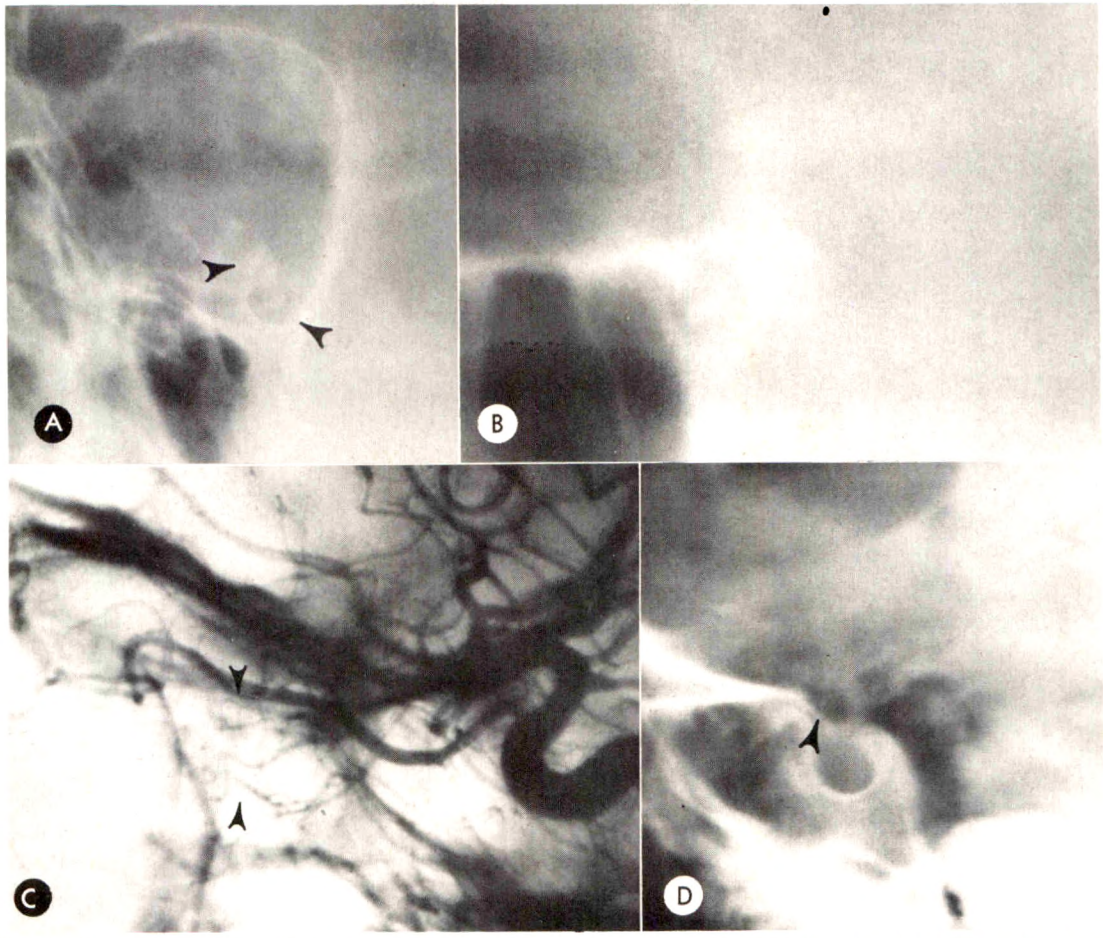


Fig. 8. Case viii. (*A*) A plain roentgenogram of the optic canal showing calcification (arrows) projected in the region of the canal. (*B*) This hypocycloidal tomographic section of the midportion of the optic canal defines dense calcification within the canal about the optic nerve. (*C*) Tumor vasculature arising from the ophthalmic artery (arrows) demonstrates orbital extension of the optic canal meningioma seen in *B*. (*D*) Pneumoencephalography showed no evidence of intracranial extension. The arrow indicates absence of soft tissue mass within the suprasellar cistern.

canal meningioma, and the arteriographic and pneumoencephalographic studies (undertaken to determine any orbital or intracranial extent of the tumor) demonstrated only orbital extension.

Comment. The elevation of the atrophic disk together with the finding of a tight orbit indicated the presence of orbital involvement in a patient with plain roentgenographic findings of a perioptic meningioma in the optic canal.

Case ix. M.H. (Hospital #479049), a 15 year old girl, noted the onset of poor vision in the left eye. Eleven months later, she consulted an optometrist who was unable to correct her vision with glasses. She was ultimately referred to our institution because of a suspected compressive lesion. Her visual acuity was 20/20 on the right and 20/25 on the left. Impaired color perception and a Marcus Gunn pupil sign were noted on the left. The left disk was pale, and the visual field examination disclosed an inferior field loss in that eye "breaking out" to the periphery. Accordingly, plain roentgenographic studies were obtained demonstrating marked enlargement of the intracranial end of the left optic canal with erosion of the left side of the tuberculum (Fig. 9, *A–C*). Pneumotomography demonstrated only a small intracranial mass, the rest of the optic nerve being normal up to the chiasm (Fig. 9*D*), which was also normal.

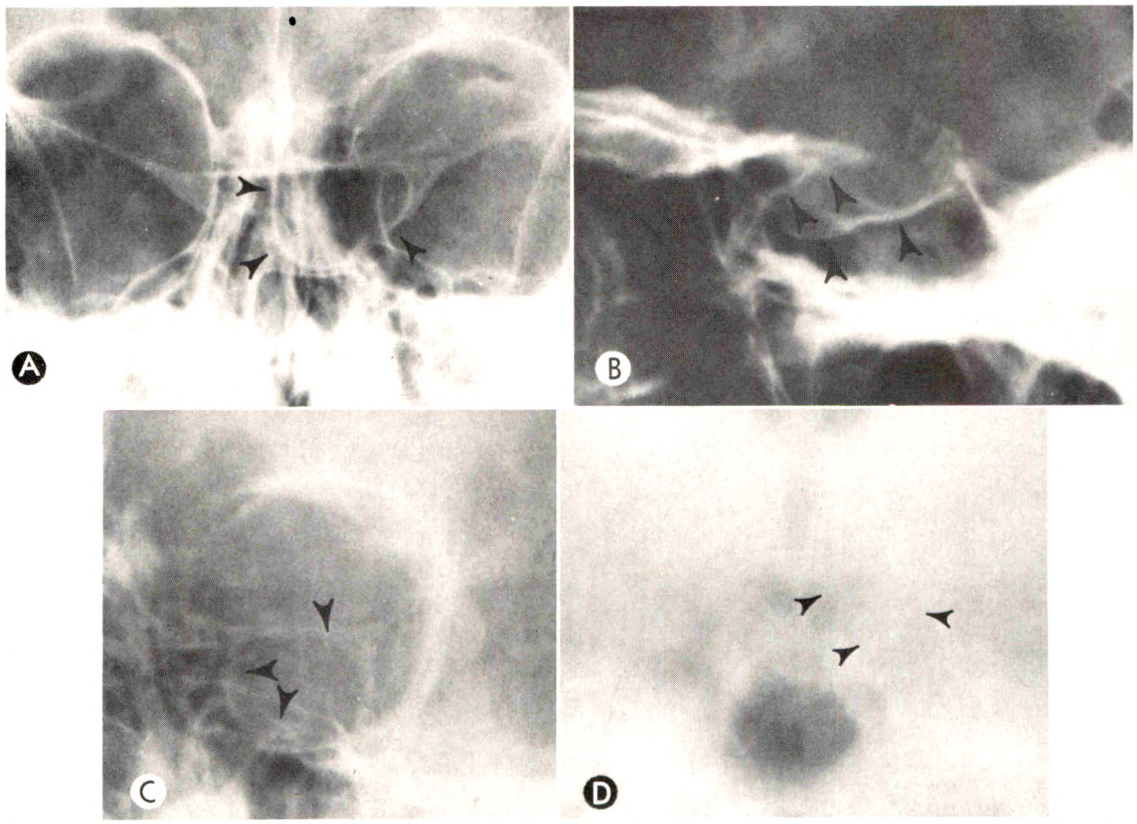


FIG. 9. Case IX. (*A*) A Caldwell projection plain skull roentgenogram detecting marked enlargement of the optic canal with erosion of the left side of the tuberculum. The combined erosion is outlined by arrows. (*B*) A lateral roentgenogram depicting combined erosion of the left portion of the tuberculum and enlargement of the optic canal intracranially (arrows). The anterior clinoid process on that side is eroded from below. (*C*) Standard plain roentgenogram of optic canal showing the massively enlarged intracranial end (lower arrows) and the almost normal orbital end (slightly enlarged), indicated by the upper arrow. (*D*) The intracranial extension of the optic glioma (lower two arrows) is well seen on the brow-up anteroposterior pneumotomogram. The upper arrow indicates the more normal proximal portion of the optic nerve (partially merged with the shadow of the carotid artery).

The findings, clinical and roentgenological, are typical of optic nerve glioma.

Comment. A compressive lesion was suspected because of the persistence of the visual defect and the disk pallor. Plain roentgenograms made the diagnosis clear and directed the further investigation to the intracranial region, as did the absence of clinical evidence of an orbital mass.

DISCUSSION

Compressive lesions of the anterior visual pathway, particularly early in their course, can mimic optic neuritis. The typical presentation of a compressive lesion is one of painless, gradually progressive visual loss with the ultimate development of optic atrophy. The time necessary for disk pallor to appear is probably proportional to the distance of the lesion from the globe. The visual field loss may be central when the optic nerve is involved and bitemporal with involvement of the chiasm. However, a great variety of other visual field changes can be seen depending upon the precise location and nature of the lesion.

In the orbit, masses often indicate their presence by producing proptosis. When such masses are outside the muscle cone, proptosis is typically eccentric and occurs before significant visual loss has taken

place. Masses within the muscle cone, however, are in an excellent position to compress the optic nerve before the onset of proptosis. Lesions of this type can produce swelling of the optic disk, the degree and frequency of swelling depending in part upon the proximity of the lesion to the globe. Lesions confined to the optic canal and intracranial lesions tend not to produce disk swelling except in those rare cases of meningioma where there is direct seeding along the optic nerve. This mechanism may be responsible for disk swelling in some meningiomas of the sphenoid wing and optic canal.[3,12] Thus a normal disk, pale disk, or swollen disk may be seen in compressive optic neuropathies as in optic neuritis. Intracranial lesions may produce neurological or endocrinological abnormalities, but very often visual dysfunction is the first and only symptom. The loss of visual acuity is usually mild at the onset, subjective dysfunction being more prominent. The course is one of slow progression with only isolated instances of periods of improvement. Nevertheless, sudden catastrophic visual loss due to hemorrhage, edema, or shift in position of the mass can occur, producing remarkable similarity to optic neuritis. Patients may also experience transient improvement if treated with corticosteroids, further mimicking optic neuritis.

The physician confronted with patients with cryptogenic optic neuritis must attempt to detect compressive lesions while vision is still salvageable, but must temper his zeal lest patients be exposed to unnecessary risk and expense. The following seems to us a rational approach to this problem. Plain roentgenograms of the skull, orbits, and optic canals should be obtained in all cases. Further roentgenologic evaluation is predicated on the presence of abnormalities on these examinations or on the presence of findings listed in Table III.

A gradual onset and slow progression under observation strongly suggest compression, and dictate thorough evaluation. When a disk is pale and elevated and vision is impaired, ischemic neuropathy and nerve compression are the only considerations. In a young person without vasculitis, a compressing lesion must be ruled out. Atypical visual field loss is always a matter for concern, but certain patterns warrant investigation. Involvement of the optic chiasm strongly suggests a compressive lesion because of the infrequency of chiasmal involvement by optic neuritis or ischemia. Indications of chiasmal involvement include bitemporal visual loss, monocular temporal defects, and involvement of the temporal field of the opposite eye in the presence of a central loss or total visual loss in the eye with the major manifestations.

Indications of an orbital mass include parallel retinal folds (produced by a mass close enough to the globe to indent it) and the presence of increased resistance to retrodisplacement of the globe ("tight orbit"). These findings dictate further evaluation. Neurologic abnormalities should be sought and, when present, require prompt, thorough evaluation. These include extraocular muscle palsies, frontal lobe symptoms and signs, anosmia due to olfactory tract involvement, and papilledema in the opposite eye with optic atrophy in the involved eye (Foster-Kennedy syndrome). Hypothalamic dysfunction should alert one to the presence of a mass arising from or impinging upon this region. Endocrinologic abnor-

Table III

FINDINGS INDICATING THE NEED FOR FURTHER INVESTIGATION

1. Gradual progression
2. Temporal visual field defects (unilateral or bilateral)
3. Atrophic disk which is elevated
4. Retinal folds when there is little or no disk swelling
5. "Tight orbit"
6. Proptosis
7. Bruit
8. Associated neurologic or endocrinologic findings
9. Systemic disorders known to be associated with compressive lesions (neurofibromatosis, bone dysplasias)

TABLE IV

COMPRESSIVE LESIONS[5,13]

1. Orbit—within muscle cone
   a. Meningioma
   b. Optic glioma
   c. Miscellaneous
      1. Cysts (dermoid, epidermoid)
      2. Metastasis
      3. Orbital ophthalmic artery aneurysm
      4. Hemangioma
      5. Lymphangioma
      6. Sarcoma
      7. Benign connective tissue tumors
2. Optic canal
   a. Meningioma
   b. Optic glioma
   c. Metastasis
   d. Hyperostotic narrowing
      1. Chronic inflammation
      2. Paget's disease
      3. Fibrous dysplasia
      4. Osteopetrosis
      5. Miscellaneous bone dysplasias
   e. Bone tumors
      e.g., eosinophilic granuloma
   f. Ophthalmic artery aneurysm
   g. Invasion by intracranial cysts and neoplasms
3. Intracranial
   a. Masses arising from sella, sphenoid bone, sphenoid sinus
      1. Pituitary adenoma
      2. Craniopharyngioma, cysts
      3. Carcinoma
      4. Metastasis
      5. Chordoma
      6. Chondroma, osteochondroma
      7. Mucocele
   b. Meningioma
   c. Craniopharyngioma
   d. Aneurysm, arterial ectasia
   e. Optic nerve or optic chiasm glioma
   f. Enlarged third ventricle (displaced or midline)
   g. Frontal lobe tumor
   f. Metastasis
   i. Meningeal carcinomatosis, sarcomatosis, and lymphomatosis
   j. Ectopic pinealoma
   k. Hypothalmic tumor
   l. Chiasmal arachnoiditis

malities indicative of pituitary or pituitary stalk impairment are of similar importance. Finally, one's suspicion should be aroused in the presence of systemic disorders known to be associated with lesions which can compress the optic nerves. The most important entity in this category is neurofibromatosis in which neurofibroma, meningioma, and optic glioma are considerations. Bone dysplasias, particularly Paget's disease and fibrous dysplasia, may be associated with optic canal narrowing and optic nerve compression.

Having been persuaded by one or more of these factors to investigate the patient further, roentgenologic and allied disciplines are called upon. The investigative tools include: plain roentgenograms; tomography; ultrasonography; isotope scanning; orbital and cavernous sinus venography; arteriography; pneumotomography; and the more recently developed computerized tomography.[9] We have not used positive contrast orbitography. The decision as to what methods shall be used is determined by the suspected site of the compressive lesion. For this reason, we have divided compressive lesions into three categories by location: orbital; within the optic canal; and intracranial. Table IV lists most of the compressive lesions to be found in each location.

The findings on clinical examination and plain roentgenograms (skull, orbits, optic canals) which direct the examination to one of these three areas are listed in Table V. Disk swelling indicates a lesion lying somewhere from the optic canal to the globe, the closer to the globe the greater the likelihood and degree of swelling. The one exception to this is the meningioma which can, on occasion, arise on the sphenoid wings and seed along the optic nerve as noted previously. A "tight orbit" (increased resistance to retrodisplacement of the globe) can be due to a mass within the orbit. Sphenoidal meningioma can be an exception if it produces enough hyperostosis to give a tight orbit. The plain roentgenograms in such cases will reveal hyperostosis. Parallel folds in the retina suggest a mass indenting the eyeball. Abnormal plain roentgenograms, including calcifications and hyperostosis of bone at the orbital apex, can be seen in meningiomas of the orbit.

Occasionally, enlargement of the orbital end of the optic canal is present. Destructive bone changes may be present in metastatic orbital lesions (without exophthalmos) and with meningiomas.

Evaluation of orbital lesions includes orbital tomography and tomography of the optic canals. At our institution optic canal tomography is performed by hypocycloidal motion with films taken at 1 mm intervals along the axis of the optic canal. The patient is in the supine position. The head is rotated 37 degrees away from the side being examined, and the chin elevated so that the orbitomeatal line makes an angle of 20 to 30 degrees with the vertical. Both sides are examined. We believe that this is the easiest method for evaluating the entire extent of the canal at all levels. The orbital end of the canal is oval with the long axis vertically oriented, the intracranial end oval with the long axis horizontal, and the central portion round. Symmetry is important, greater than 1 mm difference between the two sides being significant. Enlargement of the orbital end of the canal may be present in lesions of the optic nerve located posteriorly in the orbit. Ultrasonography for orbital masses has become quite useful and has been of great assistance in some of our cases. Computerized tomography[9] shows considerable promise also in the evaluation of orbital masses. Both of these modalities, being noninvasive techniques, will become important methods of both screening and diagnosis as they become more widely available. Isotope scanning is allegedly useful in evaluating orbital lesions but has not proved useful in detecting small orbital lesions in our experience. Orbital venography, performed quite easily in most cases, is done at our institution *via* injection of a forehead superficial vein using 8 to 10 cc of water soluble contrast medium. Orbital venography has been of great assistance in evaluating orbital lesions in general, especially masses within the muscle cone where the superior ophthalmic vein runs along the superolateral margin of the optic nerve. We have obtained quite satisfactory examinations *via* forehead vein injection using a scalp vein needle and 8 to 10 cc of renografin 60 percent. Serial filming in the lateral, submentovertical, and anteroposterior projections (the latter with the chin extended) are obtained and subtraction technique used for each projection. Angular vein puncture and bilateral internal jugular vein catheterizations are alternative routes. The ophthalmic vein is divided into three portions,[11] the third division running within the muscle cone and passing through the superior orbital fissure to join the cavernous sinus. Excellent discussions of technique and interpretation are available in the literature.[4,11]

Carotid arteriography for orbital lesions is usually performed *via* the femoral route at our institution because of ease in positioning the patient for filming; the same projections are obtained as used in orbital venography. Magnification and subtraction

TABLE V

LOCALIZING FINDINGS

1. Orbit
   a. Disk swelling
   b. Retinal folds
   c. "Tight orbit"
   d. Proptosis
   e. Abnormal plain roentgenograms
      1. Calcifications
      2. Hyperostosis
      3. Bone destruction
      4. Abnormal orbital end of optic canal
2. Optic canal
   a. Disk swelling without tight orbit
   b. Unilateral optic atrophy without other findings
   c. Known bone dysplasia
   d. Abnormal plain roentgenograms—essentially the same as for orbital lesions
3. Intracranial
   a. Bilateral visual impairment or bilateral visual field defects
   b. Temporal field loss, unilateral or bilateral
   c. Optic atrophy or normal disk
   d. Neurologic or endocrinologic findings
   e. Abnormal plain roentgenograms
      1. Sellar enlargement or erosion
      2. Calcifications
      3. Hyperostosis
      4. Bone destruction

techniques are invaluable. The ophthalmic artery and its branches within the muscle cone are of central interest in lesions primarily affecting the optic nerve. Anatomy and discussion of pathologic alterations have been well discussed in the current literature.[11,18]

Lesions of the optic canal portion of the optic nerve must be considered possibilities in all cases of disk edema without evidence of an orbital mass (exophthalmos, tight orbit). Disk pallor likewise requires investigation of the optic canal region since atrophy occurs earlier with lesions of the canal than with intracranial lesions.

Finally clinical or roentgenologic evidence of bone dysplasia (Paget's disease, fibrous dysplasia, and others)[13] should direct attention to this region in patients with visual impairment. Plain roentgenograms will confirm such a lesion involving the optic canal region and tomography will demonstrate the actual narrowing of the canal.

Tomography, perpendicular to the axis of the optic canal, should be obtained whenever a compressive lesion is suspected, since orbital and intracranial lesions may alter the canal (at their respective ends) as will intrinsic lesions of the canal. The other examination of use in evaluating lesions of the optic canal is internal carotid angiography with magnification and subtraction techniques. In this location, the ophthalmic artery courses within the canal along the inferolateral aspect of the optic nerve and may show compression or encasement by lesions. Furthermore, orbital and intracranial extension of lesions may be detected in this fashion. Such extension, suspected by involvement of one end of the canal, should direct the physician toward procedures for evaluating that region. Sometimes orbital, optic canal, and intracranial regions must all be investigated in lesions located in this junctional area.

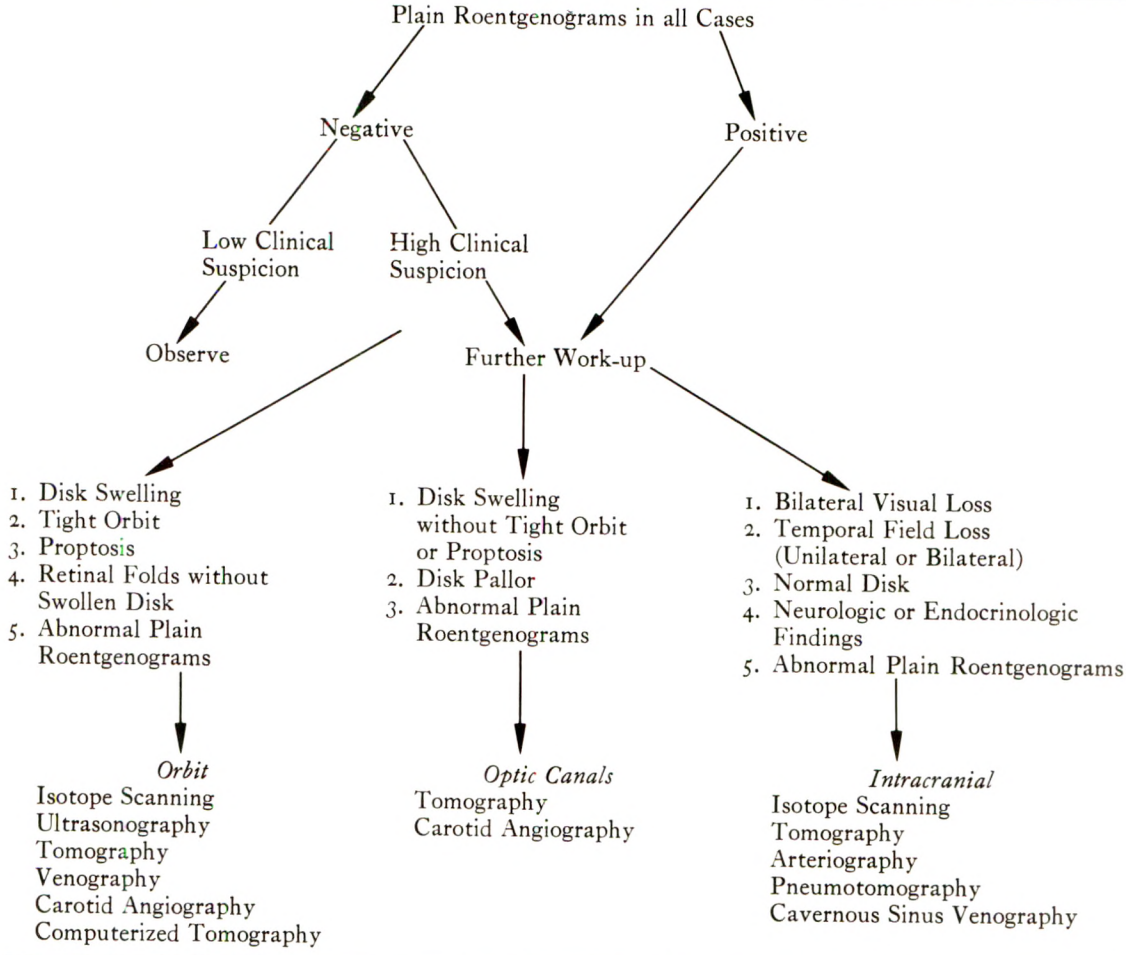
Intracranial roentgenologic investigation for suspected compressive lesions is indicated when the disk is pale but not elevated and when the disk is normal (the farther the lesion from the globe, the greater the delay in the appearance of atrophy). A temporal field loss (unilateral or bilateral) indicates intracranial localization close to or involving the chiasm. Bilateral visual loss logically indicates intracranial search for a single lesion; however, perioptic meningiomas in the region of the optic canal may be bilateral[2] and bone dysplasias can produce bilateral involvement (plain roentgenograms will detect this latter category). Neurologic or endocrinologic abnormalities discussed above also direct one to intracranial studies. Finally, abnormal plain roentgenograms are of great value in localizing lesions to this region. The sella may be enlarged by a mass arising from within it, or erosions may indicate a mass adjacent to or extending into it. Calcification, present in a variety of compressive lesions (craniopharyngioma, optic glioma, aneurysm, meningioma, etc.), may demonstrate the lesion and indicate its nature; in Case III the finding of calcification on a skull series was a major factor in leading to evaluation, since visual symptoms had abated. Hyperostosis and bone destruction may likewise direct one to an intracranial lesion.

Roentgenologic investigation for intracranial compressive lesions includes: tomography; isotope scanning; cerebral arteriography (with magnification and subtraction techniques); cavernous sinus venography (performed in the same manner as an orbital venogram); and pneumotomography. Cavernous sinus opacification may demonstrate lesions within, invading, or compressing the cavernous sinus. Pneumotomography, performed at our institution with hypocycloidal tomographic sections, has yielded exquisite demonstration of normal and pathologic anatomy; it is usually carried out in the brow-up anteroposterior, and lateral projections for evaluation of the region of the optic chiasm and nerves, but additional views (submentovertical, oblique) may be of additional value.

For an over-all view of our suggested ap-

TABLE VI

FLOW SHEET



proach to neuroradiologic evaluation of "optic neuritis" we have developed a flow sheet as demonstrated in Table VI. The radiologist can thus be of great help to the ophthalmologist in the management of patients with signs and symptoms of "optic neuritis." In dubious cases, he can either demonstrate a compressive lesion or establish that no mass lesion is present.[7,8]

Traditional teaching held that patients with slowly progressive unilateral visual loss should be explored even if roentgenologic studies are completely negative.[7] We agree with the contrary view, propounded by Knight *et al.*,[8] that negative neuroradiologic "exploration" using pneumotomography and magnification, subtraction angiography is sufficient to establish the presence or absence of an intracranial mass lesion. We are not certain that the results of neuroradiologic evaluation of the orbit warrants a similar conclusion.

SUMMARY

Optic neuritis can be simulated by lesions compressing the anterior visual pathways. The radiologist is in a position to assist in identifying such lesions or in ruling them out. While every patient in whom the diagnosis of optic neuritis is entertained should have plain roentgenograms of skull, orbits and optic canals, we have developed a

scheme for detailed neuroradiologic investigation based upon clinical and plain roentgenographic findings, illustrated by our cases and flow sheet. A classification of compressive lesions by location is illustrated.

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REFERENCES

1. Cogan, D. G. Neurology of the Visual System. Charles C Thomas, Publisher, Springfield, Ill., 1966, pp. 155–247.
2. Craig, W. M., and Gogela, L. J. Intraorbital meningiomas: clinicopathologic study. *Am. J. Ophth.*, 1949, *32*, 1663–1680.
3. Frisen, L., Hoyt, W. F., and Tengroth, B. M. Optociliary veins, disc pallor and visual loss: triad of signs indicating spheno-orbital meningioma. *Acta ophth.*, 1973, *51*, 241–249.
4. Hanafee, W. N. Orbital venography. *Radiol. Clin. North America*, 1972, *10*, 63–81.
5. Henderson, J. W. Orbital Tumors. W. B. Saunders Company, 1973, pp. 1–690.
6. Holt, W. F., Schlicke, B., and Eckelloff, R. J. Funduscopic appearance of nerve-fibre bundle defect. *Brit. J. Ophth.*, 1972, *56*, 577–583.
7. Jane, J. A., and McKissock, W. Importance of failing vision in early diagnosis of suprasellar meningiomas. *Brit. M. J.*, 1962, *25*, 5–7.
8. Knight, C. L., Hoyt, W. F., and Wilson, C. B. Syndrome of incipient prechiasmal optic nerve compression: progress toward early diagnosis and surgical management. *Arch. Ophth.*, 1972, *87*, 1–11.
9. Lambert, V. L., Zelch, J. V., and Cohen, D. N. Computed tomography of orbits. *Radiology*, 1974, *113*, 351–354.
10. Little, H. L., Chambers, J. W., and Walsh, F. B. Unilateral intracranial optic nerve involvement: neurological significance. *Arch. Ophth.*, 1965, *73*, 331–337.
11. Lombardi, G. Radiology in neuro-ophthalmology. Williams & Wilkins Company, Baltimore, 1967, pp. 9–19.
12. Moore, C. E. Sphenoid ridge meningioma with optic nerve metastasis. *Brit. J. Ophth.*, 1968, *52*, 636–639.
13. Netwon, T. H., and Potts, D. G. Radiology of the skull and brain. In The Skull. Volume 1. C. V. Mosby Company, 1971, pp. 463–567.
14. Schwartz, J. F., Chutorian, A. M., Evans, R. A., and Carter, S. Optic atrophy in childhood. *Pediatrics*, 1964, *34*, 670–679.
15. Smith, J. L., Hoyt, W. F., and Susac, J. O. Ocular fundus in acute Leber optic neuropathy. *Arch. Ophth.*, 1973, *90*, 349–354.
16. Sloan, L. L. Use of pseudo-isochromatic charts in detecting central scotomas due to lesions in conducting pathways. *Am. J. Ophth.*, 1942, *25*, 1352–1356.
17. Thompson, H. S. Afferent pupillary effects: pupillary findings associated with defects of afferent arm of pupillary light reflex arc. *Am. J. Ophth.*, 1966, *62*, 860–873.
18. Vignaud, J., Clay C., and Aubin, M. L. Orbital arteriography. *Rad. Clin. North America*, 1972, *10*, 39–61.
19. Walsh, F. B., and Hoyt, W. F. Clinical Neuro-ophthalmology. Third edition. Volume 1. Williams & Wilkins Company, Baltimore, 1969, pp. 60–95, 567–602, Volume 3, pp. 1927–2178.

Vol. 125, No. 3

BARIUM FINDINGS IN RETROGRADE INTUSSUSCEPTION*

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ABSTRACT:

Four instances of antiperistaltic intussusception are presented. These serve to depict the varied roentgen appearances in both types of intussusception on barium study and emphasize two simple interpretational principles: (1) when contrast material is administered orad to an antegrade or caudad to a retrograde intussusception, it must first enter the intussusceptum; and (2) when barium is administered orad to a retrograde or caudad to an antegrade intussusception, it must first enter the intussuscipiens.

Attention is also directed to tube related intussusceptions and to potential difficulty in their identification.

WE have recently encountered four examples of retrograde intussusception occurring in three patients. That experience affords the opportunity for describing and discussing the varied appearances of the parts or all of the intussusception complex depending on the direction of the intussusception and on whether barium is administered proximal or distal to it.

REPORT OF CASES

Case I. A 52 year old white female was admitted in the terminal phases of ovarian carcinoma, originally diagnosed two years previously. In the interim, she had been treated by surgery, radiation, and chemotherapy. Her chief complaints at the time of final admission were related to severe vomiting and abdominal distention. An upper gastrointestinal series revealed an intraesophageal mass that was clearly part of the stomach (Fig. 1, *A* and *B*) and an intraduodenal mass that was clearly a portion of jejunum (Fig. 2, *A* and *B*). At autopsy, the retrograde intussusceptions were corroborated and diffuse abdominal carcinomatosis was found. No intraluminal lesions were present.

Case II. A 68 year old white male was admitted to the hospital because of the recent onset of left lower abdominal pain and an episode of rectal bleeding one week prior to admission. Over the past few months, he had experienced some change in bowel habits. Barium enema examination and upper gastrointestinal series were unremarkable. Twenty-four hours later, the lower abdominal pain became severe. A plain roentgenogram of the abdomen revealed barium retained proximal to a concavity in the descending colon with collapsed bowel below (Fig. 3*A*). Hypaque enema examination revealed a sigmoidocolic intussusception. (Fig. 3, *B* and *C*). A leading lesion was suspected but not identified. At operation, the retrograde intussusception was easily reduced and an intraluminal tumor, histologically an adenomatous polyp, was palpated and resected. No other sigmoid abnormality was found.

Case III. A 98 year old male was admitted because of vomiting and abdominal pain. Pertinent past history included hemicolectomy 15 years previously for carcinoma. Plain roentgenograms of the abdomen revealed small bowel obstruction. At operation an internal hernia was found and reduced. One week after discharge, he was readmitted because of recurrent bowel obstruction. Decompression was attempted by means of a Miller-Abbott tube. The tube arrested in the left midabdomen and Gastrografin was injected, demonstrating a retrograde jejuno-jejunal intussusception (Fig. 4). Despite supportive therapy, the patient's condition deteriorated and death ensued. At autopsy, dilated bowel was found along the course of the tube but not distal to its tip.

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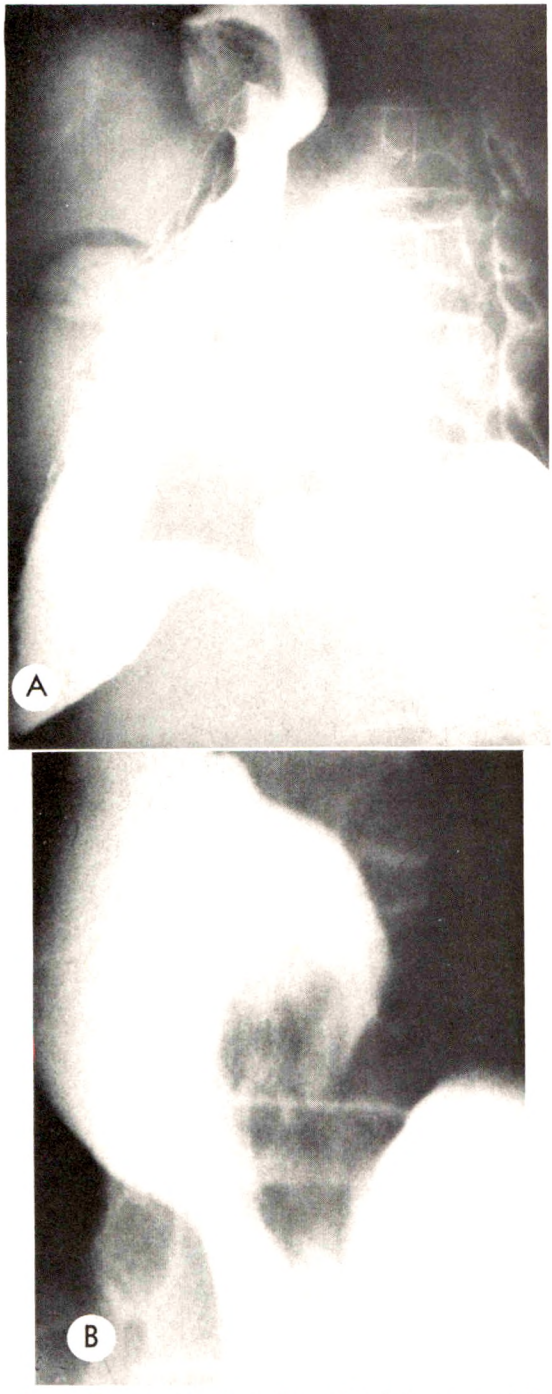

FIG. 1. Case 1. (*A*) Anterior wall of the stomach invaginating into the esophagus. (*B*) Spot roentgenogram. The distal esophagus (intussuscipiens) is dilated and contains a portion of the stomach (intussusceptum).

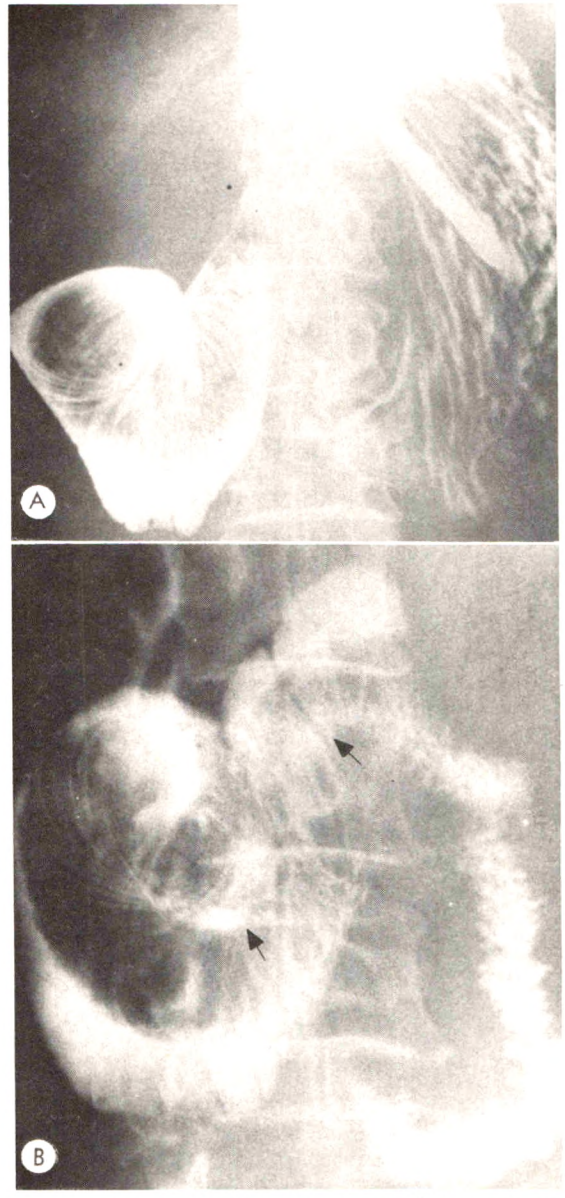

FIG. 2. Case 1. (*A*) Gastric mucosal folds converge toward that portion of its wall invaginated into the esophagus. The duodenum is dilated and has a coil-spring appearance caused by retrograde intussusception of the jejunum. (*B*) Spot roentgenogram. Barium has filled the lumen of the jejunal intussusceptum (arrow) and has passed into uninvolved bowel.

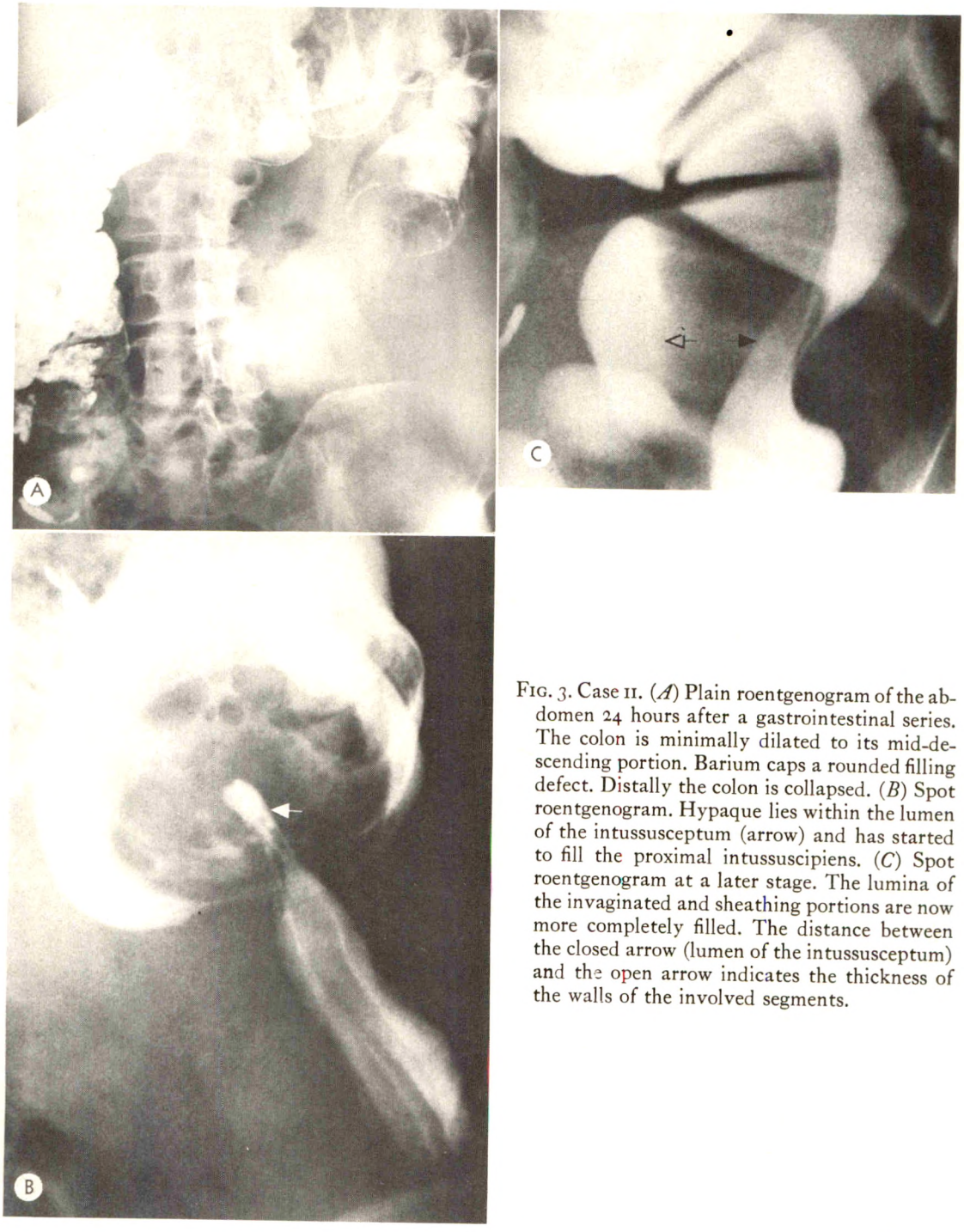

Fig. 3. Case 11. (*A*) Plain roentgenogram of the abdomen 24 hours after a gastrointestinal series. The colon is minimally dilated to its mid-descending portion. Barium caps a rounded filling defect. Distally the colon is collapsed. (*B*) Spot roentgenogram. Hypaque lies within the lumen of the intussusceptum (arrow) and has started to fill the proximal intussuscipiens. (*C*) Spot roentgenogram at a later stage. The lumina of the invaginated and sheathing portions are now more completely filled. The distance between the closed arrow (lumen of the intussusceptum) and the open arrow indicates the thickness of the walls of the involved segments.

ROENTGENOLOGIC FINDINGS

An intussusception, by definition, consists of a portion of the gastrointestinal tract that has invaginated (the intussusceptum) into another receiving or sheathing portion (the intussuscipiens). The roentgenologic findings depend on: (1) whether or not the intussusception is antegrade (isoperistaltic) or retrograde (antiperistaltic); (2) whether or not contrast material is

FIG. 4. Case III. Gastrografin injection into the lumen of the Miller-Abbott tube. The tip of the tube is within the lumen of the intussusceptum. Through proximal openings, contrast material fills the dilated intussuscipiens and produces a "coil-spring appearance." The lumen of the intussusceptum and normal distal bowel are also filled.

introduced into the intussusceptum or intussuscipiens; and (3) whether or not the lumen of the intussusceptum is filled or fillable (Fig. 5).

Barium introduced orad to an isoperistaltic intussusception or caudad to an antiperistaltic intussusception must first enter the intussusceptum (Fig. 3*B*). As contrast material enters the invaginated portion, it must flow into the compressed lumen. If the distal lumen is obstructed, contrast material will not pass into the intussuscipiens, and the bowel, proximal to the narrowed, invaginated segment, will demonstrate the typical features of obstruction, partial or complete. If the lumen is patent, the intussuscipiens fills (Fig. 3*C*), and is widened around the invaginated portion. If good coating of the mucosa of both the invaginated and sheathing portions is obtained, the so called coil spring appearance results. If significant obstruction exists, the bowel proximal to the intussusception complex is dilated and collapsed distally, regardless of whether or not the process is iso- or antiperistaltic.

Barium introduced caudad to an isoperistaltic intussusception or orad to an antiperistaltic intussusception (Fig. 2; and 4) must first enter the widened intussuscipiens in which the invaginated portion of bowel

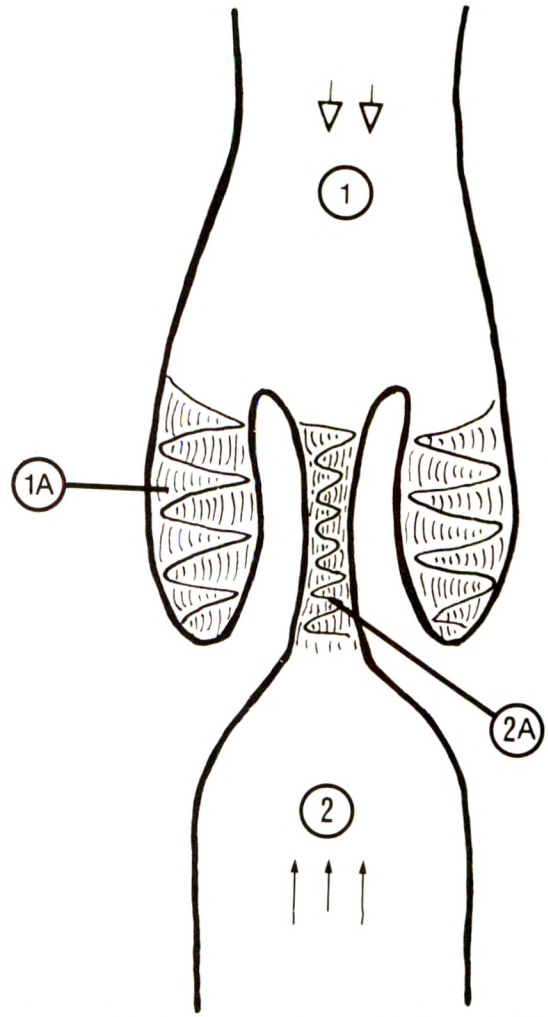

FIG. 5. Diagram of an intussusception complex. 1 = The intussuscipiens. 1A = The sheathing portion of the intussuscipiens. The combined mucosal walls of intussusceptum and intussuscipiens are responsible for the "coil spring." 2 = The intussusceptum. 2A = The lumen of the intussusceptum. Open arrows = Barium orad to a retrograde or caudad to antegrade must fill the intussuscipiens. Closed arrows = Barium orad to an antegrade or caudad to a retrograde must fill the intussusceptum.

produces the coil spring effect, with the important difference that passage of contrast material through the narrowed lumen of the intussusceptum is not required before it may be seen.

The roentgen appearance of gastroesophageal intussusception may differ from above descriptions because the entire circumference of the stomach need not invaginate. A portion of one wall of the stomach may intussuscept with mucosal folds converging toward it (Fig. 2*A*). The esophagogastric junction will, then, not appear within the esophagus. Rather, it will be in normal position with its opening narrowed by the gastric tissue passing through it (Fig. 1, *A* and *B*). Barium is obviously introduced into the intussuscipiens (the esophagus) and then demonstrates the usual features of a filled sheath. If the lumen between esophagus and stomach is obstructed, an esophageal obstruction with an intraluminal filling defect is seen. If the lumen is patent, it is possible to fill the stomach. Because only a portion of the gastric wall is involved, barium does not traverse a narrow central channel (lumen of the intussusceptum). It passes eccentrically around a defect, simulating retrograde prolapse of a polypoid gastric tumor.

When the entire intussusception complex is identified, only after it has been filled and outlined by barium, it is easy to determine that it is retrograde in type in the esophagus, duodenum and colon. The fact that the esophagus is proximal to the stomach is obvious. The second portion of the duodenum is fixed by its ductal attachments and therefore cannot intussuscept. Retrograde intussusception into it must be jejuno-duodenal. If an enteroenteric intussusception is first identified after the entire complex is filled, then it is not possible to determine whether it is iso- or antiperistaltic because of difficulty in identifying the proximal and distal ends (Fig. 6), unless, as in our third patient, a decompression tube is present. Despite the presence of obstruction, the tip of the tube may enter and pass through the lumen of the intussusceptum.

Fig. 6. Two areas of jejuno-jejunal intussusception. Because the complexes are completely filled, it is not possible to determine direction.

Barium, injected through it, will fill non-dilated bowel and progress normally. The intussusception is then not identified. The result is seemingly a paradox—the tip of the tube is in non-obstructed bowel, yet the bowel along the course of the tube is dilated. In our case, the intussusception was identified because some of the holes in the tube were proximal to the intussusception (Fig. 4). The colon is in a relatively fixed position and it is not difficult to distinguish between proximal and distal portions.

DISCUSSION

Retrograde intussusceptions are extremely rare and have been reported to occur in less than one percent of intussusceptions of all types.[3] Table 1 lists reported sites. Why they occur is unknown. Reverse peristalsis occurring because of distal obstruction or in the presence of bowel fixation by extrinsic inflammatory or neoplastic processes

TABLE I

TYPES OF RETROGRADE INTUSSUSCEPTION

1. Gastro-esophageal[1]
2. Duodeno-gastric[1]
3. Jejuno-duodenal (this article)
4. Enteric[1]
5. Caeco-ileal[1]
6. Colonic[1]
7. Jejuno-gastric[1]

might be causative. This might serve to explain our first case. Peritoneal carcinomatosis, however, is common while antiperistaltic intussusceptions are not. Further, the bowel wall was not fixed nor was there any distal obstruction in our second case.

Intussusceptions in both directions are known to occur in association with prolonged intestinal intubation, either with the tube present or after withdrawal.[2,4–6] When the tube is present, the distended balloon may behave in a fashion similar to an intrinsic tumor. Plications may develop along the mesenteric side of intubated bowel and these may be the inciting factor when intussusception occurs following tube removal.[4,5]

The coil spring appearance, described above, is not, in itself, diagnostic of intussusception. It is commonly seen when a dilated loop of small bowel presents itself on end. It is caused by the superimposition of a normal number of folds at varying depths. A "coil spring" can be seen only when barium is in the intussuscipiens. A central filling defect due to the intussusceptum should be present. These facts pertain regardless of the direction of the invagination.

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REFERENCES

1. AKHURST, A. C. Retrograde intussusception: report of case and review of 193 cases in literature. *Brit. J. Surg.*, 1955, *43*, 207–213.
2. DETERLING, R. A., O'MALLEY, R. D., and KNOX, W. Intussusception in adult, with emphasis on retrograde type. *A.M.A. Arch. Surg.*, 1953, *67*, 853–864.
3. FITZWILLIAMS, D. C. L. Pathology and etiology of intussusception from study of 1,000 cases. *Lancet*, 1908, *1*, 628–633 & 709–713.
4. GINZBURG, L., and FRIEDMAN, I. H. Delayed intussusception: following removal of long intestinal tube, with observation on its use. *New York J. Med.*, 1974, 2009–2013.
5. MOSCARELLA, A. A. Jejunal intussusception: unusual complication of use of intestinal tube. *Am. J. Surg.*, 1961, *102*, 83–85.
6. SOWER, N., and WRATTEN, G. P. Intussusception due to intestinal tubes. *Am. J. Surg.*, 1965, *110*, 441–444.

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ILEOCOLONIC PERFORATION*

A COMPLICATION FOLLOWING RENAL TRANSPLANTATION

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ABSTRACT:

During the decade, February, 1965, through February, 1975, 248 patients underwent renal transplantation at the Duke Medical Center. One hundred twenty-five were living related recipients, while 123 were cadaveric recipients. Eleven patients developed ileocolonic perforation. Six cases were felt to represent non-immunologic etiologies while the remaining five were felt to be associated with the allograft response and high dose steroid therapy. Only three of the patients survived this complication and in each case the perforation was localized. High dose steroid therapy interferes with prompt diagnosis and severely impairs successful treatment. Radiologists must be familiar with the problem of ileocolonic perforation in patients on steroid therapy, must have a high index of suspicion of the site of perforation, and must recommend and complete water soluble contrast examination for establishment of the diagnosis. Early diagnosis, prompt surgical intervention, and discontinuation of immunosuppressive therapy are recommended.

CONTINUED immunosuppressive therapy following renal transplantation is necessary for successful engraftment. Complications secondary to the prolonged use of immunosuppressive drugs in general, and high dosage steroids in particular, have been reported by many authors. The gastrointestinal complications following the use of these drugs have not gained wide recognition. Until recently, only occasional reference has been made to ileocolonic perforation as a complication following renal transplantation.[1,2,4,5,7,9,10] In most of the reports, the diagnosis was delayed and the patient died secondary to septic complications following the perforation. Radiologists, as well as all physicians, are responsible for the care of renal transplant patients and must be aware of the clinical presentation and the importance of contrast enema examinations in establishing early diagnosis since the mortality associated with a perforated viscus in the immunosuppressed patient is extremely high.

MATERIAL AND METHOD

During the decade, February, 1965, through February, 1975, 248 patients have undergone renal transplantation at the Duke Medical Center and the Durham Veteran's Administration Hospital. One hundred and twenty-five have had living related allografts and 123 have utilized a cadaveric source. Eleven patients developed ileal or colonic or combined perforations and form the clinical material for this report. The patients were maintained for varying periods of time on dialysis support prior to transplantation. Prior to 1967 only Azathioprine and prednisone were utilized as immunosuppressive agents. From 1967 to the present, the living related recipients were prepared and maintained on Azathioprine and prednisone with rejection episodes being treated by either high dose prednisone alone, or high dose prednisone in connection with irradiation to the allograft. The cadaveric recipients were prepared and maintained on Azathioprine,

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Supported in part by Clinical Research Center Grant, No. CA 11265 and Grant No. 5 POI AM13084-11, National Institute of Health.
‡ Picker Scholar, James Picker Foundation.

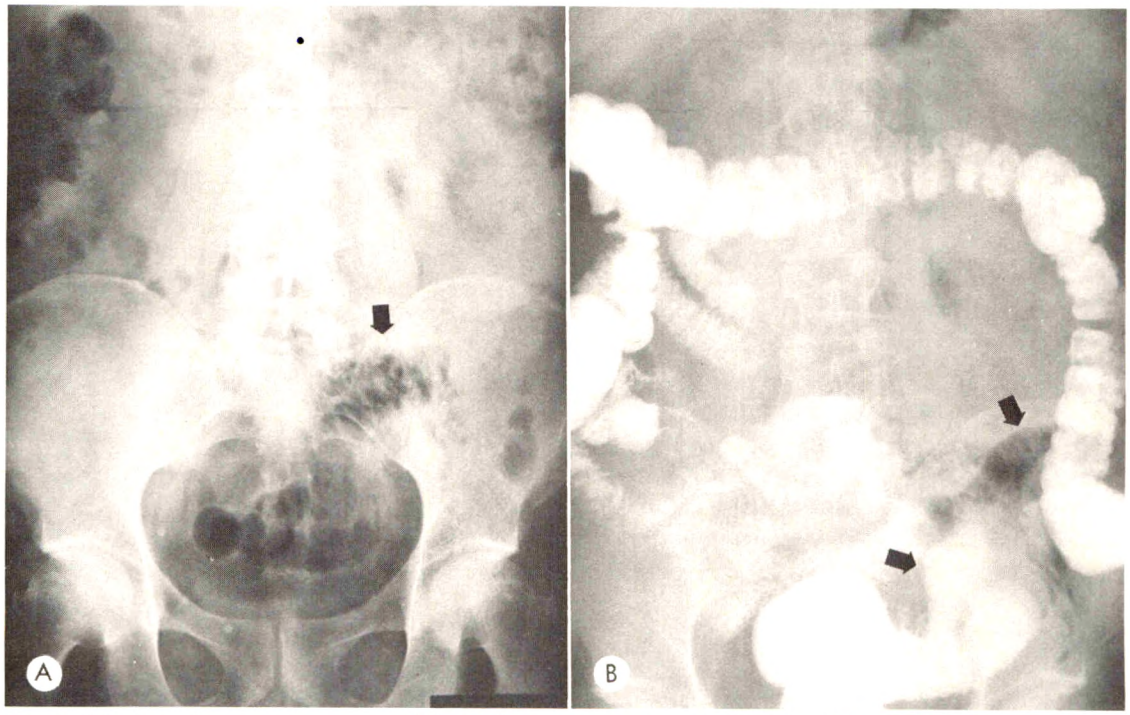

FIG. 1. (*A*) A supine roentgenogram of the abdomen on a 31 year old male with a five day history of left lower quadrant abdominal pain and fever. He had undergone a cadaveric renal transplant three years before. A peculiar irregular collection of gas is localized over the left sacroliac joint (arrow). (*B*) A hypaque enema demonstrates extravasation of contrast medium (arrows) into the abscess overlying the left sacroiliac joint. A few scattered diverticula are poorly demonstrated in the left colon. The patient survived following a colostomy and is alive three years later.

prednisone and anti-thymocyte globulin with rejection episodes being treated in the same manner as the living related recipients.

CLINICAL FINDINGS

Of the eleven patients who developed ileocolonic perforations, two were HL-A identical, three were HL-A haploidentical, and six were cadaveric recipients. Symptomatology in the group of patients who developed ileocolonic perforations was initially rather subtle. Mild abdominal tenderness, a febrile response, and some abdominal distention were present in each case. The time of perforation varied from less than one week post-transplantation to three years after allografting. In all cases the time between onset of the patient's symptoms and established diagnosis was at least 24 hours. The longest interval was 13 days. The patients were treated with either resection and bowel diversion or bowel diversion and drainage alone. A probable etiology for the perforations could be suggested in six of the patients.

Patient HH was receiving Kayexalate* enemas for the control of hyperkalemia and developed acute abdominal complaints with one of the rectal cycles. At operation there was a tear of the anterior wall of the rectum with free perforation. It was felt by his physician that the enema tube had perforated the rectum and the Kayexalate fluid had torn additional bowel wall, producing a free perforation into the peritoneal cavity (Fig. 3).

Patient WH had a peritoneal dialysis catheter injury with institution of his catheter for dialysis postoperatively. His five day dialysis was carried out uneventfully

* Sodium Polystyrene Sulfonate.

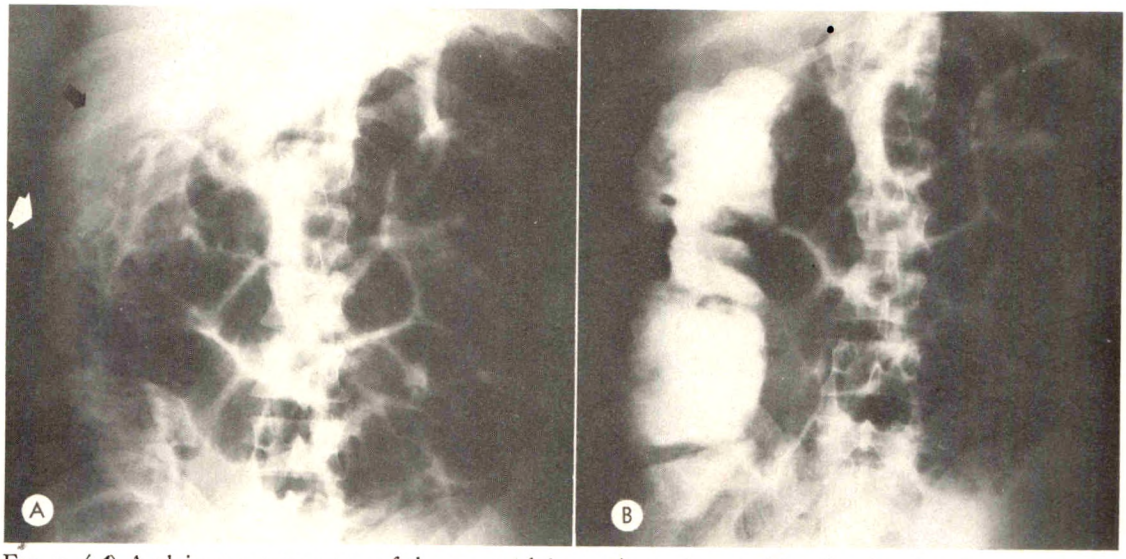

Fig. 2. (*A*) A plain roentgenogram of the upper abdomen in a 38 year old male who nine days previously underwent renal transplantation of a kidney from his sister. On the fifth postoperative day, he developed increasing abdominal distention, decreasing bowel sounds, and a rigid abdomen. A peculiar homogenous collection of gas is noted in the right upper quadrant which is outside the bowel (arrows); and there are bubbles of gas in the right pericolic gutter indicating an abscess. There is marked ileus. (*B*) The post-evacuation roentgenogram from a hypaque enema demonstrates intramural and extraluminal gas with abnormal mucosa of the ascending colon. At laparotomy, peritonitis and a right upper quadrant abscess were found secondary to necrosis of the right colon and terminal ileum. Pathological examination revealed ischemic necrosis of colon and ileum with multiple perforations. The patient died 48 hours later.

and the patient's symptoms of peritonitis did not develop until 48 hours following that particular cycle. The trocar perforation of the bowel could have contributed to the large area of necrosis.

Patient SA had a perforation of a sigmoid diverticulum into the mesentery. This did not represent a free perforation. Hypaque enema examination revealed a few other diverticula of the colon (Fig. 1).

Patient HW had a perforation of the colon at the hepatic flexure. His antacids had produced a massive fecal-antacid impactation which led to the subsequent perforation (Fig. 4).

Patients VH and LW had retroperitoneal abscess formation secondary to prior surgical procedures. In both patients a colocutaneous fistula developed secondary to the postsurgical abscess.

Four of the five remaining cases developed their ileocolonic complication while experiencing allograft rejection which was treated with high dose steroids. All five of these patients expired secondary to their complication. Table I summarizes the findings in each clinical case.

ROENTGENOLOGIC FINDINGS

The roentgenologic findings in the eleven patients are listed in Table II. In ten of the patients the upright and decubitus plain roentgenograms established a diagnosis of a perforated viscus with free air demonstrated in five and evidence of intra-abdominal abscess in five, while the eleventh patient (Fig. 4, *A* and *B*) had a severe ileus and fecal impaction. In this last patient an upper gastrointestinal examination performed six hours after the plain roentgenograms demonstrated free intraperitoneal air (Fig. 4*C*). Necrosis with free perforation of the right colon was demonstrated at surgical exploration.

Upper gastrointestinal examinations were performed in seven patients; one study con-

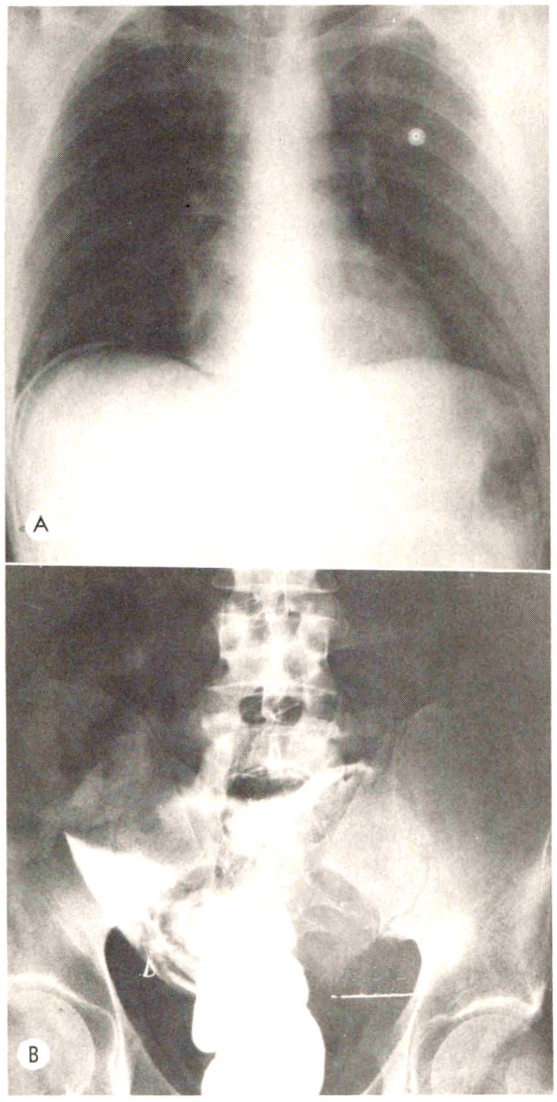

FIG. 3. (*A*) An upright chest roentgenogram on a 33 year old male shows free air under the right hemidiaphragm. The patient had received his second cadaveric renal transplant four days earlier. He had been receiving Kayexalate enemas for hyperkalemia in the postoperative period. (*B*) A hypaque enema demonstrated extravasation of contrast medium into the peritoneal cavity through an area of necrosis in the anterior rectal wall, just above the peritoneal reflection. The area of necrosis was approximately 4 cm in diameter. A sigmoid colostomy and oversewing of the perforation were performed; the patient expired on the twenty-seventh postoperative day.

firmed the diagnosis of abscess in the left upper quadrant. In the other six patients, the upper gastrointestinal series were performed to exclude a perforated peptic ulcer, and in each case the gastrointestinal tract was normal.

Water soluble contrast enema examinations were performed in six patients and demonstrated a perforation in five. One patient had a perforation of the anterior rectum (Fig. 3), which was thought to be secondary to the administration of Kayexalate enemas. One patient had a perforated sigmoid diverticulum, after the plain roentgenograms had demonstrated an abscess in the left lower quadrant (Fig. 1). A third patient had necrosis of the right colon and an abscess in the upper abdomen (Fig. 2). Patient VH, with a left upper quadrant abscess, had a contrast enema examination which demonstrated a perforation in the mid-descending colon. In the fifth patient, LW, a contrast enema examination demonstrated the abscess in the left abdomen which was related to a perforation of the descending colon. The sixth patient, SL, had a normal contrast enema examination four days prior to perforation of the sigmoid.

Two patients, (CB and WH), had perforation of the ileum with abscesses demonstrated on the plain roentgenograms. Water soluble contrast enema examinations and upper gastrointestinal examinations would not have determined the source of the perforations.

DISCUSSION

The over-all incidence of gastrointestinal complications requiring therapy in this series of 248 patients was 14 percent. Upper gastrointestinal peptic ulcer disease was diagnosed in twelve cases, and eleven additional patients experienced upper gastrointestinal hemorrhage secondary to stress ulceration. Only one patient developed free intraperitoneal air secondary to a perforated gastric ulcer.

The eleven cases of ileocolonic perforation represent a 4.4 percent incidence of

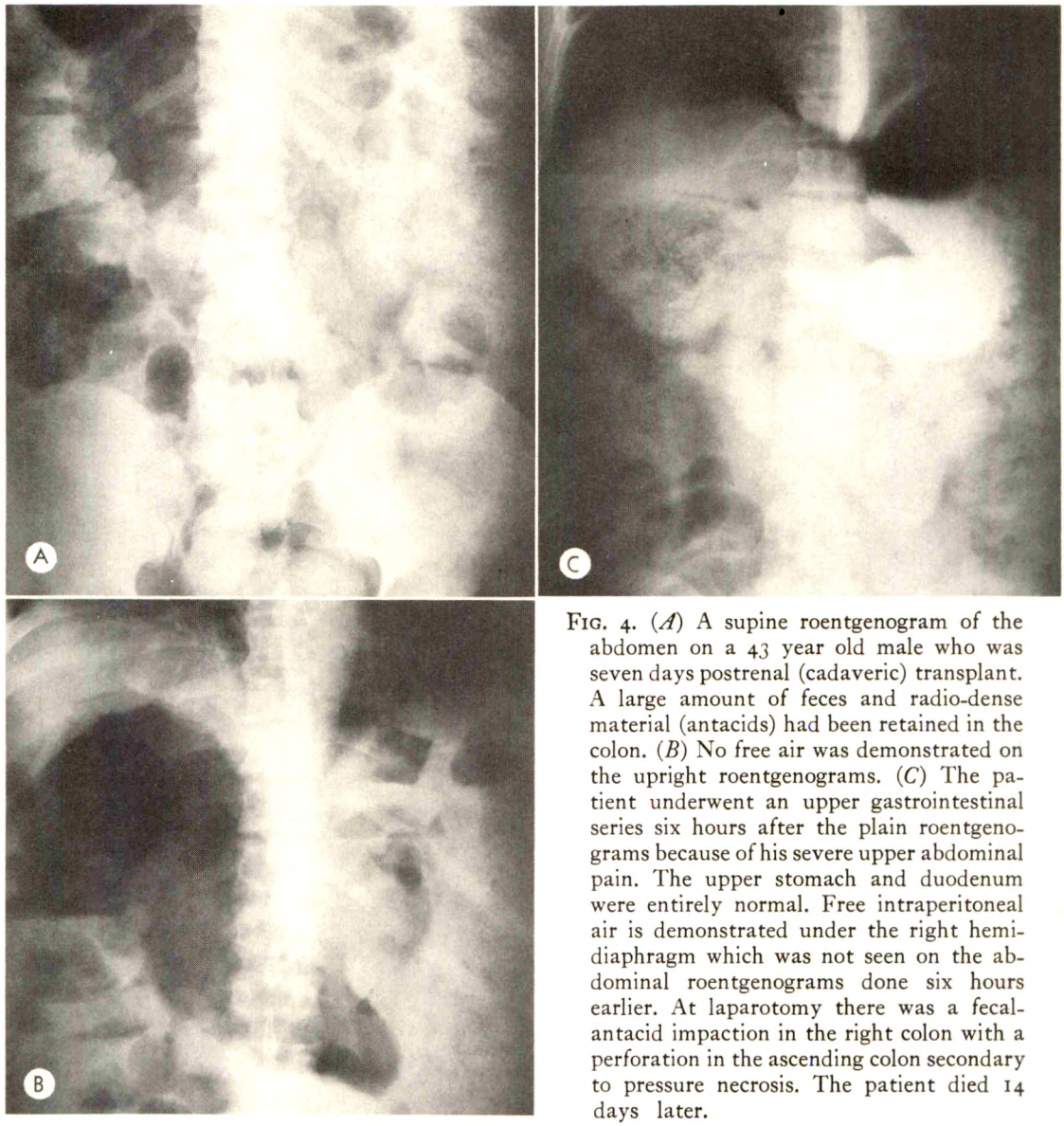

Fig. 4. (*A*) A supine roentgenogram of the abdomen on a 43 year old male who was seven days postrenal (cadaveric) transplant. A large amount of feces and radio-dense material (antacids) had been retained in the colon. (*B*) No free air was demonstrated on the upright roentgenograms. (*C*) The patient underwent an upper gastrointestinal series six hours after the plain roentgenograms because of his severe upper abdominal pain. The upper stomach and duodenum were entirely normal. Free intraperitoneal air is demonstrated under the right hemidiaphragm which was not seen on the abdominal roentgenograms done six hours earlier. At laparotomy there was a fecal-antacid impaction in the right colon with a perforation in the ascending colon secondary to pressure necrosis. The patient died 14 days later.

this complication. One patient had large bowel obstruction secondary to a bezoar with resultant perforation (Fig. 4). In two cases perforation was iatrogenic and in two cases colocutaneous fistula followed drainage of a retroperitoneal abscess secondary to prior surgery. One patient had a perforation of a pre-existing colonic diverticulum into the mesentery of the large bowel (Fig. 1). Four of the five remaining cases realized their ileocolonic perforation during an acute rejection episode that was being treated with high dose steroids.

Immunosuppressive agents may interfere with establishment of the proper diagnosis by altering physical signs, leukocyte response, and febrile response. The majority of the patients with a perforated viscus and not on immunosuppressive therapy will be diagnosed and treated within 24 hours. In contrast, patients following renal transplantation with perforation of the alimentary tract may not be diagnosed during the first 24 hours. In this

TABLE I

SUMMARY OF PATIENTS WITH ILEOCOLONIC COMPLICATIONS FOLLOWING RENAL TRANSPLANTATION

Case	Age	Sex	Source of Kidney	Time after Transplant	Time Between Onset of Symptoms and Diagnosis	Steroid Dosage at Time of Perforation	Nature of Gastrointestinal Problem	Treatment	Result
SA	31	M	Cadaver	3 years	5 days	35 mgm/da	Perforated sigmoid diverticulum-local abscess	Loop colostomy	Alive
CE	41	M	Haploidentical brother	18 months	4 days	30 mgm/da	Perforated sigmoid: peritonitis	Colostomy	Died
VH	26	F	HL-A Identical sister	2½ months	7 days	None	Perforated descending colon—left subdiaphragmatic abscess	Resecting colostomy	Alive
RM	39	M	Cadaver	11 days	5 days	200 mgm/da	Acute inflammation; perforated right colon; peritonitis	Resection colostomy	Died
LW	20	M	1st cadaver 2nd sister Haploidentical	9 months	13 days	35 mgm/da	Perforation, splenic flexure; fistula; abscess	Colostomy	Alive
HC	38	M	HL-A Identical sister	9 days	4 days	200 mgm/da	Necrosis and multiple perforations right colon; terminal ileum; peritonitis	Resection colostomy	Died
HH	33	M	Haploidentical 1st sister 2nd cadaver	3 days	2 days	60 mgm/da	Necrotic perforated rectum probably from Kayexalate enemas	Colostomy	Died
CB	23	F	Haploidentical mother	2 months	3 days	200 mgm/da	Perforation mid-ileum with necrosis and peritonitis	Perforation oversewn	Died
WH	38	M	Cadaver	10 days	2 days	60 mgm/da	Necrotic ileum; peritonitis (trocar injury)	Resection	Died
HW	43	M	Cadaver	13 days	2 days	60 mgm/da	Impaction right colon; necrosis; perforation; peritonitis	Resection colostomy	Died
SL	26	F	Cadaver	26 days	1 day	872 mgm/da	Perforated sigmoid colon	Drainage colostomy	Died

series, the average time for establishing the diagnosis was four days. In the majority of previous reports the patients had diffuse peritonitis rather than a localized abscess.[2,5,8,10,11] All three of the patients in this series who survived their perforations had a localized abscess either in the retroperitoneum or general peritoneal cavity. The eight patients who failed to survive had a free perforation with generalized peritonitis.

In each of the eleven cases the roentgenologic findings established the diagnosis of a perforated viscus. Thorough examination of the plain films is of utmost importance in all patients with abdominal pain, but is of particular importance in the postrenal transplant recipient, since many of these patients will have a moderate to marked ileus, and recognization of extraluminal gas may be difficult. When a large amount of free air is present the most likely diagnosis is ileocolonic perforation. It should be emphasized that the initial contrast study should be a water soluble contrast enema examination. If the enema examination is normal, an upper gastrointestinal series should then be performed. Water soluble iodinated contrast medium is recommended for both of these examinations. The iodinated contrast medium provides satisfactory roentgenographic contrast if low kilivoltage techniques are used. The Duke experience indicates that peritoneal soilage with water soluble contrast medium is less serious than when barium contaminates the peritoneal cavity. We have used the powdered form of Sodium Diatrizoate† for all water soluble contrast enema examinations. For an adult, 200 grams of the powder are mixed in an enema bag with 1,000 cc of tap water. This makes approximately a 20 percent solution. This

† Hypaque—Winthrop.

Table II

ROENTGENOLOGIC FINDINGS IN THE TEN PATIENTS WITH ILEOCOLONIC PERFORATION

Case	Plain Abdominal Roentgenograms	Contrast Enema Examination	Upper Gastrointestinal Examination
SA	Abscess left lower quadrant	Perforated sigmoid diverticulum	Not done
CE	Severe ileus, free air	Not done	Normal
VH	Abscess left upper quadrant	Perforation descending colon	Abscess documented
RM	Ileus, free air	Not done	Normal
LW	Ileus, abscess left mid-abdomen	Perforation descending colon	Normal
HC	Severe ileus, gas in bowel wall; abscess right upper quadrant	Perforation right colon	Not done
HH	Ileus, free air	Perforation rectum	Not done
CB	Ileus, abscess right upper quadrant	Not done	Normal
WH	Ileus, free air	Not done	Not done
HW	Severe ileus, massive fecal impaction	Not done	Normal, but free air on upright film
SL	Ileus, free air	Normal 4 days prior to perforation	Normal

mixture provides good contrast when roentgenographic techniques in the 75–80 kVp range are used.

In this series, as in others,[2,5,8,10,11] there was a wide range of time between renal allografting and the development of ileocolonic perforations. Some authors have suggested that there is a higher incidence of this complication following cadaveric renal transplantation than following consanguineous allografting. In this report, two patients were HL-A identical, three were HL-A haploidentical and six were cadaveric recipients. Non-immunologic etiologies were strongly implicated in six of the eleven patients. In four of the five remaining cases allograft rejection and high dose steroids were common to each case. A Shwarzman type phenomenon, immunologic induced vasculitis, and high dose steroid therapy have all been offered as explanations for ileocolonic perforations in renal transplant recipients.[2,5,6,8,10,11,15] There is increasing evidence that steroids are probably the most dangerous of the immunosuppressive drugs. Siegel[14] avoided any gastrointestinal complications in 60 postrenal transplant recipients by changing the daily standard dosage of prednisone while using other immunosuppressive agents in a conventional fashion. Administration of prednisone once daily progressing to alternate day steroid therapy was recommended in his report. Even though both the allograft response and chronic uremia[5,6,10,14] may predispose to ileocolonic perforation, high dose steroids would appear to be the most important causitive agent.

Colonic diverticular disease with perforation has been reported by others[3,12–14] in patients to whom steroids were being administered for reasons other than renal transplantation. Perforation of the colon in association with, or as a result of, fecal impaction secondary to antacid therapy has been reported.[2,7] Some authors[2,10] have suggested that cation exchange resins be cautiously used, that particular attention be given to proper colon preparation prior to transplantation, and that fecal impaction be arduously avoided during the postoperative period.

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REFERENCES

1. Aaron, K. E., and Dailye, T. H. Survival after colonic perforation of patient with trans-

planted kidney. *Dis. Colon & Rectum*, 1974, *17*, 103–105.

2. Bernstein, W. C., Nivatvongs, S., and Tallemt, M. B. Colonic and rectal complications of kidney transplantation in man. *Dis. Colon & Rectum*, 1973, *16*, 255–263.
3. Canter, J. W., and Shorb, P. E. Acute perforation of colonic diverticula associated with prolonged adrenocoticosteroid therapy. *Am. J. Surg.*, 1974, *121*, 46–50.
4. Demling, R. H., Salvatierra, O., and Belzer, F. O. Intestinal necrosis and perforation after renal transplantation. *A.M.A. Arch. Surg.*, 1975, *110*, 251–253.
5. Hadjiyannakis, E. J., Smellie, W. A. B., Evans, B. D., and Calne, R. Y. Gastrointestinal complications after renal transplantation. *Lancet*, 1971, *2*, 781–785.
6. Jaffe, R. H., and Laing, D. R. Changes of digestive tract in uremia. *Arch. Int. Med.*, 1934, *53*, 851–864.
7. Lipschutz, D. E., and Easterling, R. E. Spontaneous perforation of colon in chronic renal failure. *A.M.A. Arch. Int. Med.*, 1973, *132*, 758–759.
8. Misra, M. K., Pinkus, G. S., Birtch, A. G., and Wilson, R. E. Major colonic diseases complicating renal transplantation. *Surgery*, 1973, *73*, 942–948.
9. Penn, I., Groth, G. G., Brettschneider, L., Martin, A. J., Jr., Marchioro, T. L., and Starzl, T. E. Surgically correctable intra-abdominal complications before and after renal homotransplantation. *Ann. Surg.*, 1968, *168*, 865–870.
10. Penn, I., Brettschneider, L., Simpson, K., Martin, A., and Starzl, T. E. Major colonic problems in human homotransplant recipients. *A.M.A. Arch. Surg.*, 1970, *100*, 61–65.
11. Powis, S. J. A., Barnes, A. D., Dawson-Edwards, P., and Thompson, H. Ileocolonic problems after cadaveric renal transplantation. *Brit. M. J.*, 1972, *1*, 99–100.
12. Renshaw, T. S., and Phelps, D. B. A. Perforation of colonic diverticula. *J. Bone & Joint Surg.*, 1972, *54-A*, 1070–1072.
13. Sautter, R. D., and Ziffren, S. E. Adrenocortical steroid therapy resulting in unusual gastrointestinal complications. *A.M.A. Arch. Surg.*, 1959, *79*, 346–351.
14. Siegel, R. R. Gastrointestinal complications after renal transplantation. *Lancet*, 1972, *1*, 36–37.
15. Thoroughman, J. C., and Peace, R. J. Abdominal surgical emergencies caused by uremic enterocolitis: report of twelve cases. *Am. Surgeon*, 1959, *25*, 533–539.

Vol. 125, No. 3

SKELETAL ABNORMALITIES IN THE KNIEST SYNDROME WITH MUCOPOLYSACCHARIDURIA*

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ABSTRACT:

The roentgenographic and clinical findings are described in a mother and daughter with the Kniest syndrome associated with urinary keratan sulfate excretion. Osteoporosis, kyphoscoliosis, vertebral irregularity, pelvic deformity, flat femoral heads and enlargement of the ends of the long bones were the main roentgen findings. Irregularity of ossification on both sides of the growth plate was observed in the daughter, and marked degenerative changes were superimposed on several of the mother's abnormal joints. Abnormal mucopolysaccharidurıa, observed in both patients, and cataracts, fusion of the symphysis pubis, and deficiency of carpal bones, seen in the mother, have not been described previously.

IN 1952, Kniest[3] described an unusual form of short trunk dwarfism which later was considered to be a variant of metatropic dwarfism.[4,10] Recently Rimoin *et al.*[7] and Siggers *et al.*[9] have recognized the Kniest syndrome as a distinct entity.

The typical clinical features of the Kniest syndrome include short trunk dwarfism, kyphoscoliosis, enlargement of joints with limited motion, bell-shaped chest, round face with central depression, prominent eyes, myopia, deafness, cleft palate, retarded motor development, and recurrent respiratory infections. Short, deformed extremities and a relatively long trunk are seen early in life. Later in childhood there is disproportionate shortening of the trunk as kyphoscoliosis and lordosis develop.[9]

Most previous cases have been sporadic, and mucopolysaccharidurıa has not been reported. We have recently observed the Kniest syndrome in a mother and daughter who were found to excrete keratan sulfate in the urine.

CLINICAL FINDINGS

The proposita was a 13 year old Puerto Rican girl, product of a non-consanguineous marriage. Both parents were 29 years old at the time of her birth. At 18 months the diagnosis of Morquio's syndrome was entertained.

At 13 years both the height and weight were below the third percentile. The height was 129 cm and the span was 132 cm. The upper-lower segment ratio was 0.90. The facial appearance was striking with prominent eyes, epicanthal folds, mid-facial hypoplasia, and increased distance between the upper lip and nose. The teeth were malaligned. There was a marked thoracic kyphosis, and the lower ribs were flared laterally. Both the large and small joints of the extremities were enlarged, and joint motion was limited. There was bilateral hearing loss of sensory-neural origin in the left ear and mixed sensory-neural and conductive origin on the right. She was severely myopic, and the corneas were clear. Intelligence was normal.

The proband's 43 year old mother had a very similar physical appearance. The discrepancy between height (131 cm) and span (157 cm) was greater in the mother who had a more severe thoracic kyphosis. Deafness and bilateral cataracts were present.

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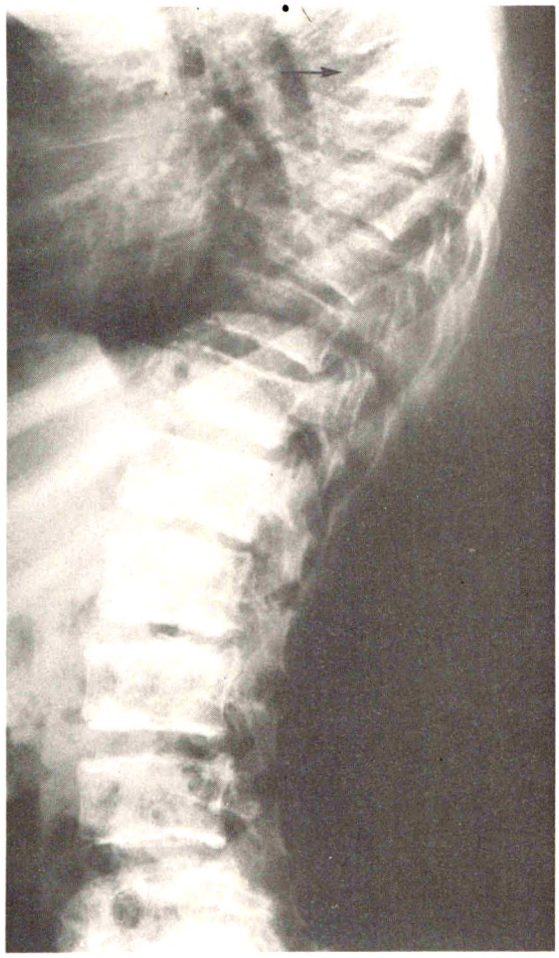

FIG. 1. Lateral view of the mid and lower thoracic and lumbar spine of the daughter. There is severe thoracic kyphosis with the apex at T6, which is wedged anteriorly (arrow). The vertebral bodies are flattened and irregular.

Both mother and daughter had abnormal mucopolysaccharidiuria. The acid mucopolysaccharide excreted was identified by Dr. Reuben Matalon, University of Chicago, as keratan sulfate. No dermatan sulfate was found in the urine. Routine blood chemical studies and urinalysis were normal. No inclusion bodies were found in peripheral leukocytes. Cultured skin fibroblasts in both patients were highly metachromatic. The lysosomal enzymes arylsulfatase A and B, alpha-glucosidase, alpha-galactosidase, alpha-iduronidase, alpha-mannosidase, alpha-fucosidase, beta-galactosidase, beta-glucuronidase and chondroitin sulfate N-acetyl hexosamine sulfate sulfatase were normal.

The father of the proband was phenotypically normal. The maternal grandparents were reported to be normal.

ROENTGENOGRAPHIC FINDINGS

In mother and daughter, the general configuration of the bones was strikingly similar. Consistent with her age, the daughter's epiphyses had not yet fused, and abnormalities on both the epiphyseal and metaphyseal sides of the growth plate were identifiable. Degenerative changes were superimposed on several of the mother's large abnormal joints.

Spine (Fig. 1–5). In the daughter there was generalized platyspondyly with irregularity of the superior and inferior surfaces of the vertebral bodies. The posterior portion of the body of L5 was wedged.

The mother's platyspondyly and vertebral irregularity primarily involved the thoracic spine. Her kyphosis was more severe and her disk spaces narrow. As with the daughter, the posterior portion of the

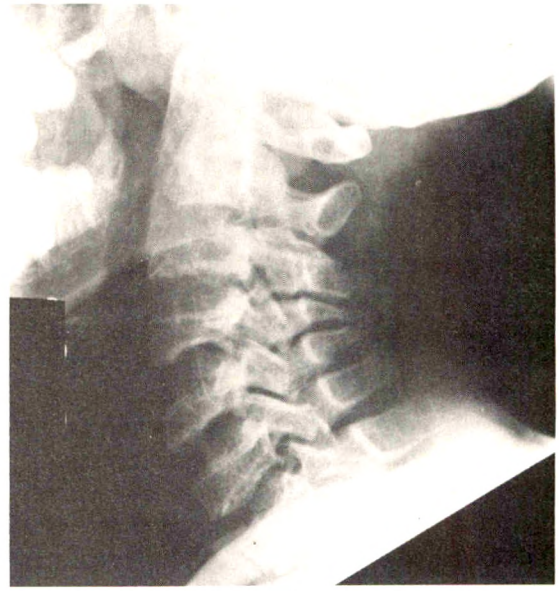

FIG. 2. Lateral view of the cervical spine of the daughter. The vertebral bodies are flat and irregular with wide intervertebral disk spaces. There is a mild increase in distance between the anterior arch of C1 and the odontoid.

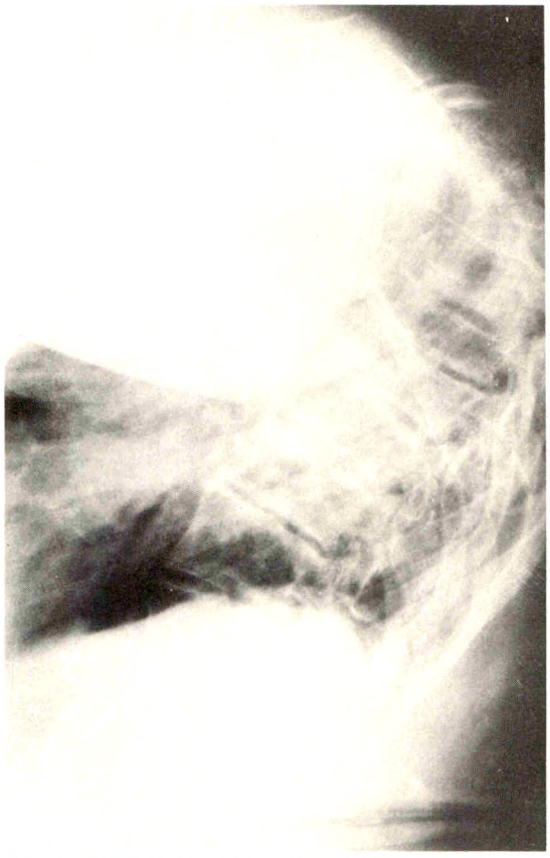

Fig. 3. Lateral roentgenogram of the thoracolumbar spine of the mother. There is a severe mid-thoracic kyphosis. Flattening and irregularity of vertebral bodies are more marked in the thoracic than lumbar spine. The intervertebral disk spaces are narrowed.

body of L5 was wedged.

Caudal narrowing of interpedicular distances was seen in both patients (Fig. 4; and 5). The daughter's coccyx was unusually large. The odontoid had a large antero-

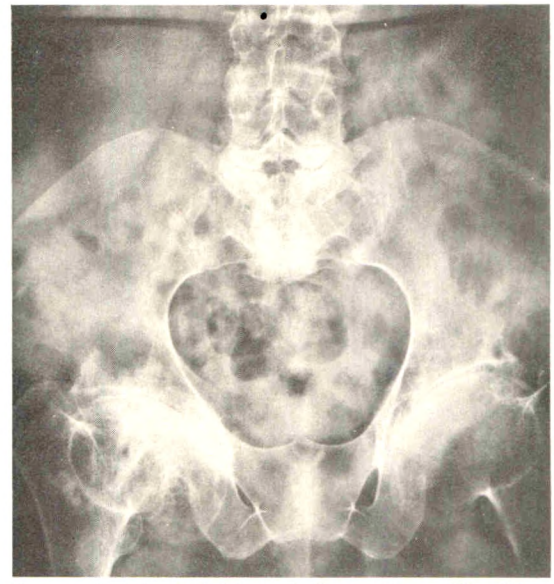

Fig. 4. Pelvis and hips of the mother. The iliac, pubic, and ischial bones are hypoplastic, and there is bony fusion of the symphysis pubis. The hip joint spaces are narrowed and sclerotic. No true femoral heads are distinguishable, and the femoral necks are thick and short with coxa vara deformities. There is caudal narrowing of the interpedicular distances of the lumbar spine.

posterior diameter in mother and daughter, but only in the daughter was there a mild increase in the distance between the anterior arch of C1 and the odontoid (Fig. 2).

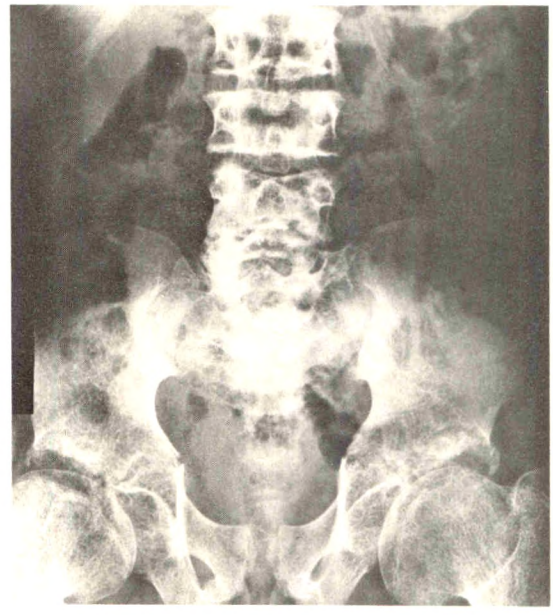

Fig. 5. Pelvis and lumbosacral spine of daughter. The shape of the pelvis is similar to that of the mother. The iliac, ischial, and pubic bones are all hypoplastic. The symphysis pubis is irregular but unfused. The ischiopubic synchondrosis is poorly ossified for the patient's age. The acetabula are irregular and the hip joint space narrowed. The femoral heads are extremely flat and wide, and there is bilateral coxa vara with marked thickening of the femoral necks. The interpedicular distances narrow caudally. The coccyx is large.

FIG. 6. The knee of the daughter shows increased density adjacent to the growth plates on both the epiphyseal and metaphyseal sides. The epiphyses and metaphyses are osteoporotic and wide. The proximal tibial metaphysis and growth plate are dome-shaped with corresponding central thinning of the epiphyseal ossification center.

Pelvis (Fig. 4; and 5). The general shape of the pelvis was similar in mother and daughter, with hypoplasia of the iliac, pubic, and ischial bones. The iliac bones had a square configuration.

There was complete bony fusion of the mother's symphysis pubis. Narrowing of the hip joint spaces and bilateral coxae varae with short, thick femoral necks were seen in both patients. In the daughter, the femoral capital epiphyses were extremely flat and wide, and in the mother there was no distinguishable transition between the femoral neck and head.

Long Bones (Fig. 6–9). The ends of the long bones were osteoporotic and bulbous. In the daughter, whose epiphyses were unfused, there was a relative increase in bone density adjacent to the growth plates on both the epiphyseal and metaphyseal sides. The proximal tibial (Fig. 6) and distal radial (Fig. 11) metaphyses were dome-shaped with corresponding central thinning of the epiphyseal ossification centers.

The mother had marked degenerative changes in multiple joints, including joint space narrowing of the hips (Fig. 4), ankles, knees, and elbows as well as marked productive changes at the elbows (Fig. 9). Multiple cyst-like lucencies were present in her distal femur.

Hands and Wrists (Fig. 10; and 11).

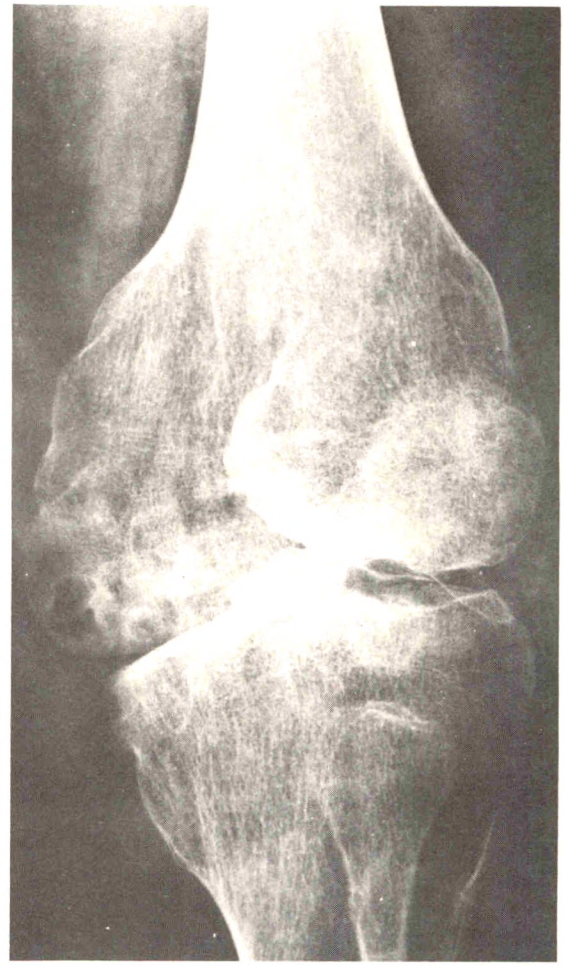

FIG. 7. Oblique view of the knee of the mother. The ends of the bones are expanded. Adjacent to the irregular articular surfaces are multiple cyst-like lucencies.

Mild narrowing of joint spaces and flattening of the metacarpal heads were seen in the daughter. In the mother these changes were much more pronounced, and the periarticular areas of the phalanges were flattened and osteoporotic as well. The daughter's carpal bones had angular contours, and in the mother the proximal row of carpal bones was deficient and the distal row deformed and small.

Feet (Fig. 12). Identical foot deformities were found in mother and daughter. The shafts of the second through the fifth metatarsals were abnormally thin, and the fourth metatarsal was shortened. In the daughter, the fourth metatarsal epiphysis was fusing while the others were still open.

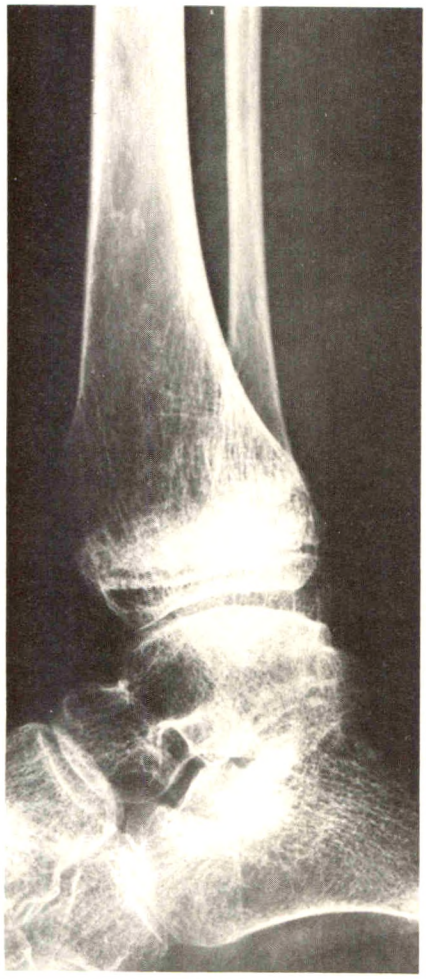

Fig. 8. Lateral view of the tibia of the daughter. There is amphorphous increased density in the metaphyseal region and osteoporosis of the epiphysis and diaphysis.

Fig. 9. Lateral view of the mother's elbow with osteoporosis, joint space narrowing and marked osteophyte formation.

Chest (Fig. 13). The chest deformity in both patients consisted of a narrow transverse diameter of the upper rib cage and

Fig. 10. The mother's hand shows generalized joint space narrowing and periarticular osteoporosis. Flattening at the ends of the bones is most pronounced at the metacarpal heads. The proximal row of carpal bones is deficient and the bones of the distal row abnormally small.

FIG. 11. The daughter's hand shows mild narrowing of joint spaces and flattening of metacarpal heads. The carpal bones are small and have angular contours. The dome-shaped distal radial metaphysis protrudes into the center of the epiphysis.

lateral flaring of the lower ribs. The orientation of the posterior portions of the ribs was more vertical than normal, secondary to kyphosis. The scapulae were hypoplastic.

Skull (Fig. 14). The over-all size and shape of the skull were normal. Each patient had a small sella turcica.

DISCUSSION

The proband and her mother had most of the clinical findings of the Kniest syndrome. However, cleft palate, usually present in this syndrome, was absent in our patients. Cataracts have not been seen in the previously reported cases. The proband had normal lenses while the mother had had cataracts since at least 25 years of age. It is not known whether the development of cataracts during the third decade is a manifestation of the syndrome, since the oldest previously reported patient was 26 years old.[5] Although most cases have been sporadic, there is evidence of dominant inheritance in another family with an affected mother and daughter.[5]

FIG. 12. Anteroposterior view of the left foot of the daughter. There is thinning of the shafts of the second through fifth metatarsals and proximal phalanges and shortening of the fourth metatarsal, the epiphysis of which is in a more advanced stage of fusion than the other epiphyses. The loose trabecular structure is well demonstrated in all the phalanges and metatarsals.

In our patients the roentgenographic findings generally resemble those previously described,[9] including generalized osteoporosis, bulbous ends of long bones, irregular trabecular structure with increased density on both sides of the region of the growth plate, vertebral flattening and irregularity, short chest, and small pelvis.

Several differences from the previous cases were noted. Bony fusion of the symphysis pubis as well as the severe carpal abnormalities of the mother have not been described before. Since these findings were not present in the daughter, they may be late manifestations. The daughter's symphysis pubis was unfused but irregular, and her carpal bones were small but all were present. The femoral capital epiphyses, usually absent throughout childhood in the Kniest syndrome, were present in the daughter, although very flat. Platyspondyly and cloudy calcification across the epiphyseal plate were less marked than usually seen in the syndrome.[8]

Of considerable importance in our cases is the urinary excretion of keratan sulfate.[2] This has not been reported in other patients with this syndrome, although not all have been tested.[8] Genetic heterogeneity in the Kniest syndrome is thus suggested, one form of the syndrome being associated with and the other without urinary keratan sulfate excretion.

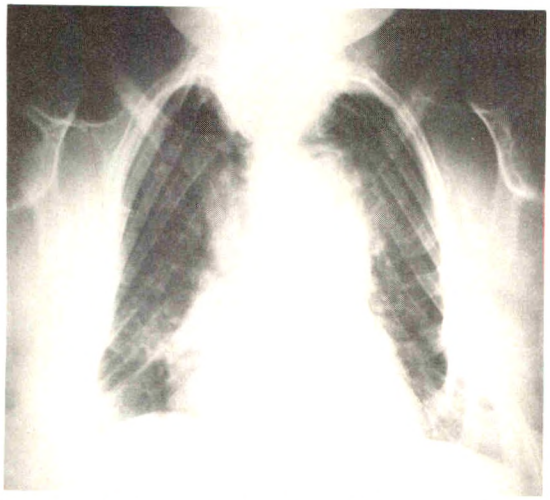

FIG. 13. Posteroanterior view of the chest of the mother. There is a narrow transverse diameter of the upper rib cage and lateral flaring of the lower ribs. The diagonal orientation of the posterior portions of the ribs is secondary to kyphosis. The scapulae are hypoplastic.

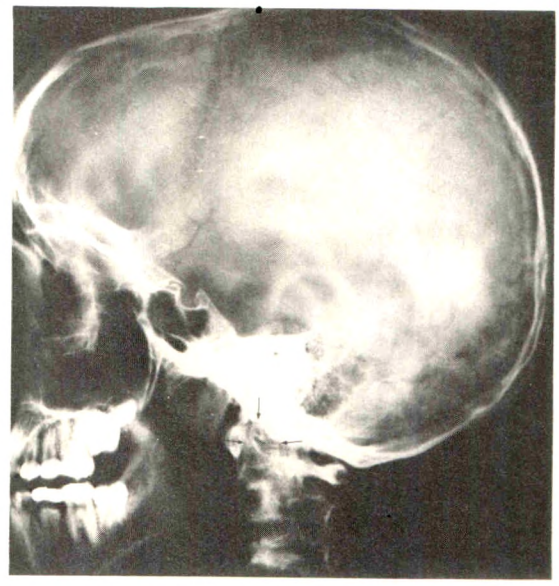

FIG. 14. Lateral roentgenogram of the skull of the daughter. The sella turcica is unusually small. The odontoid (arrows) is prominent.

The mucopolysaccharide excreted in our patients, with a dominantly inherited disease, is the same as that excreted in Morquio's syndrome, which is inherited as an autosomal recessive. It can be speculated that this variant of the Kniest syndrome is due to an enzyme inhibitor or to a mutation of a regulatory gene operating in the anabolic or catabolic side of keratan sulfate metabolism.

Skeletal abnormalities are present at birth in the Kniest syndrome but do not appear until early childhood in Morquio's syndrome.[1,6] Pelvic roentgenograms in Morquio's syndrome show supra-acetabular constriction, coxa valga, and a narrow femoral neck. In the Kniest syndrome coxa vara and a wide femoral neck are seen, and there is no supra-acetabular constriction. The apex of the kyphosis is near the thoracolumbar junction in Morquio's syndrome and was in the mid-thoracic spine in our patients, although Siggers *et al.*[9] have de-

scribed a patient with a lower kyphosis. In Morquio's syndrome the lumbar vertebral bodies tend to be more severely affected than the thoracic, and vertebral beaking is prominent. The thoracic spine is more severely affected in the Kniest syndrome. The hands of patients with the Kniest syndrome do not show the proximal metacarpal pointing of Morquio's syndrome.

Odontoid hypoplasia tends to be symptomatic in Morquio's syndrome and either absent or asymptomatic in the Kniest syndrome.[9] Our patients each have a thick odontoid of normal height. The daughter has slight increase in the distance between the anterior arch of C1 and the odontoid.

It is not yet known whether this variant of the Kniest syndrome can be distinguished clinically or roentgenologically from the type without abnormal mucopolysaccharidurıa, since few patients of similar ages have been studied roentgenologically, and mucopolysaccharide determinations have not been performed in all of them.

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The authors wish to express their appreciation to Dr. John Dorst, Dr. Ralph Lachman, and Dr. David Rimoin for their helpful discussions.

REFERENCES

1. Grossman, H., and Dorst, J. P. Mucopolysaccharidoses and mucolipidoses. *Prog. Pediat. Radiol.*, 1973, *4*, 495–544.
2. Kim, H. J., Beratis, N. G., Brill, P. W., Raab, E., Hirschhorn, K., and Matalon, R. Kniest syndrome with dominant inheritance and mucopolysaccharidurıa. *Am. J. Human Genetics*. In Press.
3. Kniest, W. Zur Abgrenzung der Dysostosis Enchondralis von der Chondrodystrophie. *Ztschr. Kinderh.*, 1952, *70*, 633–640.
4. Larose, J. H., and Gay, B. B. Metatropic dwarfism. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *106*, 156–161.
5. Maroteaux, P., and Spranger, J. La maladie de Kniest. *Arch. franç. pédiat.*, 1973, *30*, 735–750.
6. McKusick, V. A. Mucopolysaccharidosis IV. Heritable Disorders of Connective Tissue. Fourth edition. C. V. Mosby Company, St. Louis, pp. 583–605.
7. Rimoin, D. L., Hollister, D. W., Siggers, P., Silberberg, R., Lachman, R., McAlister, W., Kaufman, R., McKusick, V. A., and Dorst, J. Clinical, radiographic, histologic, and ultrastructural definition of Kniest syndrome. *Pediat. Res.*, 1973, *7*, 348.
8. Rimoin, D. L. Personal communication.
9. Siggers, D., Rimoin, D., Dorst, P., Doty, S., Williams, B., Hollister, D., Silberberg, R., Cranley, R., Kaufman, R., and McKusick, V. Kniest syndrome. In: Skeletal Dysplasias. Birth Defects: Original Article Series, The National Foundation-March of Dimes. Volume 10. 1974, no. 9, pp. 193–208.
10. Silverman, F. N. Discussion. In: Birth Defects: Original Article Series, The National Foundation-March of Dimes, Volume 5. 1969, no. 4, pp. 45–47.

Vol. 125, No. 3

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Published by: Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Illinois 62717.

Issued monthly, Annual subscription: United States, U.S. Possessions, U.S. Trusteeships, $30.00; Pan American Union and Canada, $35.00; other countries, $35.00. Current single numbers, $3.00. Advertising rates submitted on application. Editorial office, 401 Professional Building, Detroit, Michigan 48201. Office of publication, 301–327 East Lawrence Avenue, Springfield, Illinois 62717. Information of interest to authors and readers will be found on page ii.

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The Fifty-eighth Annual Meeting: Bayshore Inn, Vancouver, British Columbia, Canada, May 9–14, 1976.

EDITORIAL

INFORMED CONSENT AND THE RADIATION THERAPIST'S RESPONSIBILITY

DUE to the increased use of megavoltage radiation therapy in clinical oncology, a large number of patients are being exposed to high dose radiation therapy. Inevitably there will be complications from such treatment, particularly as the enthusiasm of fully trained radiation therapists increases in a desire to improve therapeutic results using higher doses. Such complications are, in essence, a natural sequence of radiation therapy, even with the finest equipment and the most flawless technique. It is a known fact that we are in the time of consumer protection and that we are dealing with a more informed and a more sophisticated society than has been previously seen. Therefore, potential litigation risks are obviously present.[1] A major thrust in many medical legal cases is in the direction of lack of informed consent. A recent case in California illustrates this point: A patient had gastric surgery. Postoperative bleeding necessitated a splenectomy. The patient sued on the grounds of malpractice, but a higher court ruled against actual physical malpractice but did rule in favor of the plaintiff due to the lack of informed consent about possible complications. Thus new standards of patient information have been established by the thrust of this case.[2]

What is informed consent? In essence, if a physician might possibly injure a patient he must tell the patient about the possible injury and have the patient's permission, presumably in writing. Consent for treatment from a patient is to be obtained before embarking on such a course of therapy. It is the physician's duty to obtain permission and he himself must inform the patient and not rely on his assistants.[3] Such consent must be explicit and not implied since radiation therapy is rarely an emergency procedure. A performance of such treatment, without due consent, is battery, and a procedure skillfully performed without such consent is still considered battery.[4] Therefore, absence of such consent renders a physician subject to liability from injuries from such treatment. Of course, such consent does not license a physician to do malpractice. It is the duty of the physician to inform the patient of all relevant information about a procedure so that the patient can make an intelligent decision on his own behalf, to accept or reject such treatment. Minor risks need not be discussed, but if there is a known risk, potential risk, or harmful effects the physician must disclose such in laymen's terms. Also the physician must disclose any additional pertinent information about the case as would a skilled practitioner in a similar situation On the other hand, a physician has no obligation to insure that the patient will recover from such a particular ailment.[5] A physician is not legally required to be infallible.[6]

The physician has no duty to disclose facts if such facts would so seriously upset the patient that he could not make a rational judgment, and thereby forego such treatment.[2] Frequently, slow gradual reve-

[1] EAGLETON, MARK and RAIN, J. R. Liability: what is your risk? ACR Bulletin, Nov., 1972, *28*.

[2] COBBS *vs.* GRANT. 502 P 2D, Cal., 1972.

[3] CRUM, J. F. Some advise on informed consent. CMA Special Report, Feb., 1973.

[4] RAMER *vs.* Community Memorial Hospital. 18 Cal App 3d 240, 1972.

[5] TEIG *vs.* Saint John's Hospital. 387 P 2d 527 Wash., 1964.

[6] BAJI. 6.11—1972 Revision, Civil Instructions: Reality of Consent—Physician's Duty of Disclosure.

lation of such facts to a frightened recalcitrant patient may well loosen his fears so that communication may be established. At no time should a physician start therapy until actual consent is obtained. If consent cannot be obtained, perhaps referral to another facility or to another physician should be made.

Up to this point legal considerations have been set forth that could well apply to any phase of medicine, not only radiation therapy. The following discussion will relate to the area of radiation therapy, but the principles might well apply to any branch of medicine.

It is philosophically difficult to reasonably inform a patient because words do not mean the same things to all people. Also a reasonable situation is admittedly hard to define. The authors do not intend to offer standards of conduct, but we do suggest an approach in the direction to the subject. Such an approach reflects the authors' experience in clinical radiation therapy and legal practice in southern California. We do not believe in defensive medicine, but we advocate an honest and open approach to medical practice. Such an approach would benefit the patient and doctor alike.

In order to inform a patient about radiation therapy, a description of how radiation therapy is to be given, revelation of the number of treatments, time required per session, and an explanation of the need for follow-up visits are absolutely basic. To reveal the common and immediate side effects from radiation therapy such as nausea, vomiting, diarrhea, cramps, mucous membrane reactions and possible skin reddening is also basic. Emphasis should be made that such reactions are normal and expected. It also should be made clear that the therapist is available to treat such side reactions. This information opens up a two-way street of communication based on care, trust and security.

To enumerate and describe major risks and complications of such treatment are the essence of informed consent.[7] It is suggested that simple laymen's language be used at all times. Words such as proctitis, cystitis, fibrosis, radiation ulcer and necrosis, etc. have little meaning to the layman, and hence invalidate informed consent in the eyes of the court. However language such as infection, inflammation, tissue death, tissue destruction, and ulcer formation carry an impact of information that can hardly be misrepresented. Also such words cover a vast gamut of possible reactions and obviate minute, verbose, and confusing information disclosure. A record of such conversation should appear as a notation on the patient's therapy chart.[8] This is to be considered in the course of daily business and further verifies such a conversation.

A signed and properly witnessed therapy authorization form is essential. The authors have seen verbose, technically difficult permit forms in small type that are confusing and practically useless as they carry no impact of real information. Hence it could be construed in the eyes of the court to be misleading, thus making the concept of informed consent invalid. Illustrated at the top of the next page is a form that is considered direct, simple and information giving.

Even if a patient has not indicated a desire for such information about medical risks before treatment, failure to reveal such risks may make a physician liable, solely on the basis that the patient may not have consented to the treatment if he had known about the risks.[2] The doctrine of informed consent imposes upon a physician the duty to disclose to his patient all relevant information concerned in a proposed treatment, including the collateral hazards attendant thereto, so that the patient's consent to treatment will be an intelligent one based on complete information.[9] However, no disclosure need be made if the patient requests that he not be so informed.[2] The authors suggest that in this

[7] Lopez *vs.* Sawyer, 279 A 2d 116, N. J., 1971.

[8] Elmendorf, T., and Ackerman, F. W. CMA Bulletin, Aug. 24, 1973.

[9] Nishi *vs.* Hartwell. 473 P. 2d 116 Haw., 1970.

CONSENT FOR RADIATION THERAPY

Permission is given to Dr. _____ to perform radiation therapy on _____.

The type of reactions from the treatment and the nature of major risks and complications have been explained to me. These can include: skin irritation, organ injuries, scar tissue formation, tissue destruction, infection, bleeding and inflammation. Such reactions can occur during radiation therapy or a long time after completion of such treatments. Since radiation therapy is an art and not an exact science I understand that no promise of cure or freedom from risk can be given.

All efforts possible are taken to provide for the ultimate care of the patient. Calculations of radiation doses are done by specialists trained in these arts.

During the course of treatment, I will report any symptoms if they become bothersome.

I have read the above and have no further questions about the treatment to be given.

Witness	Name
Date	Signature

case a note appear on the patient's therapy chart indicating his request with the signature of a witness. In regard to witnesses, it is suggested that a permanent employee of the x-ray department, preferably the treating technician, witness all forms. This would demonstrate a continuity in the disclosure procedure.

Regarding the validity of a permit for a form of treatment, a quotation from the Georgia State Medical Consent Law is documentary: "A consent to medical or surgical treatment which discloses in general terms the diagnosis or course of treatment in connection in which it is given and which is duely evidenced in writing, and signed by the patient, person or persons authorized to consent persuant to the terms thereof, so be concisely perceived to be a valid concept in the absence of fraudulent or misrepresentation of material facts."[10]

Mills has suggested six steps to determine whether or not informed consent was given: (1) was the patient legally capable of giving medical consent; (2) was the patient mentally capable of such consent; (3) was the proposed procedure adequately identified for the patient; (4) was the patient aware of the identity of the person who was going to perform the procedure; (5) was there sufficient disclosure of the hazard of the proposed procedure to permit the patient to weigh the risks and benefits to himself; and (6) has there been sufficient disclosure of significant feasible alternatives to the proposed procedure to allow the patient to complete his own risk *versus* benefit determination.[11] The authors suggest that a radiation therapist be prepared to prove in court that each one of these questions was answered satisfactorily before administering treatment.

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[10] Title 88 Public Health—Georgia Medical Consent Law—Section 88—2906X—1971, pp. 348, 440.

[11] MILLS, D. M. *J.A.M.A.*, July 18, 1974, Vol. *229*, No.3.

NEWS ITEMS

PEDIATRIC AND NEWBORN RADIOLOGY SEMINAR

The University of Colorado School of Medicine announces the Fourth Annual Pediatric and Newborn Radiology Seminar to be held February 9 through 13, 1976, at the Given Institute of Pathobiology in Aspen, Colorado.

The Seminar covers all aspects of Pediatric and Neonatal Radiology including the latest advances in the field. The Course Director is Richard L. Wesenberg, M.D., Pediatric Radiologist, University of Colorado School of Medicine.

The seminar is approved for 15 hours of Category 1 credit for the AMA Physicians Recognition Award. The registration fee for the five days is $150.00.

Reservations and further information may be obtained by writing to: The Office of Postgraduate Medical Education, University of Colorado School of Medicine, Cont. C295, 4200 East 9th Avenue, Denver, Colorado 80220.

EUROPEAN SOCIETY OF CARDIO-VASCULAR RADIOLOGY

The next meeting of the European Cardio-Vascular Society will be held on May 13 through 15, 1976 in Lyon, France.

Topics will include: dysplasia of the renal artery; obstructive myocardiopathy; and automatic injection.

For further information, please contact: Professeur F. Pinet, Service de Radiologie, Hôpital Cardio-Vasculaire, B. P. Lyon Montchat, 69394 Lyon Cedex 3, France.

19TH ANNUAL POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY

This five day course will be presented in San Francisco, California, March 15 through March 19, 1976.

The course is designed to provide a survey of selected subdivisions of diagnostic radiology for the radiologist in clinical practice. The faculty of this Course is comprised of faculty of the Department of Radiology, University of California School of Medicine, San Francisco and an international Guest Faculty. The complete fee of $225 includes tuition and hardbound publication of proceedings, replete with illustrative photographs. Interns, Residents and members of the Armed Forces will be admitted for $150 with a letter of verification.

For further information, please write to: Extended Programs in Medical Education, Room C-135G, University of California, San Francisco, California 94143.

POSTGRADUATE COURSE IN GYNECOLOGIC ONCOLOGY

A Postgraduate Course in Gynecologic Oncology is presented by the Women's Hospital Medical Center, Long Beach, California, January 16–18, 1976.

This two and one-half day program is designed to acquaint the practicing gynecologist, radiologist, pathologist, and medical oncologist with recent advances and current concepts in the diagnosis and management of gynecologic cancer. In addition to formal presentations by a distinguished faculty, ample time for informal question and answer periods has been provided to encourage maximal discussion between the faculty and participants.

This continuing medical education activity is acceptable for 15 hours of Category 1 credit toward the Physicians' Recognition Award of the AMA.

Further information may be obtained from the Executive Secretary, Center for Health Education, 2801 Atlantic Avenue, Long Beach, California 90801.

SIXTH ANNUAL ASPEN RADIOLOGY CONFERENCE

The Sixth Annual Aspen Radiology Conference will be held at the Aspen Institute for Humanistic Studies, Aspen, Colorado, March 1–5, 1976.

The conference is designed for physicians and scientists interested in diagnostic radiology, nuclear medicine and radiation therapy, and will explore the impact of clinical and technological advances on radiologic practice.

The topics for discussions will include advances in cardiovascular, gastrointestinal, bone and neuroradiology involving a tri-radiological approach. Each morning session will survey the advances in a single radiology subdivision as a refresher course with independent parallel diagnostic, nuclear medicine and therapy sessions. Instructive cases, illustrating these topics and previewed by the audience, will be presented for open discussion in the afternoons.

Sessions will be held in the early morning, late afternoon and evening allowing adequate time for leisure activities such as skiing and other winter sports.

For further information, please write to: Aspen Radiology Conference, Beth Israel Hospital, 1818 Gaylord Street, Denver, Colorado 80206.

DIAGNOSTIC AND THERAPEUTIC ANGIOGRAPHY

The Society for Cardiovascular Radiology is sponsoring a four day postgraduate course in angiography on February 17–20, 1976, at the El-Conquistator Hotel and Club in Puerto Rico.

A series of lectures dealing with current concepts of diagnosis and therapeutic uses of angiography will be presented by the faculty. They will lecture on the techniques, diagnostic findings, interpretation, potential complications, advantages and limitations of the different procedures. The lectures will be followed by periods of open discussion allowing the registrants to participate.

The course has been accredited by the American College of Radiology for 20 credit hours of AMA Category 1.

For further information, please write to: Department of Radiology, University of Utah College of Medicine, Salt Lake City, Utah 84132.

DETECTION AND TREATMENT OF EARLY BREAST CANCER

The Fifteenth Annual Conference on Detection and Treatment of Early Breast Cancer, including the Fourth Annual Wendell G. Scott Memorial Lecture, will be held at Caesars Palace, Las Vegas, Nevada, March 8–12, 1976. The conference is sponsored and arranged by The American College of Radiology and The Commission on Cancer, Division on Diagnosis and the Committee on Mammography and Diseases of the Breast; and supported by The American Cancer Society, and The American College of Radiology.

For further information, please write to: The American College of Radiology, 6900 Wisconsin Avenue, Chevy Chase, Maryland 20015.

POSTGRADUATE VIDEOTAPE COURSES IN DIAGNOSTIC RADIOLOGY

The Department of Radiology, University of California San Francisco, is offering a series of Videotape courses in various aspects of diagnostic radiology. These Postgraduate Videotape Programs are comprehensive and represent the current state of the art. An outstanding group of guest lecturers, in addition to the staff at the University of California, teach basic approaches to radiographic interpretation and technique. Current concepts and controversies highlight these topics.

The following courses are presently available on videotape:

1. Neuroradiology didactic lectures—25 hours
2. Neuroradiology conferences and seminars—25 hours
3. Computerized tomography—25 hours
4. Gastrointestinal radiology—25 hours
5. Pediatric radiology—25 hours
6. Cardiopulmonary radiology—12 hours

7. Genitourinary radiology—12 hours
8. Skeletal radiology—12 hours
9. Diagnostic ultrasound—12 hours
10. Nuclear medicine—12 hours
11. Technical aspects of radiology—12 hours

Category 1 credit for AMA Physicians Recognition Program may be obtained.

For further information, please write to: Videotape Course Coordinators, T. H. Newton, M.D., or Henry I. Goldberg, M.D., Department of Radiology, University of California, San Francisco Campus, San Francisco, California 94143.

BOOK REVIEWS

Tomography and Cross Sections of the Ear. By Galdino E. Valvassori, M.D., and Richard A. Buckingham, M.D. Cloth. Pp. 200, with 510 illustrations. Price, $65.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1975.

Many of you may not remember that knowledge and understanding of anatomy do not come easily. Compounding the complexity comes with correlating the three dimensional reality with two dimensional representations. The authors of this beautiful book have made available the tools necessary for attaining both the knowledge and the correlation.

This is an elegant, leisurely, complete atlas of the tomographic anatomy of the temporal bone. It is written from the point of view of both the radiologist and the surgeon. Tomographic sections of the isolated normal temporal bone were made at 1 mm intervals through the bone. The temporal bone was then decalcified and cut into 2 mm slices, in the same plane as the tomograms. The upper and lower surfaces of each of these slices were photographed in color, and each photograph is presented with (1) each corresponding tomographic cut, (2) a microroentgenogram of the slice, (3) a well-labeled drawing of the visualized structures, and (4) an elaborate word description. This procedure was carried out in each of six different roentgenographic projections, and required between 12 and 18 tomographic exposures for each projection. The reproduction of the illustrations is superb and they are life size.

The normal anatomy occupies somewhat more than one-half of the book. Selected pathological temporal bones are examined and presented in a similar manner. These demonstrate congenital abnormalities, acute and chronic inflammatory lesions, and postoperative and degenerative changes.

This volume is a complete atlas of tomograms and is a definitive study of this type. It will be a vast mine of information for the young radiologist who wants to learn; a convenient, well organized accurate reference for the sometime otologist or otologic radiologist; and the polish and finishing touch to the complete otologic radiologist.

E. Frederick Lang, M.D.

The Biology and Clinical Management of Bladder Cancer. Edited by E. H. Cooper, M.D., D.Sc., D.Phil., F.R.C.P., Mackintosh Professor of Experimental Pathology and Cancer Research, University of Leeds; and R. E. Williams, M.D., Ch.M., F.R.C.S.E., Consultant Urologist, General Infirmary at Leeds, England. Cloth. Pp. 293, with 52 illustrations. Price, $38.75. Blackwell Scientific Publications, Oxford, London, Edinburgh, and Melbourne. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1975.

Within this book of eleven chapters, three appendices, 293 pages, and contributions by eight international authors (two American, one Swedish and the remainder English) the editors have incorporated a large sum of information and material gathered over many years of investigation of bladder cancer.

As stated in its preface, "the management of this form of cancer is controversial" and the purpose of the book is to bring up to date "what can (sic) and has been done in the management of bladder cancer."

The first chapters dealing with present day experimentation, tumor biological characteristics, and epidemiology express the fact "we are" strongly "becoming aware that many cases of bladder cancer are no act of God but a disease which stems from environment."

The middle chapters dealing primarily with bladder cancer pathology are well written and summarized, and they point out particularly the variable behavior and types of bladder cancer, and the pathologists' enigma in stating diagnosis, prognosis, and course to the clinician. Perhaps by inference but nonetheless noticeable is the difference that exists in medical care and delivery between the United States and socialistic medicine in Great Britain. One whole chapter in a short book is devoted to "Cytology in the Diagnosis of Bladder Cancer" as an indirect method of postoperative bladder cancer follow-up and an industrial screening procedure. The accepted and more definitive procedure for these situations in the United States is cystoscopy. One can speculate at length on the reasons fostering the more indirect follow-up approach.

Advances in immunology in relation to bladder cancer are presented in one chapter. Though yet investigative, the findings may explain that the "subtle differences in tumor behavior are due to the uniqueness of individual host-tumor relationship."

The last chapters of diagnosis and therapy are based upon today's knowledge. They are critically discussed as to their implementation and to their limitations. The chapter on "Total Cystectomy" gives an excellent rational approach to therapy based on physical, laboratory, and histological criteria of staging and grading. It is a pleasant article to read, as the author departs from the usual manner of listing references within the body of the text, and lists them only at the end of the article.

The last chapter deals with the rarely encountered but interesting phenomenon of "Total Urothelial Neoplasia," and tries to explain pathogenicity on the basis of knowledge presented in the previous chapters.

A concise, readable book, this organizes present information on bladder cancer, and suggests goals for the future. It is a worthwhile book for the student and deliverer of care to the patient with bladder cancer, and an adjunct book for the pathologist and radiologist.

Robert C. Thumann, M.D.

BOOKS RECEIVED

Orthopedic Diseases: Physiology, Pathology, Radiology. Fourth edition. By Ernest Aegerter, M.D., Emeritus Professor of Pathology, Temple University Health Sciences Center, Philadelphia, Pa.; and John A. Kirkpatrick, Jr., M.D., Radiologist-in-Chief, The Children's Hospital Medical Center; Professor Radiology, Harvard Medical School, Boston, Mass. Cloth. Pp. 792, with many illustrations. Price, $30.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1975.

Basic Radiation Biology. Second edition. By Donald J. Pizzarello, Ph.D., Professor in Radiology, New York University Medical Center, New York, N. Y.; and Richard L. Witcofski, Ph.D., Professor in Radiology, Associate in Neurology, The Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, N. C. Cloth. Pp. 143, with 42 illustrations. Price, $11.75. Lea & Febiger, Washington Square, Philadelphia, Pa. 19106, 1975.

Physicians' Desk Reference for Radiology and Nuclear Medicine. Editorial Consultants: Leonard M. Freeman, M.D., Associate Professor of Radiology, Albert Einstein College of Medicine; and M. Donald Blaufox, M.D., Ph.D., Associate Professor of Radiology and Medicine, Albert Einstein College of Medicine. Cloth. Pp. 308. Price, $9.50. Medical Economics Company, Oradell, N. J. 07649, 1975.

Coronary Arterial Variations in the Normal Heart and in Congenital Heart Disease. By Zeev Vlodaver, M.D., Senior Research Associate in Cardiovascular Pathology, Miller Division—United Hospitals, St. Paul and Research Associate, Graduate School, University of Minnesota, Minneapolis, Minn.; Henry N. Neufeld, M.D., Chief, Heart Institute, Chaim Sheba Medical Center, Tel-Hashomer; and Professor of Medicine and Chaim Sheba Professor of Cardiology, Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel; and Jesse E. Edwards, M.D., Director of Laboratories, Miller Division—United Hospitals, St. Paul; and Professor of Pathology, Graduate School, University of Minnesota, Minneapolis, Minn. Cloth. Pp. 171, with many illustrations. Price, $12.50. Academic Press, Inc., Publishers, 111 Fifth Avenue, New York, N. Y. 10003, 1975.

Radiobiology Examination Review Book: 900 Multiple Choice Questions and Referenced Answers. By Thongbliew Prempree, M.D., Ph.D., Associate Professor of Radiology, Division of Radiotherapy, University of Maryland Hospital, Baltimore, M.D.; Anas M. El-Mahdi, M.D., Sc.D., Professor of Radiology, University of Virginia School of Medicine, Charlottesville, Va.; James Shaeffer, Ph.D., Associate Professor of Radiology, University of Virginia School of Medicine, Charlottesville, Va.; and S. C. Barranco, Ph.D., Associate Professor of Biology, Division of Cell Biology, Department of Human Biological Chemistry and Genetics, University of Texas Medical Branch, Galveston, Texas. Paper. Pp. 128, with some figures. Price, $7.50. Medical Examination Publishing Co., Inc., 65–36 Fresh Meadow Lane, Flushing, N. Y. 11365, 1975.

Third National Cancer Survey: Incidence Data. Prepared by: Biometry Branch, Division of Cancer Cause and Prevention, National Cancer Institute. Edited by: Sidney J. Cutler, Sc.D., and John L. Young, Jr., Dr. P. H. National Cancer Institute Monograph 41, March, 1975. DHEW Publication No. (NIH 75–787). U. S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, Bethesda, Md. Cloth. Pp. 454. Price, $10.45. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402, 1975.

The Radiologic Clinics of North America. April, 1975, Volume XIII, Number 1. Symposium on Orthopedic Radiology. Guest Editor, Tom W. Staple, M.D. Cloth. Pp. 166, with many illustrations. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1975.

THE RADIOLOGIC CLINICS OF NORTH AMERICA. August, 1975, Volume XIII, Number 2. Symposium on Neonatal Radiology. Guest Editor, Bernard J. Reilly, M.D., F.R.C.P.(C). Cloth. Pp. 220, with many illustrations. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1975.

TRAINING OF MEDICAL LABORATORY TECHNICIANS: A HANDBOOK FOR TUTORS. By Alex McMinn, Head of External Relations, Liverpool Polytechnic, England; Formerly, Senior Course Tutor in Medical Science; and Graham J. Russell, Vice-Principal, Central Liverpool College of Further Education, England; Formerly, Head of Teacher Education Unit. Paper. Pp. 83. Price, Sw.fr. 15.—. World Health Organization, Geneva. (WHO Offset Publication No. 21: ISBN 92 4 170021 1.) Q Corporation, 49 Sheridan Avenue, Albany, N. Y. 12210, 1975.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. James Franklin Martin, 300 S. Hawthorne Rd., Winston-Salem, N. C. 27103. Annual Meeting: Washington Hilton, Washington, D. C., Sept. 20–24, 1976.

American Radium Society
Secretary, Richard H. Jesse, Dept. of Surgery, M. D. Anderson Hosp. and Tumor Institute, Houston, Tex. 77025. Annual Meeting: Bayshore Inn, Vancouver, B. C., Canada, May 9–14, 1976.

Radiological Societyof North America
Secretary, Dr. Theodore A. Tristan, Harrisburg, Pennsylvania. Annual Meeting: McCormick Place on the Lake, Chicago, Ill., Nov. 30–Dec. 5, 1975.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting.

Section on Radiology, American Medical Association
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.
Oral examinations will be held: Statler Hilton, Dallas, Texas, Dec. 8–13, 1975; Marriott Motor Hotel, Chicago, Ill., June 14–19, 1976; Sheraton Biltmore, Atlanta, Ga., Dec. 6–11, 1976.
Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.
Deadline for filing applications for any examination in 1976 is September 30, 1975.

American Board of Nuclear Medicine, Inc. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.
Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017.

American Association of Physicists in Medicine
Secretary, Dr. Robert G. Waggener, Dept. Radiol., Univ. Tex. Health Sciences Center, San Antonio, Tex. 78284
Annual Meeting:

American Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wis., 54601. Annual Meeting:

American Nuclear Society
Program Chairman, Werner K. Kern, IRD, Space Nuclear Systems, F 309, U. S. Atomic Energy Commission, Washington, D.C. 20545.

American Institute of Ultrasound in Medicine
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

American Society of Neuroradiology
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

Fourteenth International Congress of Radiology
Rio de Janeiro, Brazil, Oct. 23–29, 1977.
President Elect: Prof. Dr. Nicola C. Caminha; *General Secretary:* Dr. Durval Couto. Av. Churchill, 97-50 and-LC-39 20,000 Rio de Janeiro, Brazil.

International Society of Radiology
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

Twelfth Inter-American Congress of Radiology
President, Dr. Publio Vargas Pazzos, Casilla 5227, Guayaquil, Ecuador. Meeting in Quito, Ecuador, 1979.

Inter-American College of Radiology
President, Manuel Viamonte, Jr., M.D., Mt. Sinai Medical Center, 4300 Alton Rd., Miami Beach, Fla. 33140.
Secretary, Gaston Morillo, M.D., Jackson Memorial Hosp., 1700 N.W. 10th Ave., Miami, Fla. 33152.
Counselor for the United States, J. A. del Regato, M.D., Univ. of So. Fla. College of Medicine, 13000 No. 30th St., Tampa, Fla. 33612.

Third Congress of the European Association of Radiology
Secretary-General, P.O. Box 14, 79 Lauriston Place, Edinburgh BHI IDB, Scotland.

Second Asian and Oceanian Congress of Radiology
Secretary, Dr. Buenaventura U. V. Angtuaco, Congress Secretariat Headquarters, 2161 Taft Ave., Manila, Philippines. Meeting: Manila, Philippines, Nov. 10–14, 1975.

Alabama Chapter of ACR
Secretary-Treasurer, Dr. Lawrence E. Fetterman, 1720 Springhill Ave., Suite 201, Mobile, Ala. 36604. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society, Chapter ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

American Osteopathic College of Radiology
Annual Scientific Meeting: Americana Hotel, Bar Harbor, Fla. Program Administrator: Lawrence J. Jordan, D.O., Suite 21, Ponce American Bldg., 4601 Ponce de Leon Blvd., Coral Gables, Fla. 33146.

American Thermographic Society
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Irwin M. Freundlich, 1501 N. Campbell Ave., Dept. Radiol., Tucson, 85724. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-Tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. Richard Seibold, Jr., Wadley Hosp., Texarkana, Ark.—Tex. 75501. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Arch W. Templeton, Univ. of Kansas Med. Center, 39th and Rainbow Blvd., Kansas City, Kansas 66103. Annual Meeting.

Atlanta Radiological Society
Secretary-Treasurer, Dr. James H. Larose, South Fulton Hosp., 1170 Cleveland Ave., East Point, Ga. 30344. Meets 4 times during the academic year at an Atlanta Restaurant.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Bavarian-American Radiologic Society
Secretary, LTC. Marco J. DiBiase, M.D., Radiology Service, Heidelberg, 130 Station Hosp., APO N. Y. 09102. Meets quarterly.

Blockley Radiological Society
Secretary-Treasurer, Dr. William H. Green, 616 East 24th St., Chester, Pa. 19013.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Ralph Brancaccio, 7901 4th Ave., Brooklyn, N. Y. 11209. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. George J. Alker, Jr., E. J. Meyer Mem. Hosp., Buffalo, N. Y. 14215. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. Duane J. Gillum, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Alfred Brettner, Auburn Mem. Hosp., Auburn, N. Y. 13021. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Daniel E. Lewis, 309 East State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, March, and May at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer Dr. E. Jerome Schoolar, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harold J. Lasky, 55 E. Washington St., Suite 1735, Chicago, Ill. 60602. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Charles M. Greenwald, 7007 Powers Blvd., X-Ray Dept., Parma, Ohio 44129. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. John Pettigrew, Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Robert R. Burns, 1400 Stemmons Ave., Dallas, Tex. 75208. Meets the 3rd Monday of every month at 6:30 P.M., at the Airport Hotel, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Ekkehard S. Schubert, Wilmington Med. Center, P. O. Box 1951, Wilmington, Del. 19899.

East Bay Radiological Society
Secretary-Treasurer, Dr. Richard H. Culhane, 4 Vista del Mar, Orinda, Calif. 74563. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary. Dr. Paul J. Popovich, 1350 S. Hickory St., Melbourne, Fla. 32901. Meets twice annually, in the spring with the Annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Herbert Johnson, Davis Blvd., Tampa, Fla. 33606. Meets on 4th Thursday of the months January, March, May, October, and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Jay I. Rozen, Suite 216, 6400 Prospect, Kansas City, Mo. 64132. Meets 5 times a year.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary, Dr. David C. Hillman, P. O. Box 610544, North Miami, Fla. 33161. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Michael J. McCabe, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:00 P.M.

Health Physics Society
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Robert D. Dooley, Hinsdale Med. Center, Hinsdale, Ill. 60521. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John A. Knote, Home Hosp., Lafayette, Ind. 47902.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Dale L. Roberson, 1948 First Ave., NE, Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific session is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph In-

firmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Harry L. Stein, North Shore Univ. Hosp., 300 Community Dr., Manhasset, N. Y. 11030.

Los Angeles Radiological Society
Secretary, Dr. Arthur F. Schanche, 333 North Prairie Ave., Inglewood, Calif. 90301. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference. Century Plaza Hotel, Los Angeles, Calif.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. J. Gardiner Bourque, 3155 Stagg Dr., Suite 230, Doctors Bldg., Beaumont, Texas 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Peter E. Giustra, Dept. Radiology, Penobscot Bay Medical Center, Rockland, Maine 04841. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Joseph T. Ferrucci, Jr., Massachusetts Gen. Hosp., Boston, Mass. 02114.

Memphis Roentgen Society
Secretary-Treasurer, Dr. R. Randolph Ramey, Baptist Memorial Hosp., 899 Madison Ave., Memphis, Tenn. 38146. Meets first Monday Oct. through June at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Francis P. Shea, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin E. Goldberg, Box 292, Mayo Memorial Health Sciences Center, Minneapolis, Minn. 55455.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Robert Bodmer, Suite 622 Doctors Bdg., Omaha, Neb. 68131. Meets the third Wednesday of the month, Oct. through March, at the Clarkson Hosp., Omaha, Neb. and the fourth Friday of April at the Lincoln Country Club, Lincoln, Neb.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

N.E. Society for Radiation Oncology
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

New England Roentgen Ray Society
Secretary, Dr. Melvin E. Clouse, 185 Pilgrim Rd., Boston, Mass. 02115. Meets the third Friday of Oct. and Nov., and third Thurs. of each month. Jan. through May, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass. at 3:00 P.M. Annual Meeting: May 20, 1976.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. W. M. Jordan, 1100 Central Ave. S.E., Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference.

New York Roentgen Society
Secretary-Treasurer, Dr. Thomas C. Beneventano, 111 East 210th St., Bronx, N. Y. 10467. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., April 22–24, 1976. A.M.A. Cat. I credit. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 6 Secor Dr., Port Wash., N. Y. 11050.

North American Society of Cardiac Radiology
Secretary-Treasurer, Dr. Erik Carlson, Univ. Calif., San Francisco, Calif. 94143.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, P. O. Box 6001, Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

NORTHEASTERN OKLAHOMA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Donald R. Morton, Dept. of Radiology, St. Clare's Hosp., Schenectady, N. Y. 12304. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. John D. Earle, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. Patrick J. Grinsell, 1207 Fairchild Ct., Woodland, Calif. 95695. Meets on the fourth Monday, Sept. through May at the Mansion Inn, Sacramento, Calif.

NORTHWESTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

NORTHWESTERN OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Richard W. Siders, 421 Michigan, Toledo, Ohio 43624. Meets 3rd Wednesday from September to May.

OHIO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. James Farmer, Cleveland, Ohio.

OKLAHOMA STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

ORANGE COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. William E. Danko, 2601 E. Chapman, Orange, Calif. 92667. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

OREGON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ray F. Friedman, 3324 S. W. 44th St., Portland, Ore. 97221. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Kenneth D. Moores, 1118 9th Ave., Seattle, Wash. 98101. Annual Meeting: Benson Hotel, Portland, Ore., May 7–9, 1976.

PENNSYLVANIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Joseph A. Marasco, Jr., St. Francis General Hosp., Pittsburgh, Pa. 19141.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. Marvin E. Haskin, 230 N. Broad St., Philadelphia, Pa. 19102. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. Klaus M. Bron, Presbyterian-Univ. Hosp., Pittsburgh 15213. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

RADIATION RESEARCH SOCIETY
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting.

RADIOLOGICAL SOCIETY OF CONNECTICUT, INC., CHAPTER OF ACR
Secretary, Dr. Gerald L. Baker, 85 Jefferson St., Hartford, Conn. 06106. Meets in Oct., Jan., March, and June.

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

RADIOLOGICAL SOCIETY OF NEW JERSEY, CHAPTER OF ACR
Secretary, Dr. Fred M. Palace, 11 Dale Dr., Morristown, N.J. 07960. Monthly meetings in East Orange and Trenton, N.J. Semi-Annual Meeting in Nov.

RADIOLOGICAL SOCIETY OF RHODE ISLAND, CHAPTER OF ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA
Secretary-Treasurer, Dr. A. Franklin Turner, LAC-USC Medical Center, Room 3418, 1200 North State St., Los Angeles, Calif. 90033. Meets three times a year, usually October, February and May.

RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

REDWOOD EMPIRE RADIOLOGICAL SOCIETY
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

RICHMOND COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Frederick S. Vines, 11830 Rothbury Dr., Richmond, Va. 23235. Meets third Thursday of each month at the Aberdeen Barn.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, Rochester General Hosp., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual Meeting:

SAN ANTONIO-CIVILIAN MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

SAN DIEGO RADIOLOGICAL SOCIETY
President, Dr. Donald J. Fleischli, 7849 Fay Ave., La Jolla, Calif. 92037. Meets the first Wednesday of each month Sept. through June at the University Club.

SAN FRANCISCO RADIOLOGICAL SOCIETY
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SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION OF RADIOLOGY, NATIONAL MEDICAL ASSOCIATION
Secretary, Dr. Ruby Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broadway, Baltimore, Md. 21205. Annual meeting: Marriott Hotel, Atlanta, Ga., Sept. 28–29, 1975.

Society of Gastro-intestinal Radiologists
Secretary-Treasurer, Dr. John R. Amberg, University Hospital, 225 W. Dickinson St., San Diego, Calif. 92103.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual Meeting.

South Bay Radiological Society
Secretary, Dr. Robert Mindalzun, Valley Radiologists, Inc., 100 O'Connor Dr., San Jose, Calif. 95128. Meets the 2nd Monday of each month at the Santa Clara County Medical Society Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society Chapter of ACR
Secretary-Treasurer, Dr. Richard T. Love, Santa Barbara Med. Clin., 215 Pesetas Lane, Santa Barbara, Calif. 93110. Meets fourth Tuesday of alternate months, Sept., Nov., Jan., March and May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Association
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. J. W. Maxwell, P.O. Box 2144 Mobile Ala. 36601. Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 30–Feb. 1, 1976.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Jerry W. Grise, Methodist Hosp., 1265 Union Ave., Memphis, Tenn. 38104. Meets annually at the time and place of the Tennessee State Medical Association meeting.

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Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bdg., San Antonio, Tex. 78205.

The Fleischner Society
Secretary, Dr. E. Robert Heitzman, State University of New York Upstate Medical Center, Syracuse, N. Y. 13210. Annual Meeting:

Tri-State Radiological Society
Secretary, Dr. Thomas Harmon, St. Mary's Hosp., Evansville, Ind. 47750. Meets bimonthly on the third Wednesday at area Hospitals.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Patrick D. Lester, Dept. Radiol., University of Utah Medical Center, Salt Lake City, Utah 84112. Meets quarterly at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 3500 Kensington Ave., Suite 2-A, Richmond, Va. 23221.

Washington, D. C., Chapter of ACR
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Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Donald J. Hesch, 3216 N.E. 45th Place, Seattle, Wash. 98105.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Leonard Cutler, 16 Guion Place, New Rochelle, N. Y. 10802. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. June Unger, Wood VA Hosp., Wood, Wis. 53193. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wis. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
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Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de Salvador
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Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9², Calle A 0–05, Zona 1, Guatemala

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Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. César Rosa, Box 9387, Santurce, Puerto Rico 00908. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St. Catherine St. West, Montreal 107, Que., Canada. Annual Meeting:

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, P. D. Thomson, 28 Portland Pl., London, W1N 4DE.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie
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The Hospital Physicists' Association
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Toronto Radiological Society
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The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
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Federación Argentina de Sociedades de Radiología
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Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

Sociedad Argentina de Radiología
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Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 p.m.

Sociedad Colombiana de Radiología
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Sociedad Ecuatoriana de Radiología
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Sociedad Paraguaya de Radiología
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Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
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Sociedad Salteña de Radiología y Medicina Nuclear
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Sociedad Venezolana de Radiología y Medicina Nuclear
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Continental Europe

Österreichische Röntgen-Gesellschaft
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Société Royale Belge de Radiologie
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Société Européenne de Radiologie Pédiatrique
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Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 730, Paris Cadet 15, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger.
Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
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Československá Radiologická Společnost
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Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
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Societas Radiologica Danica
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Nederlandse Vereniging voor Radiologie
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Scandinavian Radiological Society
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Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
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Svensk Förening för Medicinsk Radiologi
Secretary, Dr. Hanz Ringertz, Röntgendiagnostika Avdelningen, Karolinska Sjukhuset 104 01 Stockholm 60, Sweden.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Bengal Radiological Association
Honorary Secretary, Dr. B. Chatterji, 262 Rash Behari Ave., Calcutta 700019, India. Meets 1st Fri. and 3rd Wed. of every month.

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Majid Rooholamini, P.O. Box No. 14–1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
Secretary, Dr. M. A. Lerner, Hesharon Hosp., Petah-Tigva, Israel.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
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Radiological Society of South Africa
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Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

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(Act of August 12, 1970: Section 3685, Title 39, United States Code)

1. TITLE OF PUBLICATION: THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE	2. DATE OF FILING
3. FREQUENCY OF ISSUE: MONTHLY	3A. ANNUAL SUBSCRIPTION PRICE: $30.00

4. LOCATION OF KNOWN OFFICE OF PUBLICATION *(Street, city, county, state and ZIP code) (Not printers)*
301-327 East Lawrence Avenue, Springfield, Illinois 62717

5. LOCATION OF THE HEADQUARTERS OR GENERAL BUSINESS OFFICES OF THE PUBLISHERS *(Not printers)*
CHARLES C THOMAS, PUBLISHER, 301-327 East Lawrence Ave., Springfield, IL 62717

6. NAMES AND ADDRESSES OF PUBLISHER, EDITOR, AND MANAGING EDITOR

PUBLISHER *(Name and address)*
CHARLES C THOMAS, PUBLISHER, 301-327 East Lawrence Ave., Springfield, IL 62717

EDITOR *(Name and address)*
E. FREDERICK LANG, M.D., ACTING EDITOR, 401 Professional Building, 10 Peterboro, Detroit, MI 48201

MANAGING EDITOR *(Name and address)*
NONE

7. OWNER *(If owned by a corporation, its name and address must be stated and also immediately thereunder the names and addresses of stockholders owning or holding 1 percent or more of total amount of stock. If not owned by a corporation, the names and addresses of the individual owners must be given. If owned by a partnership or other unincorporated firm, its name and address, as well as that of each individual must be given.)*

NAME	ADDRESS
The American Roentgen Ray Society, A non-profit organization incorporated in the District of Columbia	300 S. Hawthorne Rd., Winston-Salem, NC 27103
Dr. James E. Martin, Secretary	Dr. Paul A. Riemenschneider, Chairman Publication Committee, Cottage Hosp., Santa Barbara, CA 93105

8. KNOWN BONDHOLDERS, MORTGAGEES, AND OTHER SECURITY HOLDERS OWNING OR HOLDING 1 PERCENT OR MORE OF TOTAL AMOUNT OF BONDS, MORTGAGES OR OTHER SECURITIES *(If there are none, so state)*

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A. TOTAL NO. COPIES PRINTED *(Net Press Run)*	14,965	15,165
B. PAID CIRCULATION 1. SALES THROUGH DEALERS AND CARRIERS, STREET VENDORS AND COUNTER SALES	—	—
2. MAIL SUBSCRIPTIONS	13,344	14,138
C. TOTAL PAID CIRCULATION	13,344	14,138
D. FREE DISTRIBUTION BY MAIL, CARRIER OR OTHER MEANS SAMPLES, COMPLIMENTARY, AND OTHER FREE COPIES	221	190
E. TOTAL DISTRIBUTION *(Sum of C and D)*	14,065	14,328
F. COPIES NOT DISTRIBUTED 1. OFFICE USE, LEFT OVER, UNACCOUNTED, SPOILED AFTER PRINTING	900	837
2. RETURNS FROM NEWS AGENTS	—	—
G. TOTAL *(Sum of E & F—should equal net press run shown in A)*	14,965	15,165

I certify that the statements made by me above are correct and complete.

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Paul A. Riemenschneider

PS Form 3526 (Page 1)
Jan. 1975

ABSTRACTS OF RADIOLOGIC LITERATURE

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ROENTGEN DIAGNOSIS

Head

Poisson, M., Buge, A., Pouillart, P., Mathé, G., Huguenin, P., Morin, P., Gautier, H., and Parrot, R. Traitement des glioblastomes inopérables par une combinaison d'adriamycine, de VM 26 et de CCNU. (Treatment of inoperable glioblastomas using a combination of adramycin, VM 26 and CCNU. *Presse méd.*, July, 1975, *4*, 1957–1961. (Address: M. Poisson, Groupe hospitalier Pitié-Salpêtrière, 83, boulevard de l'Hôpital, F. 75634 Paris, France.)

Since the first use of chemotherapy for solid tumors, it has been demonstrated that certain nitroso-urea derivatives, in particular 1, (2-chloroethyl)-3-cyclohexyl-1-nitrosourea (CCNU), have a marked effectiveness as far as the progression of primary malignant cerebral tumors is concerned. More recently, animal experiments have shown that the effects of CCNU may be potentiated by 4-dimethylepipedophylotoxine beta-D-thenylideneglucoside (VM 26) and adriamycin.

These three drugs were used sequentially in the treatment of 18 patients with inoperable glioblastoma. Tolerance to the drugs is quite satisfactory, and toxicity is limited to the bone marrow, with no change in liver or kidney function. The only important effect is diminution in the number of platelets during the first three weeks of each cycle of treatment. Occasionally the thrombocytopenia required delaying the beginning of the next cycle of treatment. In only 2 of 11 cases did the level fall as far as 40,000 per cubic mellimeter.

Objective evidence of improvement was obtained in 12 of the 18 patients. Five returned to their previous life habits and style. One of these, a distinguished physician who was initially aphasic, returned to his practice successfully. The other seven were able to take care of their basic needs by themselves, and could thus return to their families. The improvement varied from a few months to more than ten months in the most favorable cases.

These objective results in both duration and quality of survival are encouraging and point to new progress in the chemotherapy of glioblastomas.—*Augmented published English abstract*

Prather, Jerry L., Long, John M., van Heertum, Ronald and Hardman, John. Multicentric and isolated multifocal glioblastoma multiforme simulating metastatic disease. *Brit. J. Radiol.*, Jan., 1975, *48*, 10–15. (Address: Jerry L. Prather, M.D., William Beaumont Army Medical Center, El Paso, Texas.)

Roentgenographic differentiation of solitary vascular cerebral metastases from glioblastoma multiforme may be impossible. However the demonstration of multiple vascular cerebral tumors by arteriography and 99^{m} Tc pertechnetate brain scanning usually denotes the presence of metastatic lesions or rarities such as hemangioblastoma, multiple meningiomas, and multiple vascular neuromas of von Recklinghausen's disease.

The frequency of occurrence of multifocal (*i.e.* tumors with gross or microscopic continuity, with or without cerebral fluid spread) or multicentric glioblastoma multiforme is unknown, but is not common.

Two cases were presented. In the first patient, autopsy demonstrated two separate foci of glioblastoma multiforme, which had been demonstrated by arteriography and brain scanning. The second patient harbored four isolated multifocal tumors, two of which had been shown ante mortem.

The authors suggest that multicentric or multifocal glioblastoma multiforme should be added to the differential diagnosis of multiple expanding vascular lesions demonstrated angiographically and by repeated brain scanning, particularly if an extracranial primary malignancy has been excluded.—*Adele R. Altman, M.D.*

Neck and Chest

Rose, Judith S., Kassner, E. G., Jurgens, K. H., and Farman, J. Congenital oesophageal strictures due to cartilaginous rings. *Brit. J. Radiol.*, Jan., 1975, *48*, 16–18. (From: The Departments of Radiology and Pathology, Downstate Medical Center, Brooklyn, N. Y.)

Embryonic tracheobronchial remnants, containing cartilage, mucus glands, and respiratory epithelium, may sequestrate into the esophagus, causing non-neoplastic strictures of the distal esophagus. Presumably, this anomaly represents incomplete separation of the respiratory tract from the primitive esophagus. Dysphagia, vomiting, and aspiration pneumonia usually herald the diagnosis in the neonatal period. However, if symptoms are less severe, detection of the lesion may be delayed into adulthood.

Two patients were reported: one seven months, the other eight years old. Barium esophagrograms showed dilatation of the esophagus proximal to persistently narrowed distal segments, respectively 2 cm and 4 cm in length. The second case demonstrated a globular collection of barium projecting medially from the mid-point of the stricture, resembling a penetrating ulcer. Examination of the resected specimen proved that the 0.5 cm barium filled tract represented dilated ducts communicating with the esophagus.

Differential diagnosis includes stenosis due to ingestion of caustic substances, peptic esophagitis, and, less commonly, achalasia.—*Adele R. Altman, M.D.*

Genitourinary System

Moore, William B., Matthews, Terence J., and Rabinowitz, Ronald. Genitourinary anomalies associated with Klippel-Feil syndrome. *J. Bone & Joint Surg.*, April, 1975, *57*-A, 355–357. (Address: William B. Moore, M.D., 416 Pinehurst Avenue, Salisbury, Md. 21801.)

The authors studied 39 patients with Klippel-Feil (KF) syndrome. The average age at diagnosis of KF was 2.9 years. There was a female-male ratio of 1.8/1.0. The group included 21 Type I, which have block fusion of all cervical and upper thoracic vertebrae; four Type II with fusion of one or two pairs of cervical vertebrae; and 14 Type III where the anomalies of Type I or II are combined with either lower thoracic or lumbar vertebral fusion.

Of the 39 patients, 23 had significant upper urinary tract anomalies but only one of them had symptoms suggesting a renal anomaly. This patient had a Type III KF and developed pain and anuria. She was found to have unilateral renal agenesis with an obstructing calculus. One patient had vaginal agenesis and a bicornuate uterus, one had cryptorchidism and one had hypospadias. The total of genitourinary anomalies was 64 percent.

Unilateral renal agenesis or complete absence of a kidney was the most common abnormality and was present in five Type I, one Type II and five Type III KF syndrome patients. Malrotation, duplication, ectopia, and renal dysgenesis with subnormal growth and function were also found in a higher incidence than in the general population. A Table is included giving the incidence rates in the present study and in the normal population. The genitourinary anomalies were present in 62 percent of Type I patients, 50 percent of Type II and 71 percent of Type III.

The authors briefly discuss the embryological development of the axial skeleton and the genitourinary tract and the explanation for the association of these anomalies. An insult to the fetus between the fourth and eighth weeks of development would produce both genitourinary and KF syndrome. A routine intravenous urogram is therefore suggested in all patients with KF syndrome.—*Martha Mottram, M.D.*

Skeletal System

Himmelfarb, Elliot, Sebes, Jeno, and Rabinowitz, Jack. Unusual roentgenographic presentations of multiple myeloma: report of three cases. *J. Bone & Joint Surg.*, Dec., 1974, *56*-A, 1723–1728. (Address: Elliott Himmelfarb, M.D., 865 Jefferson Avenue, Memphis, Tenn. 38163.)

Three cases of unusual roentgenographic manifestations of untreated multiple myeloma have been reported.

In the first case there was diffuse osteosclerosis of the spine, pelvis, and extremities which simulated extensive osteoblastic metastasis. Microscopic examination of sections of the spine at autopsy 11 months later showed marked thickening of bone trabeculae adjacent to primitive plasma cells.

In a review of the literature the authors found reports of about 100 cases of sclerotic myeloma.

In the second case there were bone lesions of coexisting myeloma and amyloidosis. Frank myeloma was found in an extradural mass adjacent to the collapsed body of the 9th dorsal vertebra. Amyloidomas were found at biopsy of several of the numerous expansile lytic lesions in the extremities. Some of the peripheral lesions showed the following changes: marginal sclerosis, subchondral erosion, cortical bone destruction, and sequestrum-like lytic areas.

The coexistence of amyloidosis with myeloma is significant since it will not respond to the usual alkylating agents nor to radiation therapy, but will react to chloroquine and methotrexate.

The third case showed an extensive solitary destructive myeloma in the proximal right femur with a pathologic fracture. The medullary canal was widened and there were areas of bone sequestration. Prominent periosteal reaction and soft-tissue extension were present. A diagnosis of myeloma was made from a biopsy specimen and confirmed on subsequent tissue study of the disarticulated extremity.

The differential diagnosis in this case included primary bone tumors and solitary metastasis. The unusual manifestations of myeloma were periosteal new-born formation of sclerotic foci.—*Paul A. Dassel, M.D.*

Brand, Richard A., and Black, Howard. Pseudomonas osteomyelitis following puncture wounds in children. *J. Bone & Joint Surg.*, Dec., 1974, *56*-A, 1637–1642. (Address: Richard A. Brand, M.D., Department of Orthopaedic Surgery, University of Iowa Hospitals, Iowa City, Iowa 52242.)

Pseudomonas aeuroginosa is an uncommon causative organism of osteomyelitis. Nine cases have been reported in children following puncture wounds of the feet.

In the typical case there was onset of pain and swelling two to five days following a puncture wound. The primary treatment often consisted of local cleansing, soaks, tetanus toxoid, and occasionally antibiotics. There usually was partial resolution of the inflammatory process, but symptoms and signs of infection either returned or did not completely subside.

Osseous changes were first detected on roentgenograms made between the 8th and 21st day after the injury. The following bone and joint changes have been reported in Pseudomonas osteomyelitis: joint-space narrowing, cyst formation, osteophytosis,

joint rigidity, and premature closure of the epiphyseal plate.

All infections in these cases responded well to surgery and antibiotics; however, in three patients there was permanent but minor joint impairment.

Carbencillin and gentamicin are the antibiotics of choice in Pseudomonas osteomyelitis.—*Paul M. Dassel, M.D.*

Bruce, Henry E., Harvey, J. Paul, Jr., and Wilson, John C., Jr. Monteggia fractures. *J. Bone & Joint Surg.*, Dec., 1974, *56*-A, 1563–1576. (Address: Henry E. Bruce, M.D., 1352 Ballena Boulevard, Alameda, Calif. 94501).

Complications from Monteggia fractures were studied in 35 patients evaluated one year or more after injury.

A Monteggia fracture is an uncommon injury complex consisting of a fracture of the proximal third of the ulnar shaft and dislocation of the radial head. The proximal radio-ulnar joint is disrupted or dislocated. There are four subclassifications of this fracture.

These fractures were rather evenly distributed during the first five decades of life.

The most common causes of this fracture were motor-vehicle and falling injuries.

The results of these fractures were rated according to range of motion, activities of daily living or work, pain and anatomy. The authors evaluated the end results of these cases as excellent in eight, good in six, fair in eight and poor in (13). The major factor which led to poor results in adults was limitation of motion. Other contributing factors were pain and decreased function.

There were nerve palsies either at the time of injury or after treatment in eleven patients. The nerve injury was transient in all but one patient.

Non-union occurred in eitht patients. Synostosis developed in two patients.

The authors stress the importance of prompt recognition and treatment of the dislocation of the radial head in this injury.—*Paul M. Dassel, M.D.*

Kamhi, Edouard, and MacEwen, G. Dean. Osteochondritis dissecans in Legg-Calvé-Perthes disease. *J. Bone & Joint Surg.*, June, 1975, *57*-A, 506–509. (Address: Edouard Kamhi, M.D., 325 Park Avenue, Nuntington, N. Y. 11743.)

Osteochondritis dissecans has been reported infrequently as a complication of Legg-Calvé-Perthes disease (LCP). However, it was found in seven male patients out of the first 200 with LCP seen at the duPont Institute, Delaware. Involvement was unilateral in six and bilateral in one. Although 20 percent of the patients with LCP were female, no females developed osteochondritis dissecans.

The seven cases are briefly reported and there are a number of diagrams. Roentgenograms of one patient show a loose fragment 20 years after LCP was treated. A large cartilaginous fragment was outlined by a spontaneous air arthrogram in another patient who had only mild symptoms and no locking nor catching. The patients had been treated with various methods and two had no treatment; there was no evident difference in the type or duration of treatment of LCP as to whether or not they developed a later osteochondritis dissecans. The older age group had a greater risk of developing this complication.

The interval between the end of treatment and the diagnosis of osteochondritis dissecans ranged from one to 20 years; in three patients, it was diagnosed during therapy for LCP. Perhaps an unhealed necrotic fragment persists into adult life without symptoms or with very minor complaints. Separation of the fragment from its bed was found to be uncommon. Spontaneous healing of osteochondritis dissecans after LCP has not been reported.

Diagnosis may be difficult on routine films. A persistent indentation or irregularity of the head should be suspect, and the authors urge tomograms in the frog-leg position for optimum visualization. Treatment is preferably conservative.—*Martha Mottram, M.D.*

Reckles, Lawrence N., Peterson, Hamlet A., Bianco, Anthony J., Jr., and Weidman, William H. The association of scoliosis and congenital heart defects. *J. Bone & Joint Surg.*, June, 1975, *57*-A, 449–455. (Address: Lawrence N. Reckles, M.D., c/o Section of Publications, 200 First Street, S. W., Rochester, Minn. 55901.)

Scoliosis is more frequent in patients with congenital cardiac defects than in the general population. In the Mayo Scoliosis Clinic, 5 percent of the patients have significant congenital cardiac lesions. Treatment of the scoliosis is often difficult in these patients. The authors quote statistics from various centers on the incidence of scoliosis and congenital heart defects, and although there are expected differences depending on method of survey, age at time of surgery and degree of curvature, there is an increased incidence of scoliosis in congenital heart disease in all reported series. Out of 998 patients under 16 having cardiac surgery at Mayo Clinic between 1950–1959, 377 were checked with standing roentgenograms of the spine ten years or more after surgery (average 15 years). The average age at follow-up was 21 years 4 months. In this group of 377 patients, there were 32 patients (8.5 percent) with curves greater than 20 degrees and these were studied and compared with the group without clinically significant scoliosis. Those with curves under 20 degrees were considered non-problem cases; they often have multiple small curves. The authors found that

the patterns of the scoliosis in patients with congenital heart disease and curves greater than 20 degrees were approximately the same as those in patients with idiopathic scoliosis.

There was no correlation between the development or the degree of the scoliosis with cardiac size or a specific cardiac defect. There was a higher percentage of patients with cyanosis in the scoliosis group, but this was not statistically significant. The age at surgery, type or site of incision, and the surgical procedure had no effect on the development of scoliosis. Only one patient had a true congenital scoliosis with hemivertebra. The number and side of removed ribs had no constant relationship to the development of scoliotic curves or patterns.

It was found that the incidence of scoliosis with curvature greater than 20 percent in patients who had surgery for congenital heart disease is more than ten times that found in the general population.—*Martha Mottram, M.D.*

Scatliff, James H., Till, Kenneth, and Hoare, Richard D. Incomplete, false, and true diastematomyelia: radiological evaluation by air myelography and tomography. *Radiology*, Aug., 1975, *116*, 349–354. (Address: James H. Scatliff, M.D., Department of Radiology, University of North Carolina School of Medicine, Chapel Hill, N. C. 27514.)

Six cases of possible diastematomyelia were studied by air myelography and tomography as well as initial plain roentgenogram studies. The paper deals with the roentgenographic evaluation of the cases and examines the pitfalls in the diagnostic procedures. Each of the surgically proved cases was thought roentgenographically to demonstrate either fibrous or bony septa splitting the spinal cord at the point of involvement. The surgical intervention in each of these cases showed no fibrous or bony septa. Varying abnormalities were found including posterior grooves in the cord, fibrous brands tethering the cord, unusual posterior redundancy or infolding of the arachnoid in the midline (septum posticum), and true hemicord without bony or fibrous septum.

The points to be learned from this correlative study are as follows: (1) Linear tomography can produce spurious lucent defects particularly when spina bifida or spinal dysraphism is present. (2) Fibrous septa cannot be defined with certainty on air myelography and tomography. (3) The presence of tethering bands or clusters of nerve roots cannot be resolved by air myelography. (4) Hemicord or double cords are not necessarily associated with a bony spur or fibrous septum. (5) Presence of tethering dural bands in the absence of dividing septa should be documented since surgical treatment is based upon the release of the tension on a fixed cord.

In summary, each case of spinal dysraphism and suspected diastematomyelia should be evaluated on an individual basis with close correlation of clinical and roentgenologic findings associated with the spinal canal.—*J. P. Eberts, M.D.*

RADIATION THERAPY

Turek-Maischeider, Milada, and Kazem, Ismail. Palliative irradiation for liver metastases. *J.A.M.A.*, May, 1975, *232*, 625–628. (Address: Dr. Ismail Kazem, Department of Radiation Therapy and Nuclear Medicine, Sint Radboud Hospital, Geert Grooteplein Zuid 18, Nijmegen, The Netherlands.)

Eleven patients with symptomatic liver metastases received 1,600 to 2,500 rads total dose administered to the liver in two to three weeks. All of these patients had abnormal liver scans and all complained of upper abdominal pain. Initially, four patients were jaundiced, nine were nauseated and four had recurrent vomiting. Nine of the eleven patients had abnormal liver function studies.

Eight patients were completely relieved of their symptoms with remissions ranging from two to 38 months. Two patients had partial relief of symptoms and one patient showed no improvement. Beside the relief of symptoms, some patients showed decrease in the size of the liver metastases as delineated in the radioisotope scans and some patients' liver function studies improved.

Autopsies were performed on three patients and histological evaluation of the liver showed no evidence of radiation induced injury.

The palliative treatment of hepatic metastases include systemic chemotherapy, intrahepatic arterial chemotherapeutic infusion, partial hepatectomy, and liver irradiation. The threshold dose for radiation hepatitis is approximately 3,000 rads.

The primary tumors in these patients include mammary carcinoma, reticulum cell carcinoma, Ewing's sarcoma, Wilms' tumor, colon adenocarcinoma and hemangioendotheliosarcoma. The results of radiation therapy suggest that local palliation can occur regardless of the type of primary tumor possibly on the basis of better vascularization in the liver with increased oxygenation, immune response of the liver on metastases potentiating the radiation effect, etc. The relief of clinical symptomatology appeared to be more rapid following irradiation than with other modalities of treatment of metastatic disease to the liver.—*Ronald J. Ross, M.D.*

Moritsch, E., and Neumann, H. Zur Behandlung des "beweglichen" Stimmbandkarzinoms: Chordektomie, Bestrahlung. (The treatment of the "mobile" vocal cord carcinoma: cordectomy versus irradiation.) *Wiener klin. Wochen.*, Feb., 1975, *87*, 116–120. (Address: Doz. Dr. E. Moritsch, I.

Hals-, Nasen- und Ohrenklinik der Universität Wien, Lazarettgasse 14, A-1097, Wien, Austria.)

In the treatment of Stage I carcinoma of the vocal cords the five year cure rates with cordectomy and with irradiation have each been described as about 80 percent. Between 1965 and 1969, 49 patients were cordectomized and 71 patients were subjected to irradiation. The three year operative cure rate was 85.7 percent and the five year cure rate was 75 percent. With irradiation the corresponding figures were 74.6 percent and 68.6 percent. Irradiation therapy was given in the earlier part of the period with conventional kilovoltage and in the latter part of the period with a cobalt source.

The two types of treatment, operation and irradiation, were compared to determine the advantages and disadvantages of each.

The following points are in favor of cordectomy: The tumor can be directly examined. There is immediate histological control during the operation. The period of treatment lasts about seven to ten days, as opposed to four to five weeks with irradiation. There is a rather short healing period with operation, while irradiation may entail chronic aftereffects. Perichondritis is not observed after operation but may follow irradiation. In the event of recurrence, after cordectomy either a full radiation dose may be administered or operation may be carried out in non-irradiated tissue; after irradiation, neither a full radiation dose may be given nor may the operation be carried out through unirradiated tissue.

There are points in favor of radiation therapy. Surgery may be contraindicated by medical considerations. A prophylactic tracheotomy is usually required after operation and is usually not necessary with irradiation. The quality of the voice is clearly better after irradiation than after operation and irradiation is therefore preferable in patients in whom preservation of voice is essential.

The problem should be thoroughly discussed with the patient.—*Modified Published English Abstract.*

BAEZA, MARIO R., BARKLEY, H. THOMAS, JR., and FERNANDEZ, CARLOS H. Total-lung irradiation in the treatment of pulmonary metastases. *Radiology*, July, 1975, *116*, 151–154. (Address: H. Thomas Barkley, Jr., M.D., Department of Radiotherapy, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025.)

Total lung irradiation has been employed for many years in the treatment of pulmonary metastases arising from a variety of primary malignant neoplasms. Despite its frequent use, there is very little objective evidence that irradiation materially alters the quality or length of survival. The chief obstacle to successful sterilization of metastases is lung parenchymal tolerance which is influenced by total dose, volume, fractionation, infection, and possibly certain chemotherapeutic agents. The authors reviewed the records of 62 patients given total lung irradiation at M. D. Anderson Hospital from January, 1956, through December, 1972, in order to assess the efficacy of such treatment in pulmonary metastases from a variety of primary malignant neoplasms.

Three were treated with irradiation alone while the remainder received adjunctive chemotherapy. The entire lungs were irradiated and the mediastinal structures were not shielded. The two most common treatment schedules were: (1) 1,500 rads/ten fractions/two weeks; and (2) 2,000 rads/fifteen fractions/three weeks.

The results showed that only patients with metastatic Wilms' tumor had a satisfactory response as evaluated by chest roentgenogram. Three-fourths of these patients had complete or nearly complete regression. The response of metastases from Ewing's sarcoma and rhabdomyosarcoma indicated only a 25 percent reduction in half of the patients while over 50 percent of patients with all other types of primary tumors had no response at all.

Radiation pneumonitis was diagnosed in 13 patients, nine of whom received concomitant chemotherapy. It was fatal in three as confirmed by autopsy; two of them had received chemotherapy.

Using graphic means and regression analysis, an attempt was made to formulate a time-dose relationship that would separate success, failure, and complications. This was nondeterminate.

It was concluded that doses in the range of 1,500–2,000 rads to the entire thorax were of value only in the treatment of patients who had metastases from Wilms' tumor and who also received adjunctive chemotherapy (actinomycin D). An exception might result from the development and adjuvant use of radioprotective agents which increase parenchymal tolerance, radiosensitizing agents which selectively sensitize the metastatic neoplasm, or effective chemotherapeutic agents which destroy additional magnitudes of tumor cells.—*P. M. Kroening, M.D.*

JAMPOLIS, SAMUEL, ANDRAS, E. JAMES, and FLETCHER, GILBERT H. Analysis of sites and causes of failures of irradiation in invasive squamous cell carcinoma of the intact uterine cervix. *Radiology*, June, 1975, *115*, 681–685. (Address: Gilbert H. Fletcher, M.D., Professor and Head, Department of Radiotherapy, The University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025.)

Records of 916 patients who received radiation therapy for Stage IB through Stage III invasive squamous cell carcinoma of the intact uterine cervix were analyzed for sites and causes of treatment failure. Staging by the M. D. Anderson method according to the volume of cancer was utilized. Micro-

invasive carcinomas were not included. Minimum follow-up for all patients was four and one-half years. Survival rates were as follows: Stage I, 91 percent; Stage II_A, 82 percent; Stage II_B, 65 percent; Stage III_A, 54 percent; and Stage III_B, 40 percent.

Two important developments occurred during the period of analysis and contributed to the over-all results—use of computer dosimetry for intracavitary radium and the use of conservative extrafascial hysterectomy for bulky central disease in Stages I_B and II following a slightly less radical dose of irradiation. Ninety-four percent of central and regional failures occurred within the first three years after treatment.

In Stages I_B and II_A (494 patients) eleven patients (two percent) had central failures. Eight of the eleven could be directly attributed to improper placement of ovoids or the use of a vaginal cylinder in place of ovoids because of poor geometry of the vaginal vault. Central failures also occurred in large bulky central lesions. There were regional failures in 23 (4.5 percent), mainly due to skewed radium systems without appropriate compensation with external beam. The use of a vaginal cylinder in place of ovoids was another cause.

Stage II_B (204 patients) had ten central failures (five percent), and all occurred in conjunction with regional failure. Six cases were due to massive central disease and three resulted from less than optimum radiation dosage. There were 27 regional failures (13 percent) and 17 could be correlated with skewed radium systems away from the side of recurrent disease without additional external irradiation.

In Stages III_A and III_B (218 patients) there were 31 central failures (14 percent) and 41 regional failures (19 percent). Main causes of central failure were attributed to massive central and regional disease or distorted anatomy precluding the use of ovoids and requiring the use of a vaginal cylinder. The overwhelming cause of regional failure was the presence of massive disease. In most patients, doses up to 7,000 rads failed to control regional disease.

In conclusion, early stage (Stage I_B and II_A) central failures are usually caused by improper positioning of radium tandem and ovoids, or the use of a vaginal cylinder in place of ovoids. In Stage II_B patients, the addition of an extrafascial conservative hysterectomy six weeks after irradiation in those patients with large, barrel-shaped lesions has resulted in a low incidence (five percent) of central failure. No central failures were seen in patients who had had hysterectomy. All central failures occurred in conjunction with regional failures, and the obvious cause was massive disease in the pelvis. In Stage III patients, massive tumor size limits control of disease.—*Odis D. Skinner, M.D.*

Douglass, H. O., Jr., Wang, J., Takita, H., Wallace, H. J., Friedman, M., and Mindell, E. Improvement in the results of treatment of osteogenic sarcoma. *Surg., Gynec. & Obst.*, May, 1975, *140*, 693–700. (From: The Departments of Surgery, Pediatrics, Thoracic Surgery, Medicine A, Radiation Therapy, and Bone Clinic, Roswell Park Memorial Institute, and the Department of Surgery and Orthopedic Surgery, State University of New York, Buffalo, N. Y.)

A review of results of treatment of patients with osteogenic sarcoma seen at Roswell Park Memorial Institute, Buffalo, New York, was divided into the pre and post 1968 periods.

Prior to 1968, treatment of osteogenic sarcoma by amputation resulted in a 30 percent two year survival rate, and twelve percent five year survival rate. In three of the eight who survived five years, metastases developed. One patient with metastases was apparently salvaged as a consequence of tumor transplantation and exchange of white blood cells between partners. Excision of pulmonary metastases in four of eleven patients resulted in four survivals beyond two years, and two of seven patients treated with radiation therapy survived over two years.

Review of 70 cases of osteogenic sarcoma seen between 1968 and 1973, showed that adriamycin is a major advance in chemotherapy, but its significant role is as adjuvant therapy. It delayed the clinical appearance of metastases by one to two years, with the threat of cardiotoxicity limiting its usefulness.

Only one patient in the recent series had a response to a chemotherapeutic agent other than adriamycin. One patient had a three month remission of pulmonary metastases while being treated with Actinomycin D prior to referral to Roswell Park Memorial Institute. The experience with methotrexate suggested that it was of no value in patients who did not respond to adriamycin. There was no repetition of early successes with immunotherapy and many programs were discontinued.

A patient treated with high dosage radiation therapy to the primary tumor plus a high dose methotrexate-citrovorum rescue showed sterilization of the primary tumor and no evidence of metastases at autopsy six months later (death from unrelated cause). This sterilization has not been previously reported even with equivalent doses of radiation therapy, but radiation therapy has been effective in management of patients.

The dictum of amputation through the joint proximal to the involved bone was not confirmed. There were no local recurrences after high amputation of the femur in eight patients in whom the primary tumor was in the femur. All patients now have bone scans and tomograms prior to amputation so that the line of transection can be planned ten cm or more proximal to the highest level. Tetracycline is administered 18 hours preoperatively and the bone examined under black light at the time of amputation. Also, the resected femur is split longitudinally and viewed with black light to be certain that the

fluorescence is confined to the tumor. A marrow smear at the site of resection is examined immediately for evidence of tumor cells. Immediate fitting of a prosthesis and early ambulation have reduced rehabilitative problems.

Thoracotomy for pulmonary metastases, as often as necessary, has increased the three year survival rate severalfold. The authors stress the value of persistence in management of disease in these patients.—*Lois Cowan Collins, M.D.*

RADIOISOTOPES

FEIGIN, DAVID S., WELCH, DENNIS M., SIEGEL, BARRY A., and JAMES, A. EVERETTE, JR. The efficacy of the brain scan in diagnosis of brainstem gliomas. *Radiology*, July, 1975, *116*, 117–120. (Address: David S. Feigin, M.D., Department of Radiology and Radiological Science, The Johns Hopkins Medical Institutions, Baltimore, Md. 21205.)

The authors reviewed 19 cases of brainstem gliomas proved by surgical biopsy (twelve cases), autopsy (five cases), or pneumoencephalography (four cases). The brain scan results (performed with Tc^{99m} pertechnetate) were correlated with results of other roentgenological investigations (angiography, ventriculography or pneumoencephalography) and with the pathological anatomy.

The brain scan demonstrated the lesion in only one of these patients. This patient had extension of tumor outside the brainstem, infiltrating the superior vermis of the cerebellum and the right brachium conjunctivum into the middle fossa. The angiogram was abnormal in ten of 18 cases. The air study was abnormal in 17 of 19 cases.

The authors conclude that a negative brain scan cannot be used to exclude the existence of a brainstem glioma and that further roentgenological evaluation is necessary.—*W. R. Carpentier, M.D.*

HATTNER, ROBERT S., MILLER, SHELBY W., and SCHIMMEL, DANIEL. Significance of renewal asymmetry in bone scans: experience in 795 cases. *J. Nuclear Med.*, Feb., 1975, *16*, 161–163. (Address: R. S. Hattner, M.D., Department of Radiology, Nuclear Medicine Section, University of California Medical Center, San Francisco, Calif. 94143.)

In a retrospective survey, 795 consecutive whole body bone scans were analyzed to determine the presence of significant renal asymmetry. Thereafter the patient's record and roentgenograms were reviewed. Patients were then categorized into unclassifiable (no clinical data available), concordant with clinical-pathophysiological findings (unequivocal evidence of renal abnormality) and discordant with clinical-pathophysiological findings (not supported by clinical data). Scans were performed following the intravenous administration of fluorine-18 or technetium pyrophosphate. Asymmetry was suspected in 17 patients scanned with fluorine-18 and the renal status was confirmed in twelve as being abnormal. Twenty patients were detected with pyrophosphate and abnormal renal status confirmed in 18. This study suggests that the technetium pyrophosphate scan is more sensitive to the detection of renal asymmetry (approaching ten percent of patient scan) and these images are a sensitive index of renal abnormality.—*William H. Strauss, M.D.*

GATES, GARY F., and DORE, EARL K. Detection of craniosynostosis by bone scanning. *Radiology*, June, 1975, *115*, 665–671. (Address: Gary F. Gates, M.D., Division of Nuclear Medicine, Stanford Medical Center, Stanford, Calif. 94305.)

Twenty-nine children were studied with bone-seeking radiopharmaceuticals (F^{18} and Tc^{99m} polyphosphate) to determine normal and abnormal growth patterns of cranial sutures. Fifteen were normals, being examined for other reasons, 13 had known craniosynostosis of the varying types, and one had typical Lückenschädel skull. The age range of these children was from newborn to 16 years. Rectilinear scanner and camera images gave identical results. Three surgical specimens became available, and these were imaged for comparison to the preoperative scans. The lateral skull scan provided the most information over-all. It is imperative that the scan be correlated with the child's age.

Localized sutural hyperactivity is demonstrable during periods of normal active growth of the skull, *i.e.*, lambdoidal from seven weeks to two years, while the posterior fontanelle is closing; and bregmatic increasing progressively during the first three years of life when the anterior fontanelle closes. The mid-portion of the sagittal curve appeared to lag behind the former two areas. Squamosal growth activity was rare after the first year, metopic activity diminished after the second year, and coronal activity was demonstrable up to the third year.

Craniosynostosis was identifiable as increased radioactivity (osteoblastic response), in the suture(s) of origin, above the norm, so long as the fusion process was active and progressing along the course of the suture. When fusion became complete, radioactivity was noted to diminish in relatively intensity.

The one case of Lückenschädel skull exhibited relatively decreased radioactivity in all sutures and membraneous bone centers, but dense concentration in the enchondral base.

Calvarial bone imaging appears to be a sensitive indicator of regional growth in the developing skull, and discloses the prematurely closing suture by the abnormal concentration pattern as the process re-

lentlessly extends itself along the length of the involved suture.—*F. C. Petty, M.D.*

Teates, Charles D., and Hunter, James G., Jr. Gallium scanning as a screening test for inflammatory lesions. *Radiology*, Aug., 1975, *116*, 383–387. (Address: Charles D. Teates, M.D., Department of Radiology, University of Virginia School of Medicine, Charlottesville, Va. 22901.)

In this study, 42 patients with unexplained fever were scanned 48 hours after the intravenous injection of gallium-67 citrate to locate occult inflammatory lesions. Repeat scans of the abdomen were done at 48 and 96 hours when initial bowel evacuation was inadequate.

Twenty-five patients had scans which were interpreted as normal or only questionably abnormal. Of these, one patient had widespread histiocytic lymphoma, or possible sarcoidosis; eight patients were discharged with a diagnosis of fever of unknown origin; and eleven had diseases not usually associated with focal inflammation.

Seventeen patients had abnormal scans. Of these, one was due to a fecal impaction, and four had no proof of focal disease at the positive site. Twelve patients had abnormally increased gallium activity corresponding to focal inflammatory disease such as abscess or cellulitis. In nine of these patients, the scan yielded information not provided by previous studies.

The authors feel that gallium scanning has proved useful as a screening test for localized inflammatory disease. A normal scan did not rule out inflammatory lesions, but no significant pyogenic lesions were missed in this series.—*W. R. Carpentier, M.D.*

Waxman, Alan D., and Siemsen, Jan K. Gallium gallbladder scanning in cholecystitis. *J. Nuclear Med.*, Feb., 1975, *16*, 148–150. (Address: A. D. Waxman, M.D., Department of Radiology, Nuclear Medicine Section, LAC/USC Medical Center, 1200 N. State Street, Los Angeles, Calif. 90033.)

Ten patients with suspected gallbladder disease were scanned 20–48 hours following intravenous administration of gallium citrate. Marked increase in tracer concentration was found in six of ten patients, five of whom had acute cholecystitis and one found to have chronic cholecystitis. The region of the gallbladder was not visualized in four patients, all of whom had chronic cholecystitis. Gallium scanning of the gallbladder proved to be most helpful in the evaluation of acute inflammatory disease but becomes less helpful as the disease enters the chronic stages.—*William H. Strauss, M.D.*

Krishnamurthy, G. T., Huebotter, R. J., Walsh, C. F., Taylor, J. R., Kehr, M. D., Tubis, M., and Biahd, W. H. Kinetics of ^{99m}Tc-labeled pyrophosphate and polyphosphate in man. *J. Nuclear Med.*, Feb., 1975, *16*, 109–115. (Address: G. T. Krishnamurthy, Nuclear Medicine Service (691/172A), VA Wadsworth Hospital Center, Los Angeles, Calif. 90073.)

Ten patients with suspected bone lesions were studied following the intravenous administration of 15 mCi of pyrophosphate and repeated two to 14 days later with 15 mCi of a polyphosphate. Following injection, blood was collected at ten and 30 minutes and then hourly for four hours following injection. Radioactivity in whole blood, plasma, and red cells was determined and results expressed as percent of the injected dose per liter. Prior to counting, the red cells were washed in saline three times, the activity remaining in the cells after the first wash was considered 100 percent and the reduced activity following subsequent washes expressed as a percentage of loss after each wash. The activity in plasma and that bound to protein and to each protein fraction was determined. Urine was collected at hourly samples for four hours and the tracer concentration in each sample expressed as a percentage of the administered dose excreted/hour. In addition, serum calcium, alkaline phosphatase and total protein were measured before and one hour after administration of each tracer. Skeletal images of identical regions were obtained with both agents four hours following injection using a scintillation camera. The time taken to accumulate 50,000 counts for each view was noted.

The blood clearance of both technetium pyrophosphate and technetium polyphosphate was bi-exponential. The clearance of half-time of fraction one was 13.6 minutes for both tracers and 380 minutes for the second fraction with pyrophosphate was 512 minutes. At four hours, a liter of blood had a mean radioactivity of 2.5 percent of the injected dose with polyphosphate compared to 1.9 percent with technetium pyrophosphate. One hour following tracer administration, 84 percent of plasma radioactivity was protein bound with pyrophosphate and 74 percent with polyphosphate. Most of the bound activity was associated with the globulin fraction. Both tracers were bound by the red cells; technetium pyrophosphate was less firmly bound than polyphosphate. There were no significant changes in the serum calcium values. Urinary excretion of both tracers was similar in the first hours; 16.4 percent for pyrophosphate and 15.8 percent for polyphosphate. During the next three hours, pyrophosphate was excreted slightly more rapidly than polyphosphate and at the end of four hours, the mean of 32 percent of pyrophosphate and 29 percent of polyphosphate had been excreted in the urine. Bone images obtained with pyrophosphate were qualitatively sharper and had better resolution than similar images obtained with polyphosphate. There was no

difference between the agents in the sensitivity of lesion detection.—*William H. Strauss, M.D.*

ULTRASOUND

Smith, Edward H., Bartrum, Royal J., Jr., Chang, Young C., D'Orsi, Carl J., Lokich, Jacob, Abbruzzese, Americo, and Dantono, John. Percutaneous aspiration biopsy of the pancreas under ultrasonic guidance. *New England J. Med.*, April, 1975, *292*, 825–828. (From: Department of Radiology, Harvard Medical School, 25 Shattuck Street, Boston, Mass. 02115.)

Carcinoma of the pancreas is the fourth most frequent fatal neoplasm in the United States. The pancreas is one of the most difficult organs to evaluate for neoplastic lesions. The development of special ultrasound biopsy transducers that allow accurate placement of the biopsy needle has made percutaneous biopsy of the pancreas an easier procedure to perform.

Seven patients who were clinically suspected of having carcinoma of the pancreas but with little or no roentgenologic or biochemical confirmation of the diagnosis, were examined with percutaneous aspiration biopsy of the pancreas utilizing ultrasonic guidance. Although masses in the region of the pancreas greater than 3 cm in diameter can be identified with ultrasound, these lesions cannot be determined as being benign or malignant. The fine-needle aspiration is used to obtain a cytologic specimen for specific diagnosis.

In the seven patients examined with this method, five had cytologically conclusive or strongly suggestive evidence of carcinoma. During biopsy, either the bowel or liver is traversed by the needle. However, no serious morbidity occurred with the 23 gauge needle.

A negative cytologic diagnosis does not exclude the presence of a malignant lesion but the ability to obtain a high percentage of positive cytologic results adds to the diagnostic armamentarium in suspected carcinoma of the pancreas. Since the prognosis for carcinoma of the pancreas is poor in that only 25 percent of lesions are considered resectable and the five year survival rate is twelve percent, it is unlikely that biopsy will result in earlier diagnosis or prolonged life expectancy since a mass must be at least 3 cm in diameter before it can be reliably detected by ultrasound. However, the percutaneous biopsy can possibly provide a definitive diagnosis with less morbidity and patient discomfort.—*Ronald J. Ross, M.D.*

McCaffrey, J., Williams, O., and Stathis, M. Diagnosis of deep venous thrombosis using a Doppler ultrasonic technique. *Surg., Gynec. & Obst.*, May, 1975, *140*, 740–742. (From: The Department of Surgery, University of Queensland, and Repatriation General Hospital, Greenslopes, Australia.)

The Doppler Ultrasonic Technique offers an acceptably accurate and completely non-invasive, non-hazardous method of diagnosing deep venous thrombosis, particularly above the level of the knee where the thromboses are most likely to be responsible for pulmonary emboli. Venography is highly accurate but requires special roentgenographic skills and techniques, is not entirely without hazard, and can be performed only once, or at most twice. The iodinated fibrinogen labeled fibrinogen test is also highly accurate, but must be administered preoperatively with the thyroid gland blocked by the administration of iodine. The actual testing is time consuming.

At the Repatriation Hospital, University of Queensland, Greenslopes, Australia, 118 patients were examined postoperatively by the Doppler Ultrasonic technique and also by venography (42 patients) or iodinated fibrinogen test (86 patients), or both (ten patients). Of these, 22 patients (18.6 percent) were shown to have deep venous thrombosis by either venography or the fibrinogen test. The ultrasonic test detected 21 of these (17.7 percent), two of which had ileofemoral thromboses which were detected by the ultrasonic method. Of the remaining 19 patients, 17 had thromboses in the calf by both the ultrasonic method and one, or both, of the other methods. The three false negative studies by ultrasound showed moderate but definite rises in isotopic counts on the third, fifth and sixth days, respectively, with no proximal extension, and no evidence of pulmonary embolism.

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The method does require careful technique and anatomical knowledge and experience on the part of the operator. A machine using a frequency of at least eight megahertz must be used.

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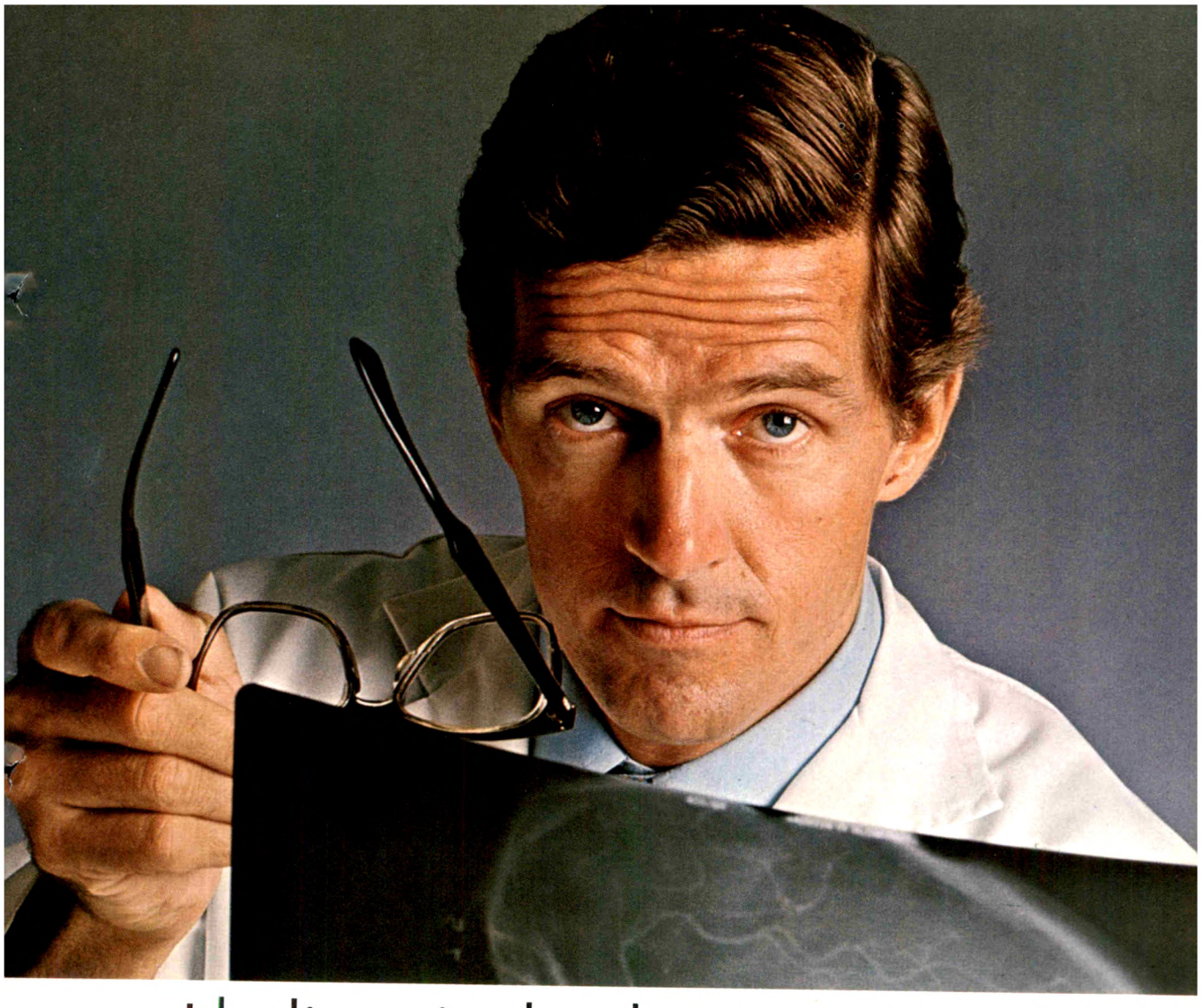

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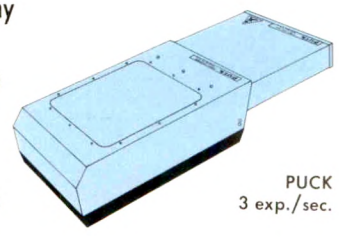

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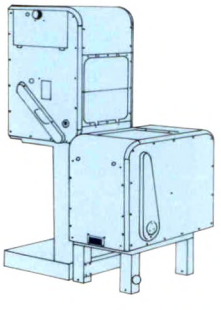

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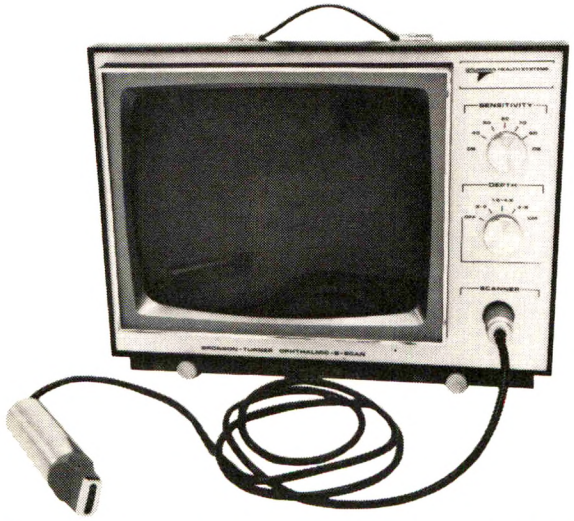

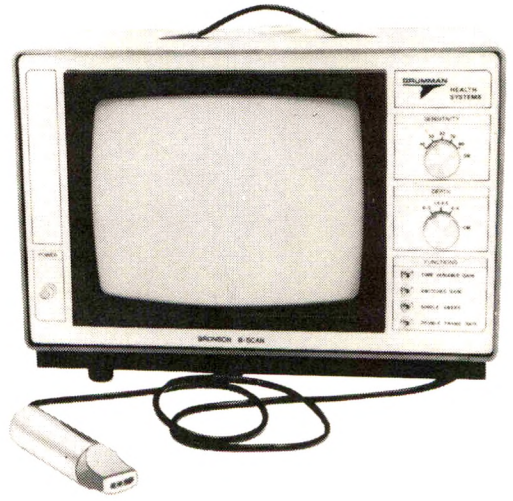

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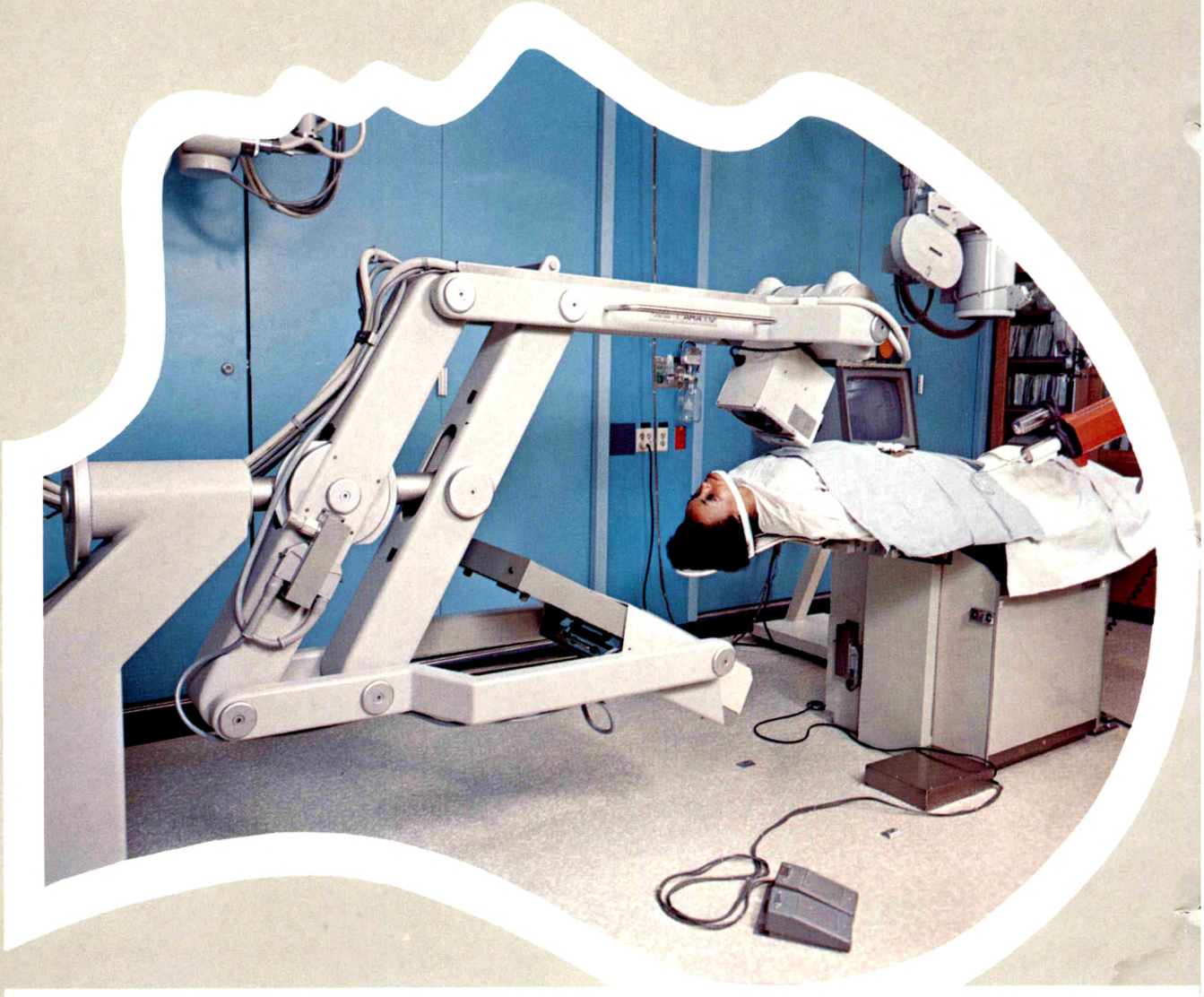

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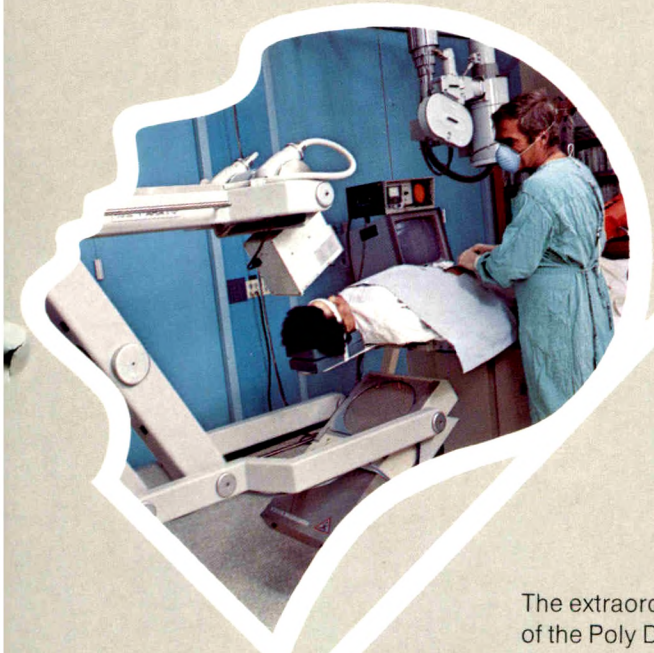

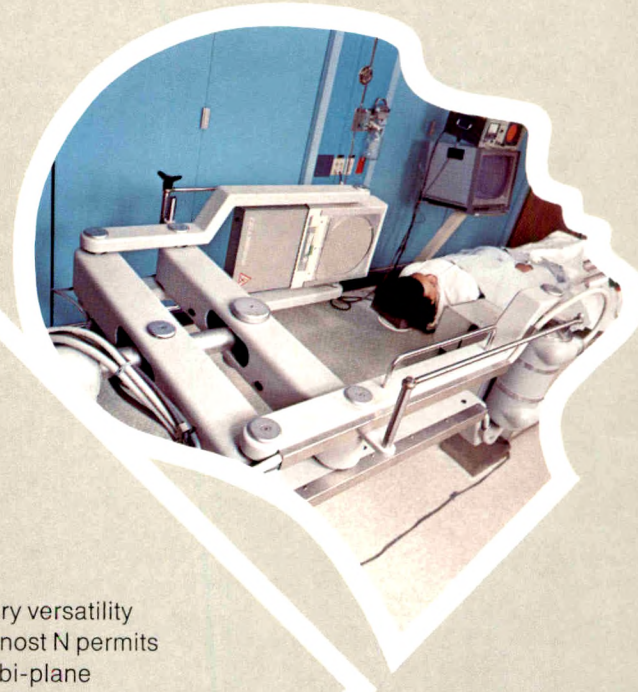

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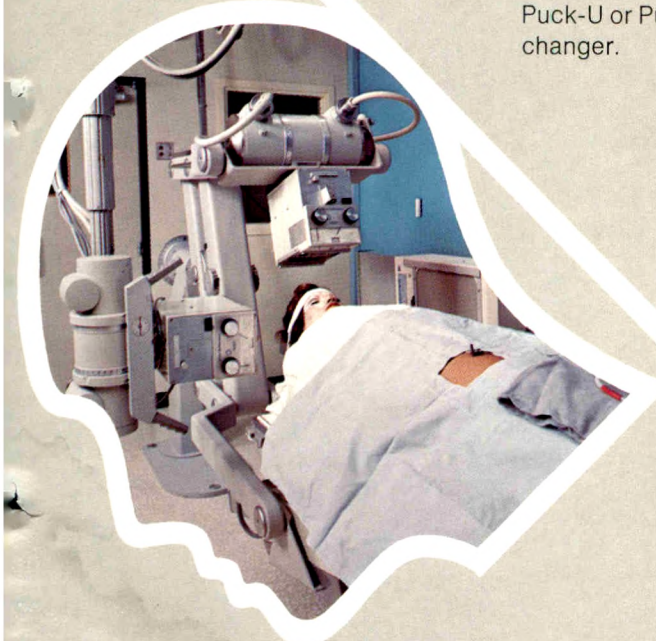

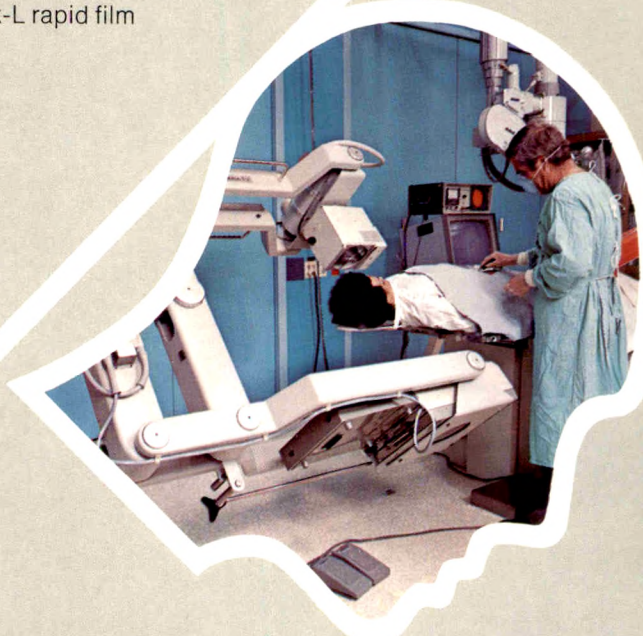

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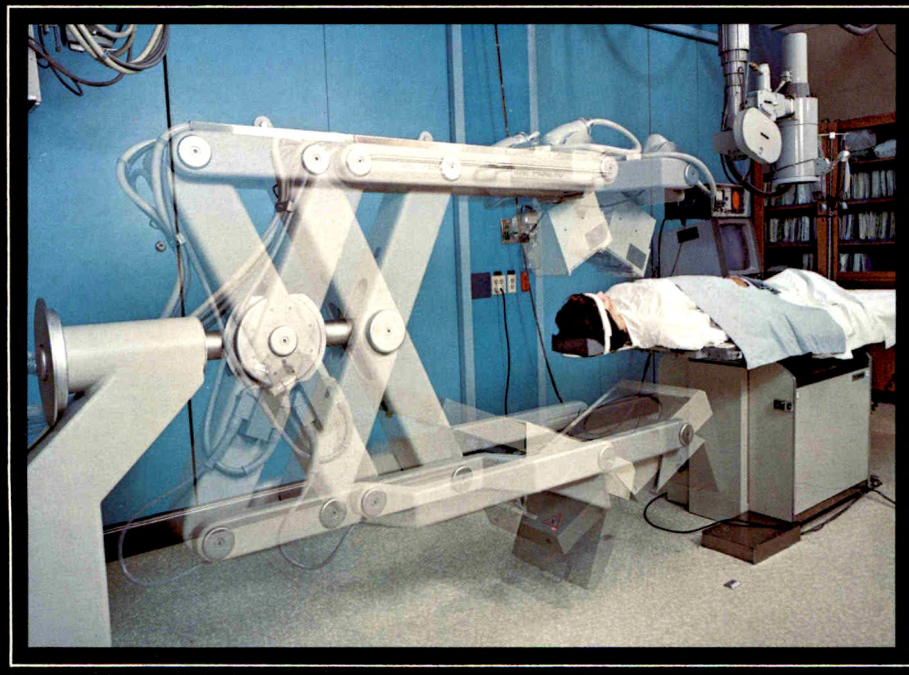

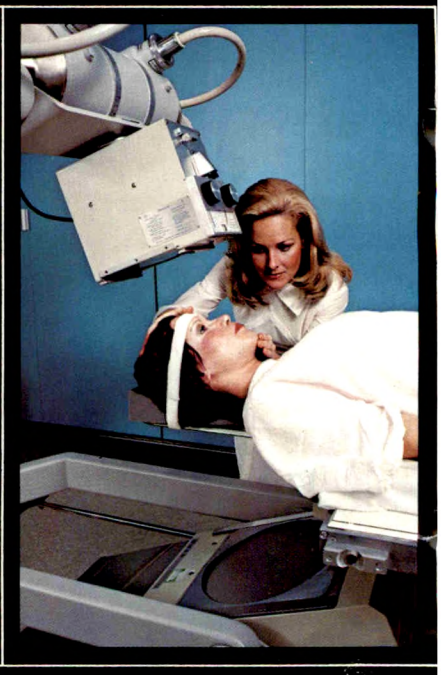

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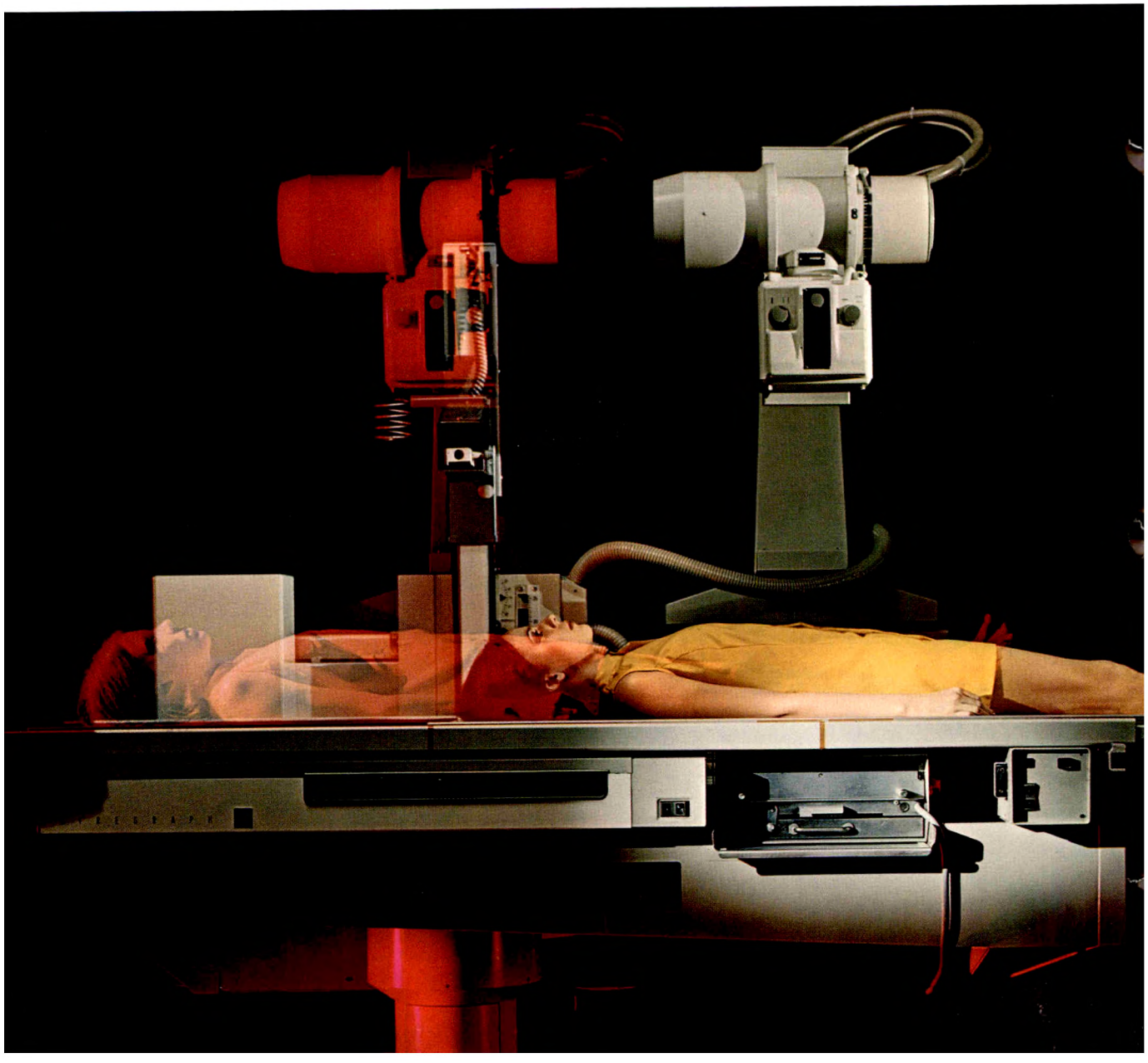

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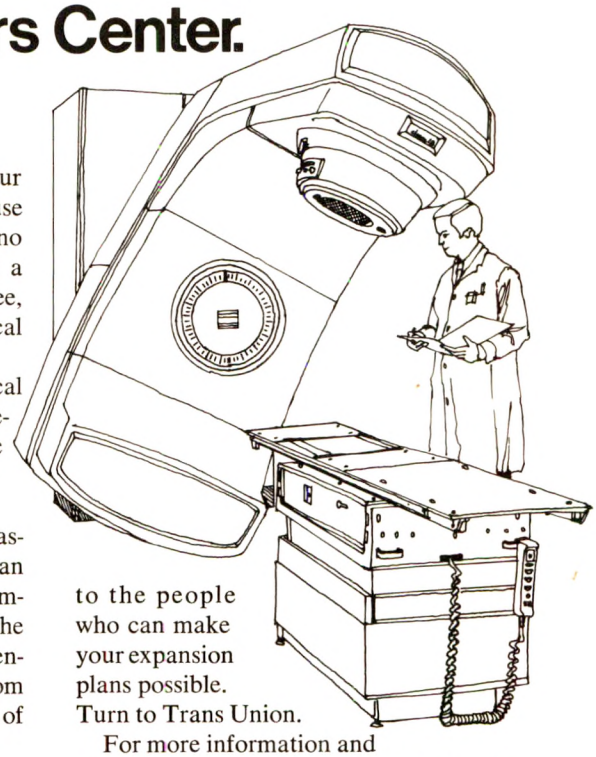

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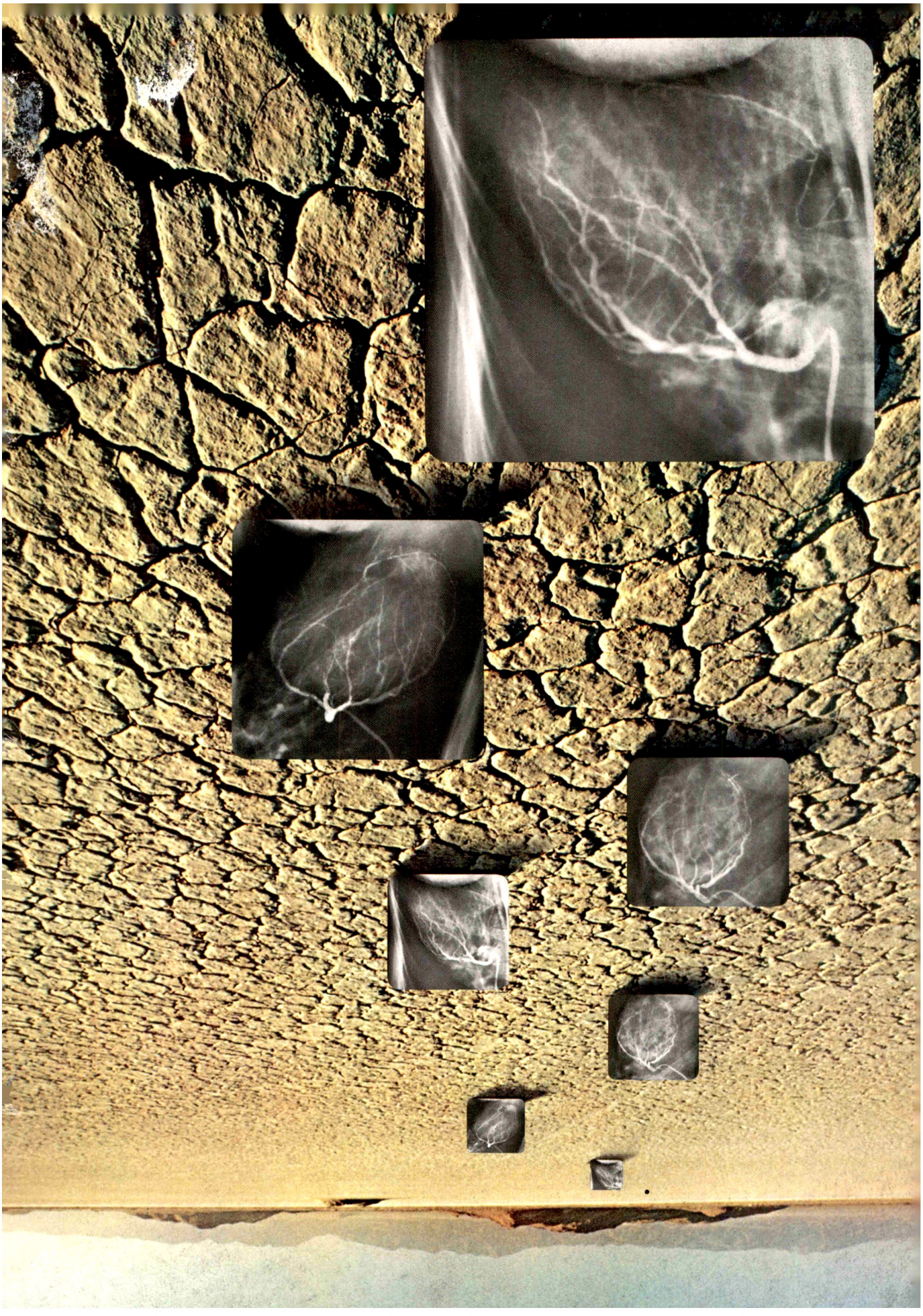

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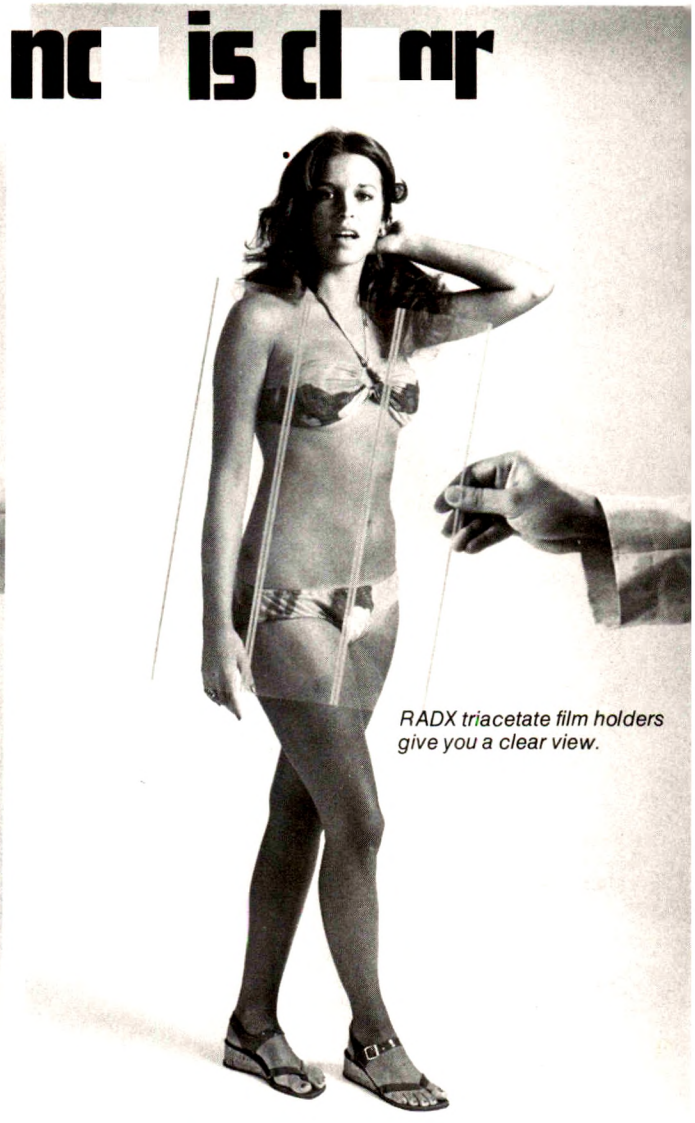

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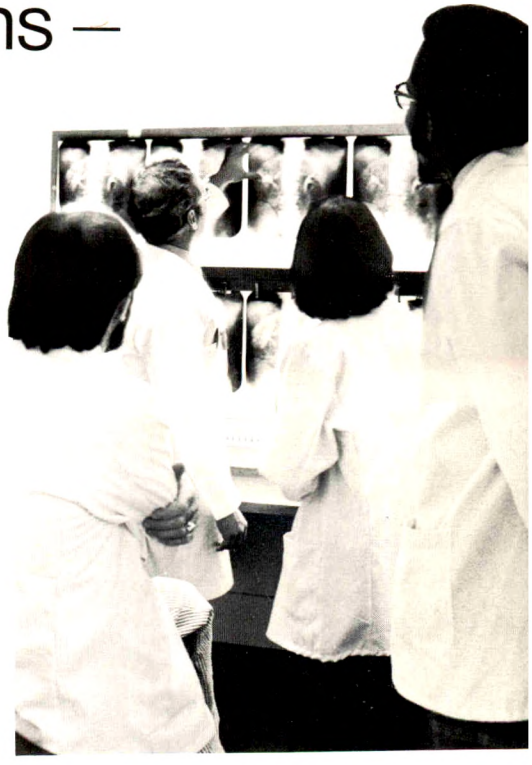

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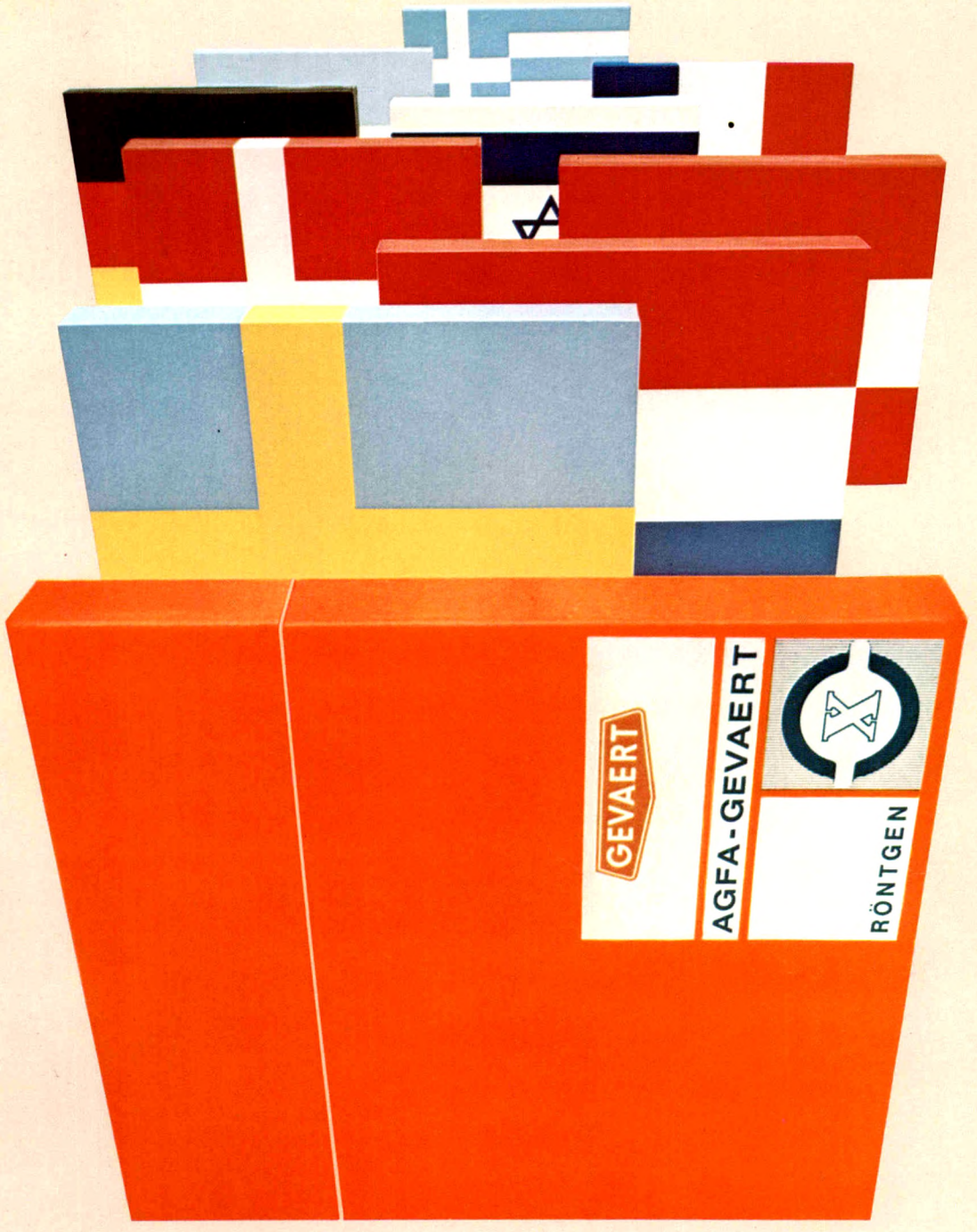

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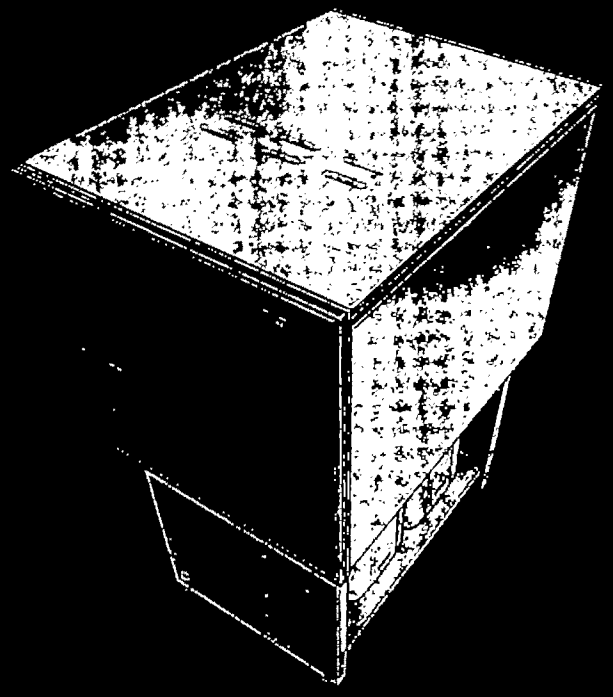

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The first day will consist of small workshops in all the disciplines mentioned. The next two days will consist of formal lectures and round-table discussions with the speakers. A Saturday evening dinner dance is again scheduled. The second largest Radiological Technical Exhibit in the country will be open for viewing on all three days.

A post convention seminar in Hawaii is scheduled again because of the enormous success of last year's program. This faculty will cover topics in Diagnostic Radiology, Nuclear Medicine and Ultrasound. This week long seminar will be equally divided on the islands of Maui (Maui Surf) and Hawaii (Kona Surf). An optional extra week after the seminar can be arranged at Kahala Hilton (Oahu) or a week in Tahiti.

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Robert N. Berk, M.D. . . . . University of Texas
Robert G. Fraser, M.D. . . . . Royal Victoria Hospital, Montreal
Robert H. Freiberger, M.D. . . . . The Hospital for Special Surgery, N.Y.C.
Derek C. Harwood-Nash, M.B., Ch.B., F.R.C.P. (C) . . . . . The Hospital for Sick Children, Toronto
Erich K. Lang, M.D. . . . . Louisiana State University

Radiation Therapy

Thomas C. Hall, M.D. . . . . Los Angeles County-University of Southern California Medical Center
Richard H. Jesse, M.D. . . . . M.D. Anderson Hospital and Tumor Institute
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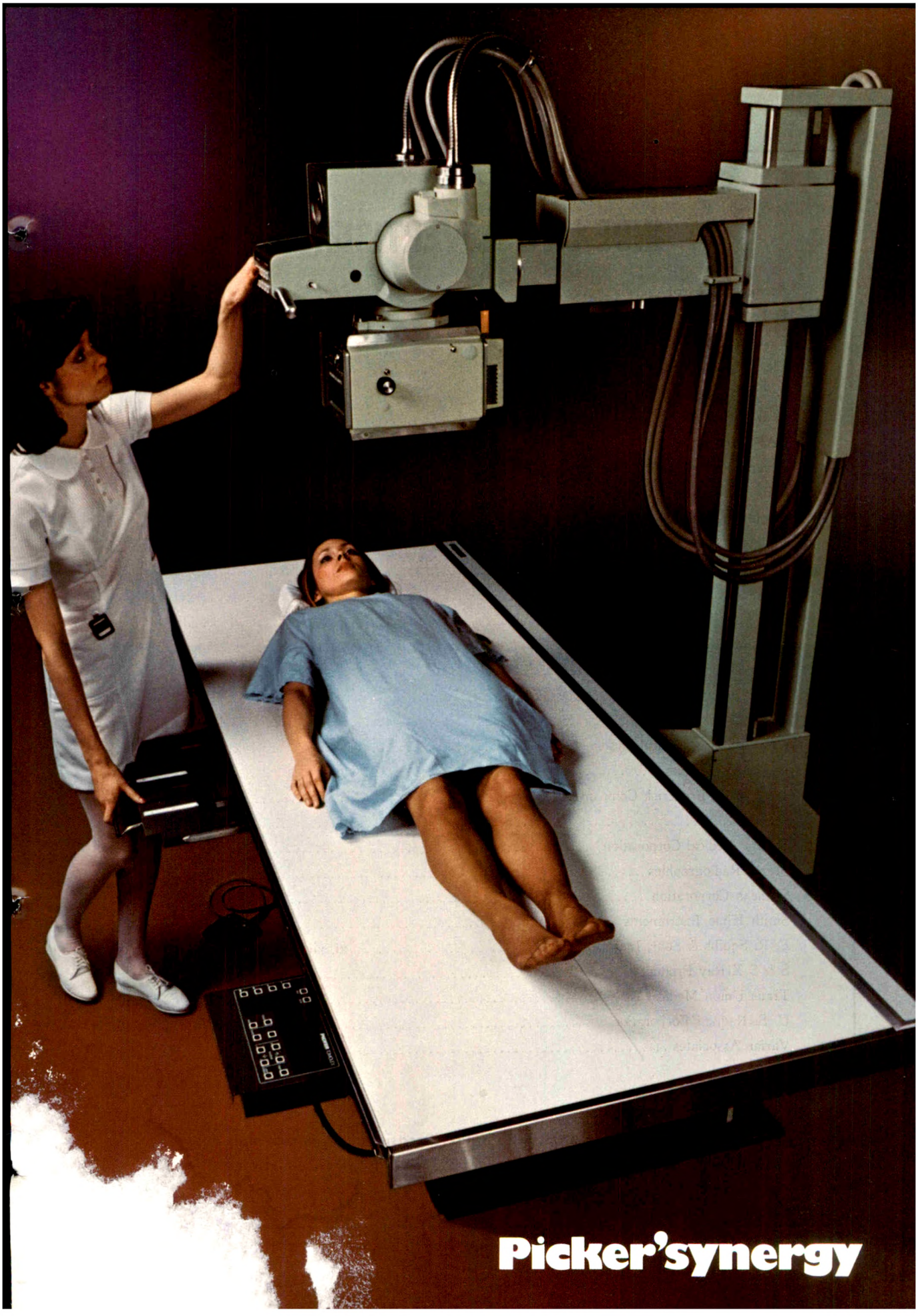
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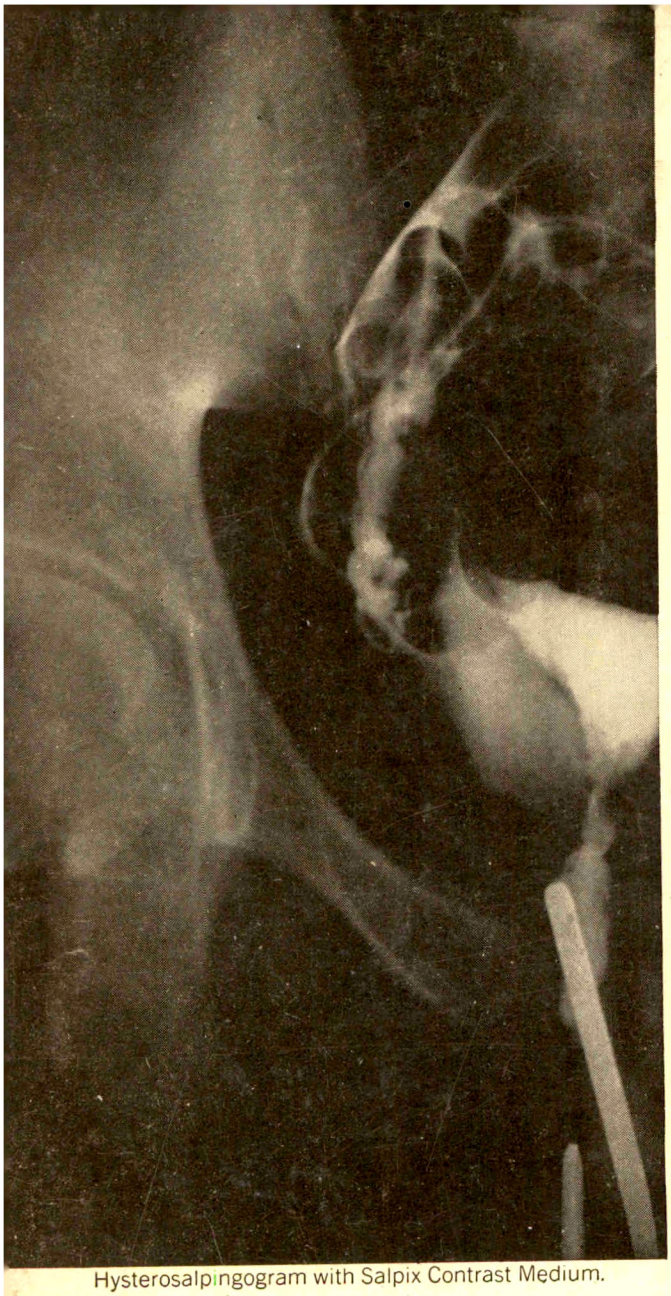

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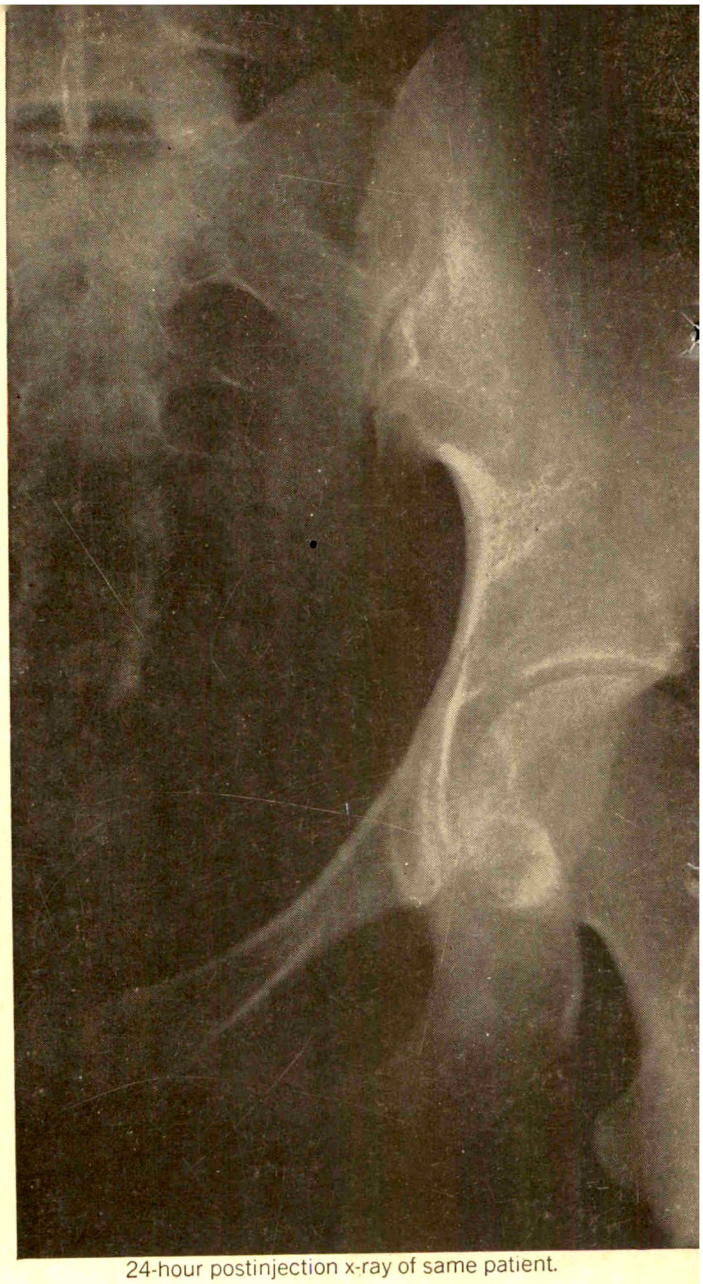

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Issued Monthly for the American Roentgen Ray Society by
CHARLES C THOMAS · PUBLISHER · SPRINGFIELD · ILLINOIS · 62717

VOLUME 125 DECEMBER, 1975 NUMBER 4

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[FOUNDED IN 1906 AS THE AMERICAN QUARTERLY OF ROENTGENOLOGY]

The AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

TRAIAN LEUCUTIA Editor Emeritus

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VOLUME 125

SEPTEMBER–DECEMBER, 1975

CHARLES C THOMAS : SPRINGFIELD, ILLINOIS

1975

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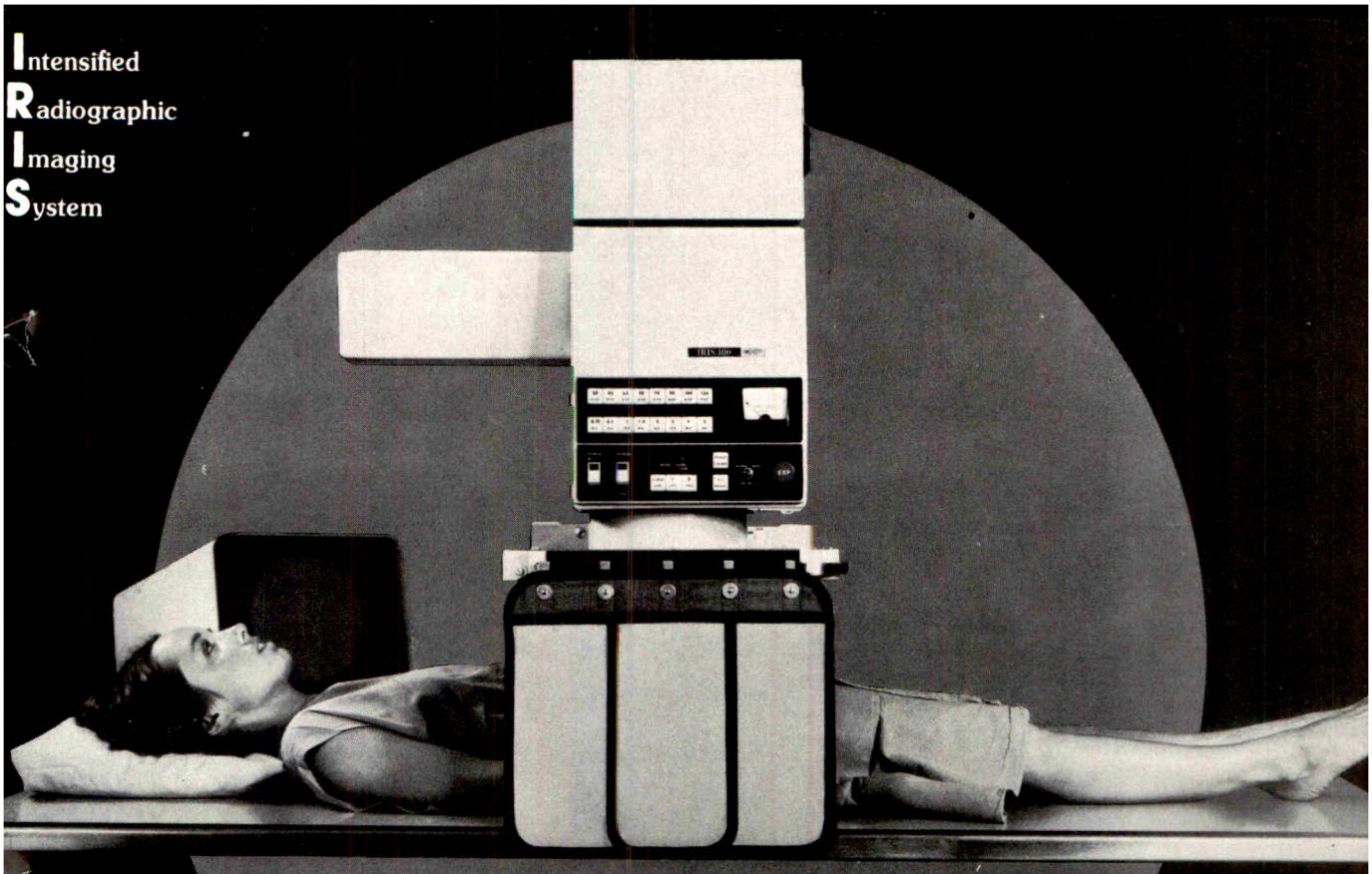

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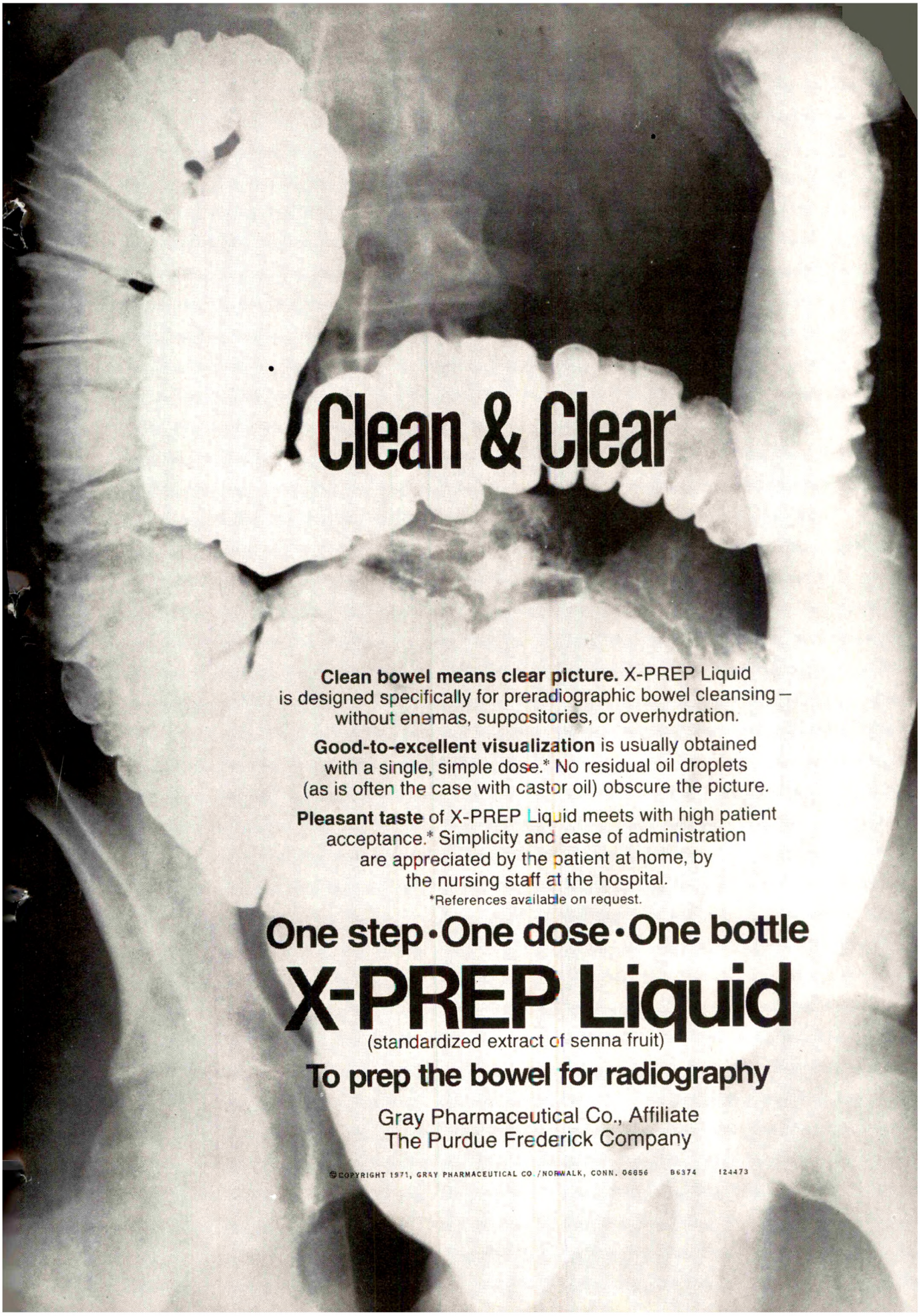

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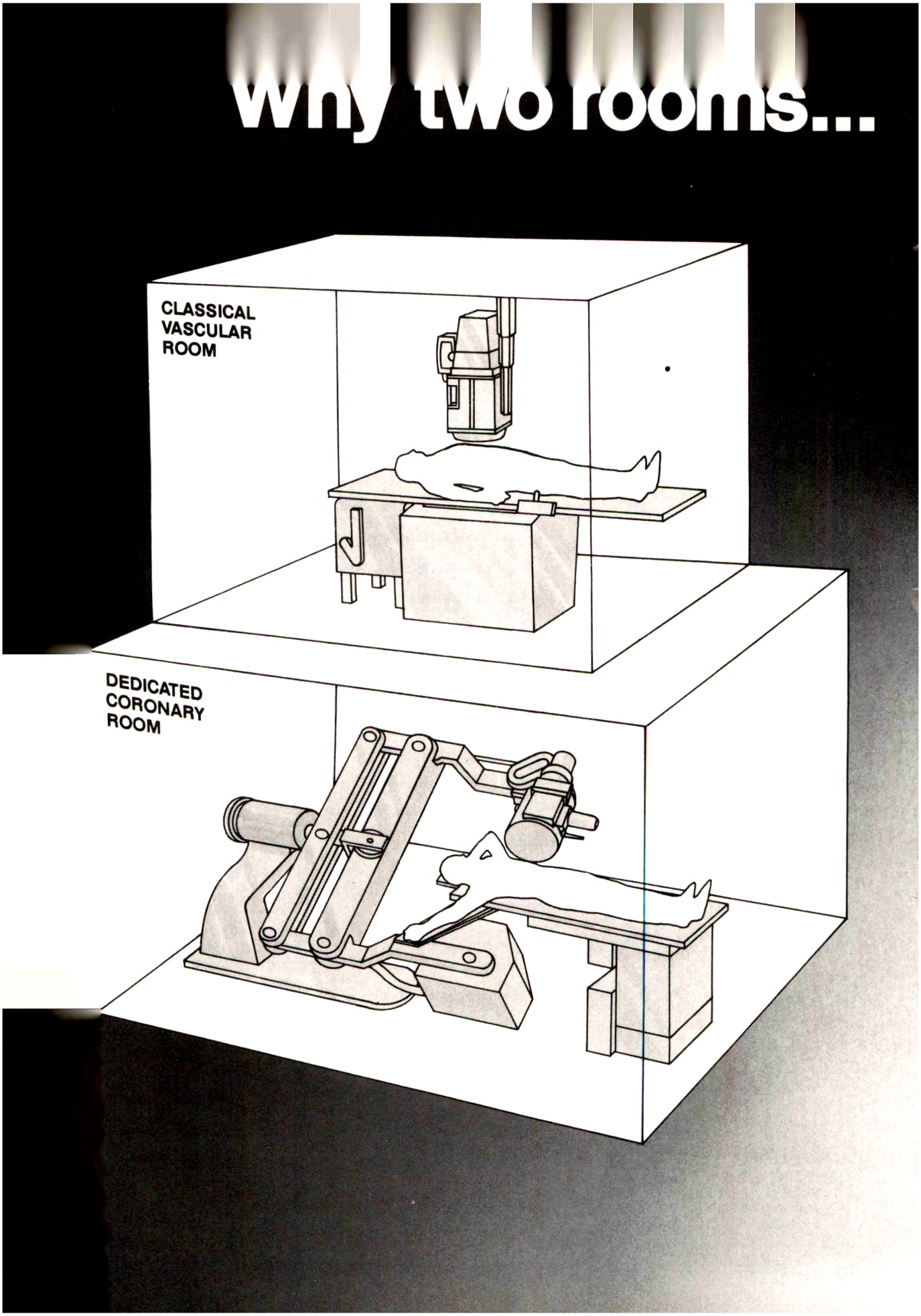

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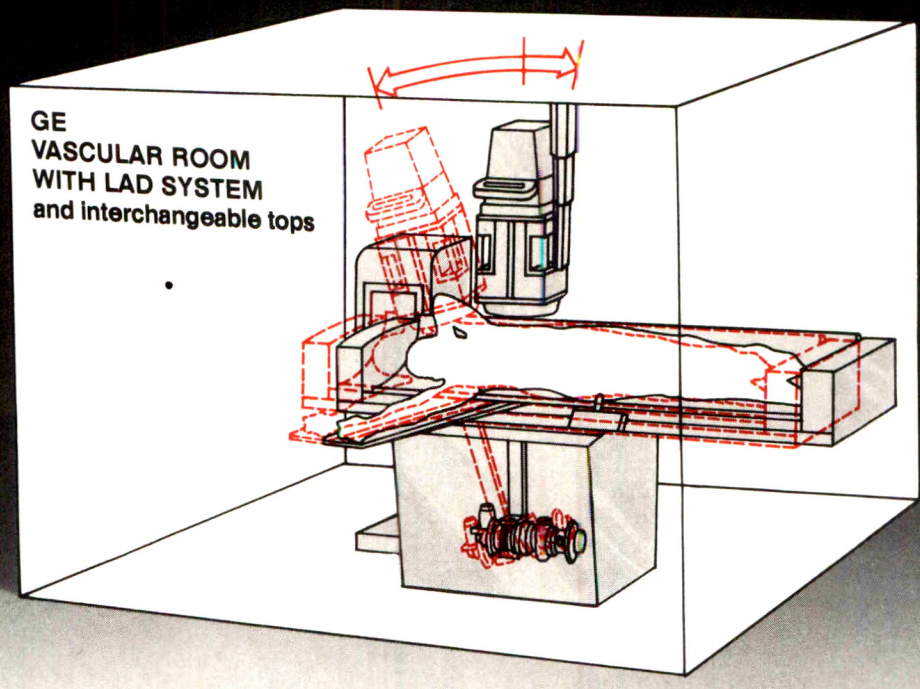

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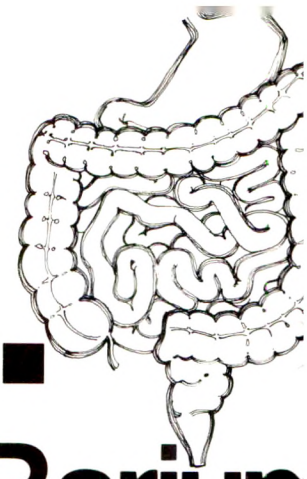

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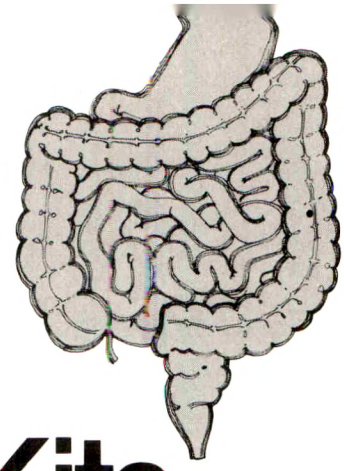

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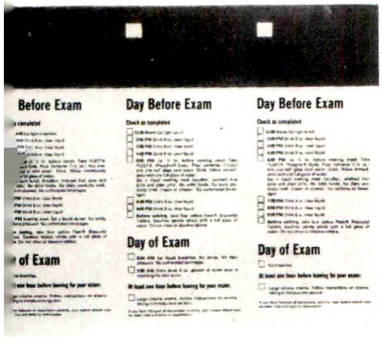

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References:
1. *Detection of Colon Lesions*, First Standardization Conference, Amer. College of Radiology, (Chicago, Ill., 1973), pp. 203-204.
2. *Ibid*, p. 76.

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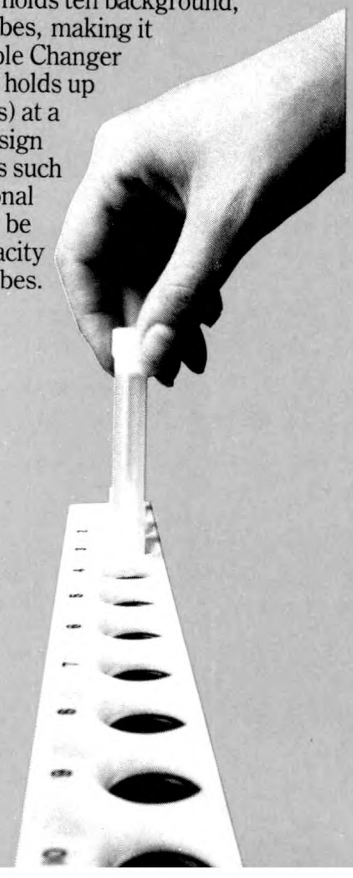

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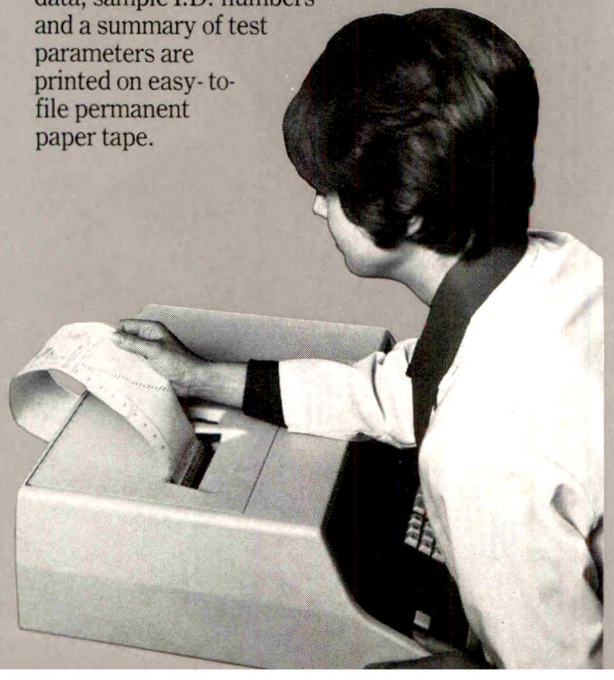

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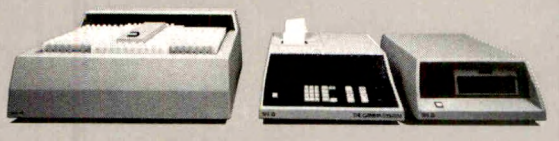

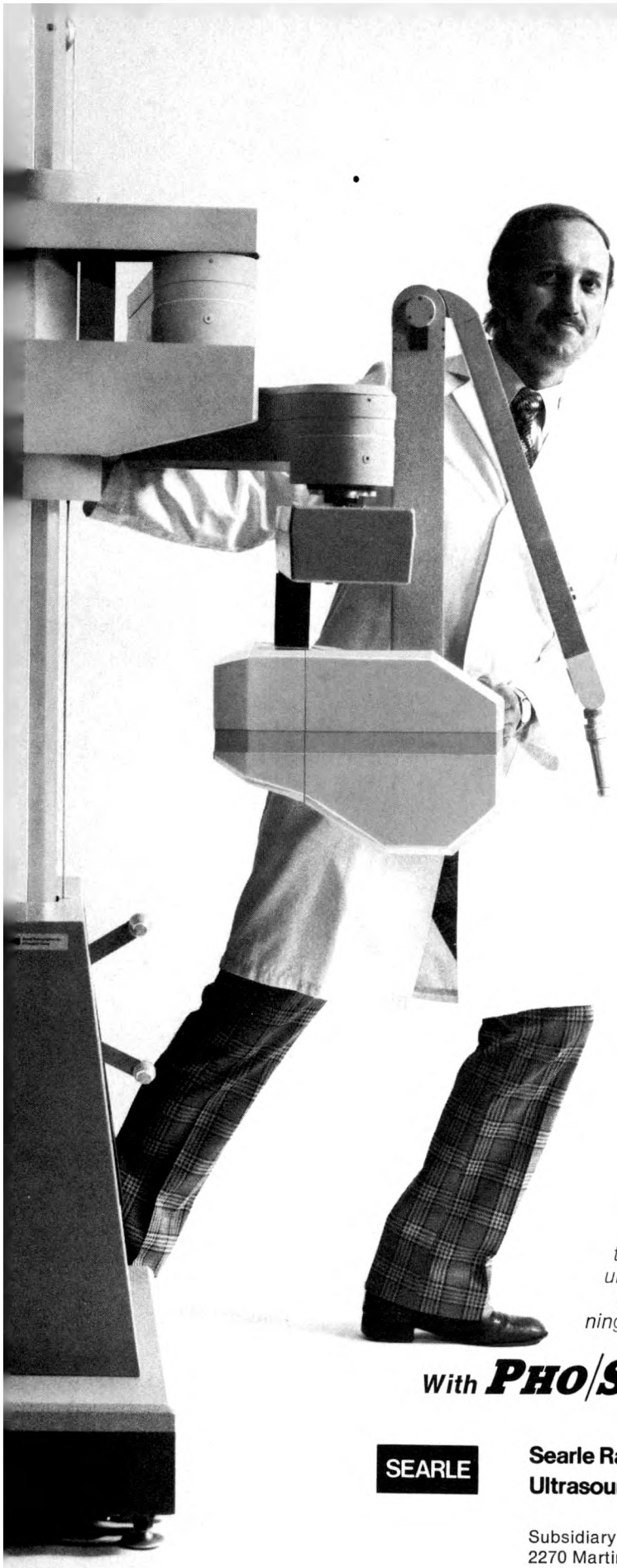

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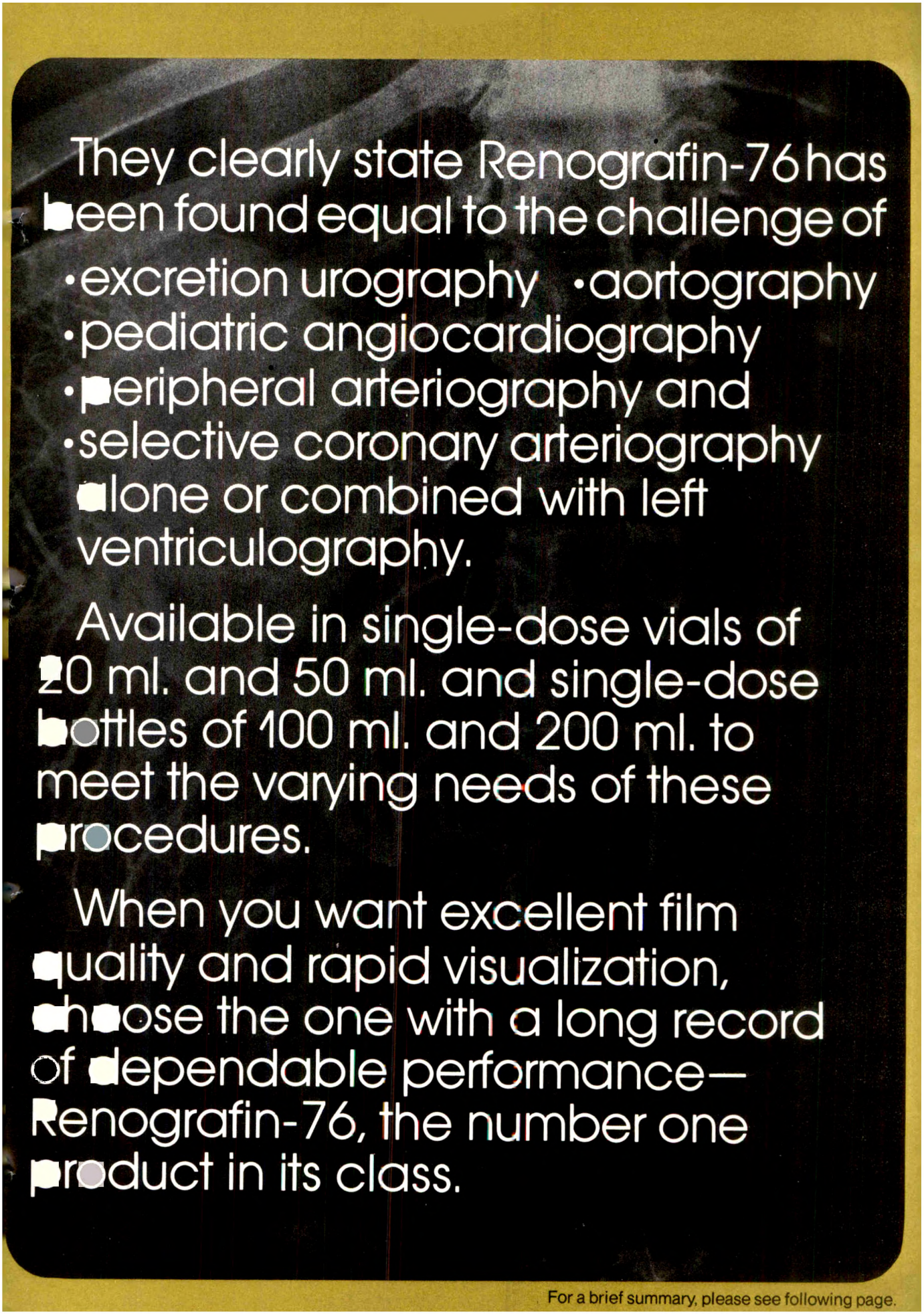

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Renografin-76 is supplied as a sterile, aqueous solution providing 66% meglumine diatrizoate and 10% sodium diatrizoate with 0.32% sodium citrate as a buffer and 0.04% disodium edetate as a sequestering agent. The solution contains approximately 37% (370 mg./ml.) bound iodine and *approximately 4.48 mg. (0.19 mEq.) sodium per ml.*

CONTRAINDICATIONS: In patients with a hypersensitivity to salts of diatrizoic acid. Urography contraindicated in patients with anuria.

WARNINGS: A definite risk exists with the use of contrast agents in excretion urography in patients with multiple myeloma. There has been anuria with progressive uremia, renal failure and death. This risk of the procedure in these patients is not a contraindication; however, partial dehydration in preparation for study is not recommended since it may predispose for precipitation of myeloma protein in renal tubules. No therapy, including dialysis, has been successful in reversing this effect. Myeloma should be considered in persons over 40 before undertaking urographic procedures.

In cases of known or suspected pheochromocytoma, if the physician feels that the possible benefits outweigh the considered risks, radiopaque materials should be administered with extreme caution; however, an absolute minimum of material should be injected, the blood pressure should be assessed throughout the procedure, and measures for treating a hypertensive crisis should be available.

Contrast media may promote sickling in homozygous individuals when injected I.V. or intra-arterially. Although a history of sensitivity to iodine *per se* or to other contrast media is not an absolute contraindication, administration of diatrizoate requires extreme caution in such cases. Renografin-76 should be used in pregnant patients only when the physician deems its use essential to the welfare of the patient since safe use during pregnancy has not been established. Perform thyroid function tests prior to administration of Renografin-76 since iodine-containing contrast agents may alter the test results.

Weigh the inherent risks against necessity for performing *angiocardiography* in cyanotic infants and patients with chronic pulmonary emphysema. In *pediatric angiocardiography*, a dose of 10 to 20 ml. may be particularly hazardous in infants weighing less than 7 kg.; this risk is probably significantly increased if these infants have preexisting right heart "strain," right heart failure, and effectively decreased or obliterated pulmonary vascular beds. Perform *urography* with extreme caution in persons with severe concomitant hepatic and renal disease. Perform *selective coronary arteriography* only in selected patients and those in whom expected benefits outweigh the procedural risk.

PRECAUTIONS: Diagnostic procedures involving use of contrast agents should be performed under the direction of personnel with prerequisite training and a thorough knowledge of the particular procedure. Appropriate facilities should be available for coping with situations which may arise as a result of the procedure and for emergency treatment of severe reactions to the contrast agent itself; competent personnel and emergency facilities should be available for at least 30 to 60 minutes after I.V. administration since delayed reactions have been known to occur. These severe life-threatening reactions suggest hypersensitivity to the contrast agent. A personal or family history of asthma or allergy or a history of a previous reaction to a contrast agent warrants special attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 ml. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated.

Renal toxicity has been reported in a few patients with liver dysfunction who were given oral cholecystographic agents followed by urographic agents; therefore, if known or suspected hepatic or biliary disorder exists, administration of Renografin-76 should be postponed following the ingestion of cholecystographic agents. Consider the functional ability of the kidneys before injecting Renografin-76. Use cautiously in severely debilitated patients and in those with marked hypertension. Bear in mind the possibility of thrombosis when using percutaneous techniques.

In *excretion urography*, adequate visualization may be difficult or impossible in uremic patients or others with severely impaired renal function (see Contraindications). In *aortography* repeated intra-aortic injections may be hazardous; this also applies to *pediatric angiocardiography* particularly in infants weighing less than 7 kg. (see Warnings). In *peripheral arteriography*, hypotension or moderate decreases in blood pressure seem to occur frequently with intra-arterial (brachial) injections; this is transient and usually requires no treatment. Monitor blood pressure during the immediate 10 minutes after injection. It is recommended that *selective coronary arteriography* not be performed for about 4 weeks after diagnosis of myocardial infarction; mandatory prerequisites to this procedure are experienced personnel, ECG monitoring apparatus, and adequate facilities for immediate resuscitation and cardioversion.

ADVERSE REACTIONS: Nausea, vomiting, flushing, or a generalized feeling of warmth are the reactions seen most frequently with intravascular injection. Symptoms which may occur are chills, fever, sweating, headache, dizziness, pallor, weakness, severe retching and choking, wheezing, a rise or fall in blood pressure, facial or conjunctival petechiae, urticaria, pruritus, rash, and other eruptions, edema, cramps, tremors, itching, sneezing, lacrimation, etc. Antihistaminic agents may be of benefit; rarely, such reactions may be severe enough to require discontinuation of dosage. There have been a few reports of a burning or stinging sensation or numbness, of venospasm or venous pain, and of partial collapse of the injected vein. Neutropenia or thrombophlebitis may occur. Severe reactions which may require emergency measures (see Precautions) are a possibility and include cardiovascular reaction characterized by peripheral vasodilatation with hypotension and reflex tachycardia, dyspnea, agitation, confusion, and cyanosis progressing to unconsciousness. An allergic-like reaction ranging from rhinitis or angioneurotic edema to laryngeal or bronchial spasm or anaphylactoid shock may occur. Temporary renal shutdown or other nephropathy may occur.

Adverse reactions as a consequence of *excretion urography* include cardiac arrest, ventricular fibrillation, anaphylaxis with severe asthmatic reaction, and flushing due to generalized vasodilatation. Risks of *aortography* procedures include injury to aorta and neighboring organs, pleural puncture, renal damage (including infarction and acute tubular necrosis with oliguria and anuria), accidental selective filling of right renal artery during translumbar procedure in presence of preexistent renal disease, retroperitoneal hemorrhage from translumbar approach, spinal cord injury and pathology associated with syndrome of transverse myelitis, generalized petechiae, and death following hypotension, arrhythmia, and anaphylactoid reactions. In *pediatric angiocardiography*, arrhythmia and death have occurred. During *peripheral arteriography*, hemorrhage from puncture site, thrombosis of the vessel, and brachial plexus palsy (following axillary artery injection) have occurred. During *selective coronary arteriography* and *selective coronary arteriography with left ventriculography*, transient ECG changes (most patients); transient arrhythmias (infrequent); ventricular fibrillation (from manipulation of catheter or administration of medium); hypotension; chest pain; myocardial infarction; transient elevation of creatinine phosphokinase (occurred in about 30% of patients tested); fatalities have been reported; hemorrhage, thrombosis, pseudoaneurysms at puncture site, dislodgment of arteriosclerotic plaques, dissection of coronary vessels, and transient sinus arrest have occurred due to the procedure.

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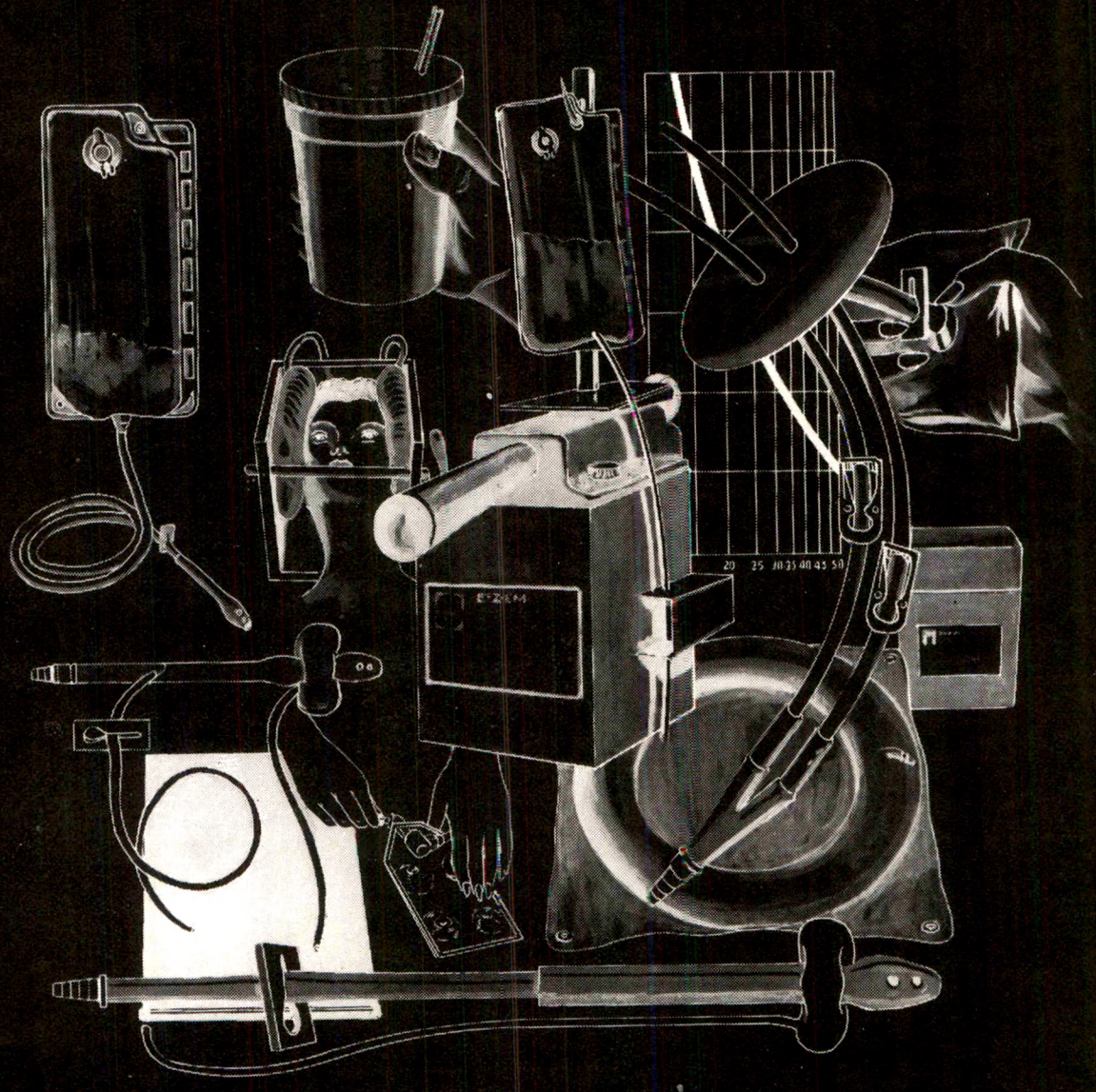

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VOL. 125 DECEMBER, 1975 No. 4

TRACHEAL AGENESIS*

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NEW HAVEN, CONNECTICUT; NEW YORK, NEW YORK; BUFFALO, NEW YORK; AND KANSAS CITY, MISSOURI

ABSTRACT:

Tracheal agenesis is a rare anomaly that produces neonatal respiratory distress and has been uniformly lethal to date. In this malformation the entire trachea is usually absent, air reaching the bronchi through a communication with the esophagus, and the lungs are normally formed. The diagnosis should be suspected in any infant in whom improved ventilation is obtained despite difficult intubation and abnormal tracheal tube placement.

Coexistent cardiac, gastrointestinal, and genitourinary anomalies are common.

The growth of the tracheoesophageal septum and the elongation of the dividing foregut are crucial phases in the morphogenesis of this malformation.

TRACHEAL agenesis is one of the rarest and most refractory causes of neonatal respiratory distress. In only one[11] of the 21 reported cases[1,3,4,7,9,11,12,19,21,24,26–30,33,38,42,46,48] did the infant survive the first week of life, and this child succumbed six weeks after an ambitious corrective procedure.

The clinical features and morphological findings reported in cases of tracheal agenesis are strikingly uniform. In most, the entire trachea is absent, there is esophagobronchial fistulous communication, and the lungs are normally formed. Many have had a wide spectrum of associated congenital anomalies.

The purpose of this paper is: (1) to present five additional cases of tracheal agenesis gathered from four medical centers; (2) to review the reported cases with emphasis on clinical similarities, roentgenologic aspects, and associated anomalies; and (3) to review the embryology of tracheal agenesis and related malformations.

REPORT OF CASES

CASE 1. (T.B.; Y.N.H.H.—No. 770292.) This 2,050 gram male infant was born to a multiparous mother after a 36 week gestation complicated by hydramnios. He developed respiratory distress immediately after birth, but improved when he was intubated.

* From the Departments of Radiology, the Yale-New Haven Hospital, New Haven, Connecticut; The College of Physicians and Surgeons, Columbia University, New York, New York; The Buffalo Children's Hospital, Buffalo, New York; and The Children's Mercy Hospital, Kansas City, Missouri.

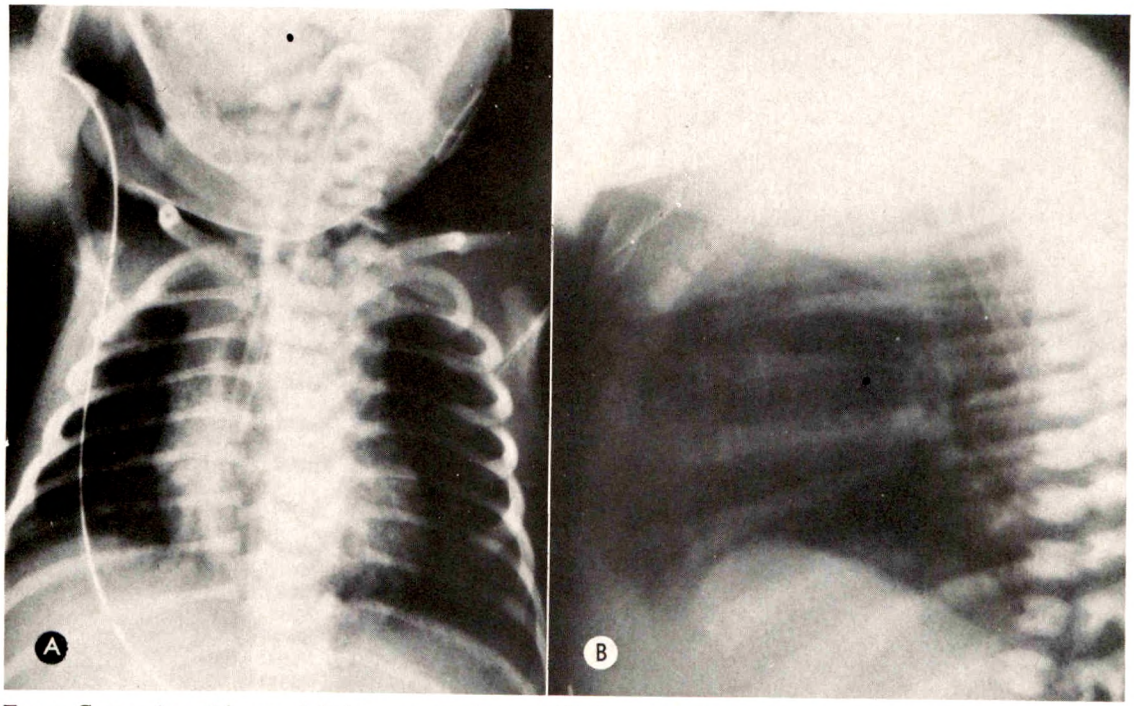

FIG. 1. Case 1. Age 48 hours. (*A*) Anteroposterior and (*B*) lateral chest roentgenograms show the endotracheal tube posteriorly positioned in the air distended esophagus. Adequate ventilation was maintained with the tube in this position. The lungs are well aerated and clear.

Extubation at 48 hours was followed by apnea and then a difficult reintubation. Roentgenograms showed the endotracheal tube in an abnormally posterior position (Fig. 1, *A* and *B*), but each time it was removed the infant became apneic and each time it was replaced it was again in abnormal position. Because tracheal agenesis was suspected, contrast medium was injected into the esophagus through a feeding tube (Fig. 2). This demonstrated a communication between the esophagus and bronchial tree at the level of the bifurcation of the main bronchi. Fluoroscopically, the esophagus would distend markedly with each inspiration and collapse with expiration (Fig. 3, *A* and *B*). Attempts to maintain aeration by the esophageal airway were unsuccessful and the infant died at 59 hours of age.

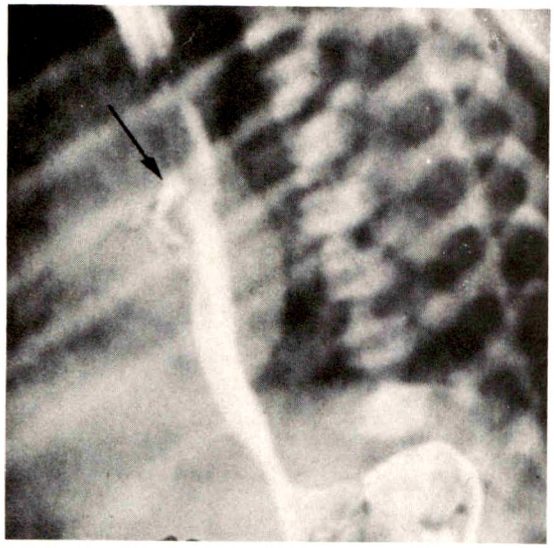

FIG. 2. Case 1. Age 50 hours. A small amount of contrast material was instilled in the esophagus through an infant feeding tube and outlines a communication from the anterior aspect of the esophagus to the proximal bronchi near the carina (arrow).

Autopsy disclosed a normal larynx. The trachea ended blindly just below the cricoid cartilage. No tracheal vestige was noted between this blind pouch and the site of fusion of the main bronchi at the carina. A fistula one millimeter in diameter connected the carina with the esophagus. The lungs, although normally developed, showed evidence of aspiration pneumonia. A ventricular septal defect and a large patent ductus arteriosus were found, only one umblical artery was present, and the right kidney was dysplastic. There was agenesis of the right radius and thumb.

Comment. As in several other cases in the

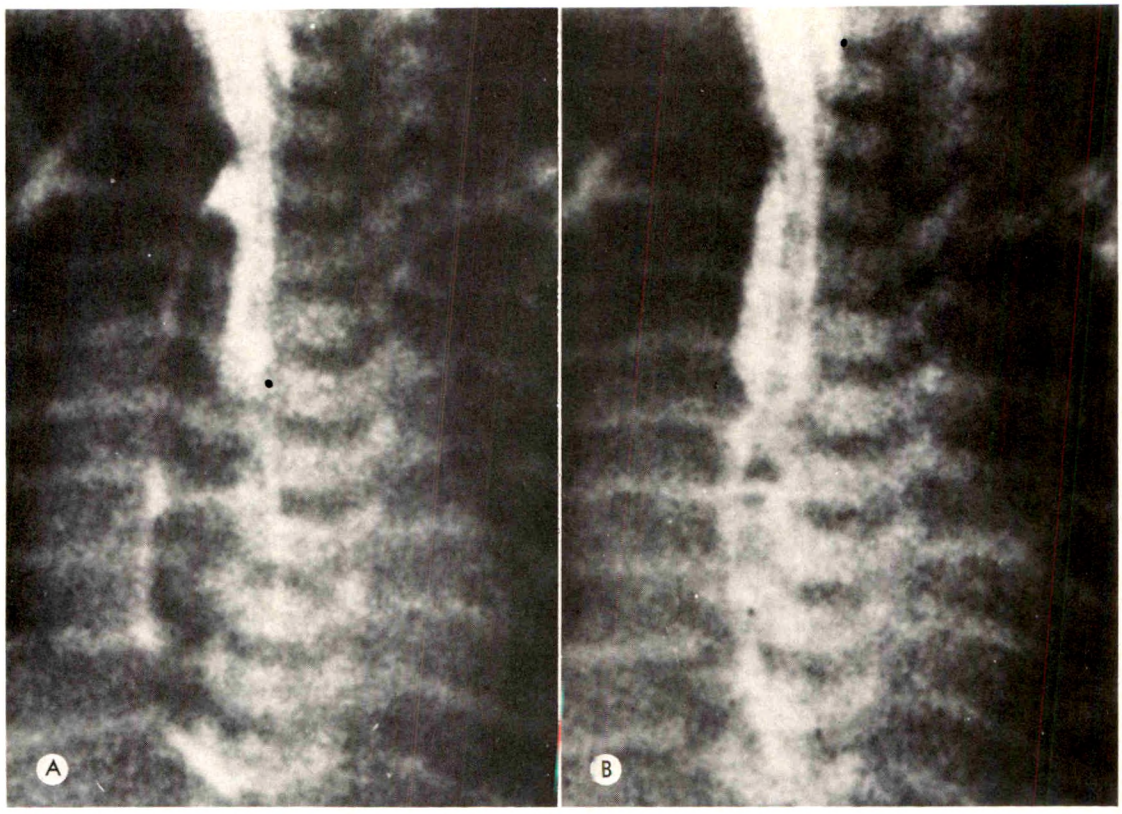

Fig. 3. Case I. At 50 hours of age. Two photographically enlarged frames from a cineroentgenographic recording made at the time of contrast material study show a wide variation in esophageal caliber between inspiration (*A*) and expiration (*B*). This "bellows" effect is a clue to the presence of a communication between the trachea and esophagus.

literature, the placement of a non-collapsible tube in the esophagus improved aeration. While this infant did not meet the strict diagnostic features of the Holt-Oram syndrome,[36] he belonged in a similar group with associated defects termed ventriculoradial dysplasia.[18]

Case II. (B.D.; Y.N.H.H.—No. 64672.) This 1,120 gram male infant was born to a 14 year old primiparous mother after an estimated 30 week gestation period. The mother had sickle cell anemia and the pregnancy was complicated by hydramnios.

At birth the infant was apneic. Despite what was felt to be a successful intubation, no ventilation was achieved. The infant failed to respond to several reintubations and died at 15 minutes of age. Postmortem roentgenograms (Fig. 4, *A* and *B*) showed air in the esophagus and upper gastrointestinal tract. The lungs were opaque but central air bronchograms were noted bilaterally.

The larynx was normal at postmortem examination and the trachea ended approximately one centimeter below the vocal cords. The lungs were individually attached to the mid-esophagus by the main bronchi (Fig. 5). The right lung was bilobed and the left was trilobed, but both were of normal size. Microscopically the lungs were immature and the alveoli were filled with proteinaceous material and squamous epithelial cells. A small membranous ventricular septal defect and a single umbilical artery were present.

Case III. (N.B.; Babies Hospital—No. 187-33-37.) This 2,200 gram female infant was born to a 24 year old gravida II, para I mother eleven hours after spontaneous rupture of the membranes. The gestation was 41 weeks by dates but the infant appeared to be premature by physical examination. The Apgar score at one minute was one. A nasotracheal tube was inserted with difficulty and produced some improvement in respiratory status. A catheter for suctioning of the nasotracheal tube could be

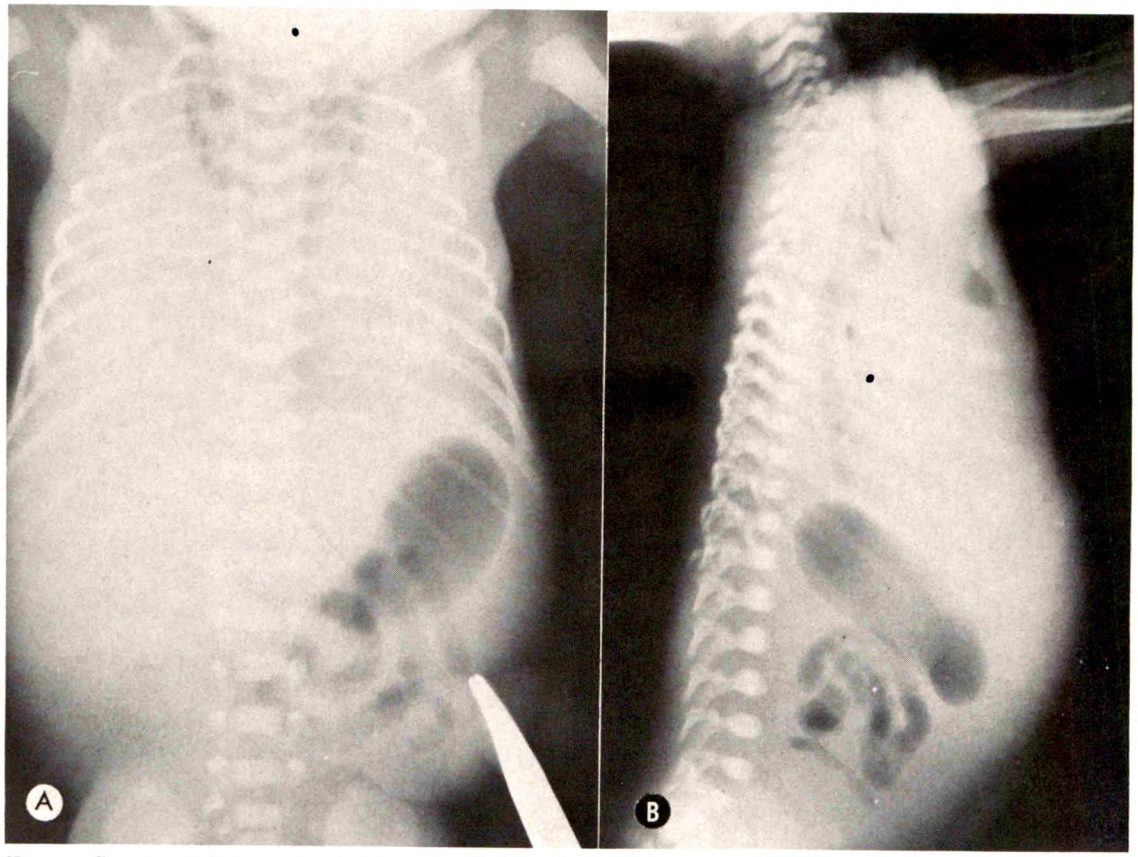

FIG. 4. Case 11. (*A*) Anteroposterior and (*B*) lateral roentgenograms obtained shortly after death. Air is present in the esophagus, stomach, proximal small bowel, and the larger airways. The subcutaneous and mediastinal air was secondary to the resuscitation attempts.

passed to 32 centimeters. A roentgenogram (Fig. 6, *A* and *B*) at that time showed the tip of the suction catheter in the stomach. Adequate respirator ventilation was maintained without altering the position of the nasotracheal tube until 21 hours of age when, after a grand mal seizure and episodes of bradycardia, the infant died.

FIG. 5. Case 11. Photograph of autopsy specimen of the esophagus and lungs (from behind). The main bronchi attached directly to the esophagus. The lungs are airless, and the left lung (on left) has three lobes and the right has two.

At postmortem examination the larynx was small, and no remnant of the trachea was identified either grossly or microscopically from below the level of the cricoid cartilage to the carina. The two main bronchi fused to form a normal carina and tiny distal tracheal stump. A small tubular structure connected the carina and the esophagus (Fig. 7). The lungs were morphologically normal but there was extensive hemorrhage into the alveoli. A few squamous epithelial cells were present within the alveoli. A complex congenital heart lesion was present (atrial septal defect, ventricular septal defect, over-riding aorta, patent ductus arteriosus, anomalous left pulmonary venous drainage into an enlarged coronary sinus and sclerosis of the pulmonary valve). Single umbilical artery, ectopic pancreatic tissue in the jejunum, and multiple diaphragmatic eventra-

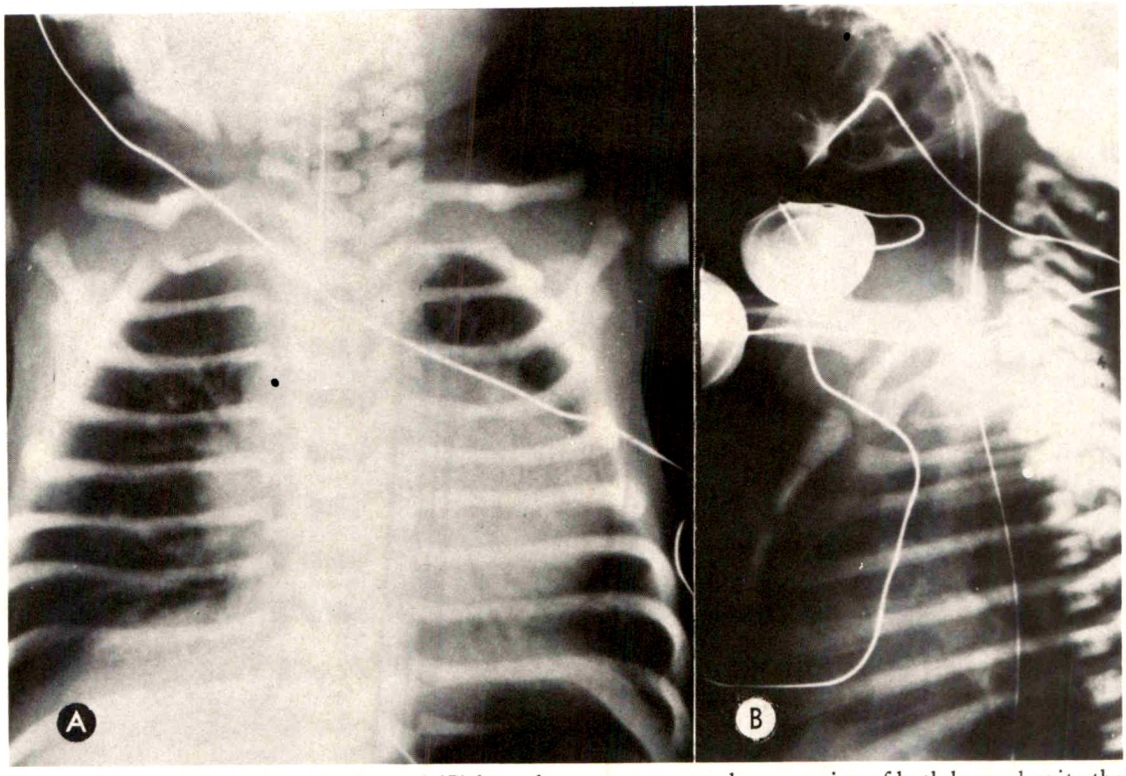

Fig. 6. Case III. (*A*) Anteroposterior and (*B*) lateral roentgenograms show aeration of both lungs despite the intraesophageal location of the nasotracheal tube. A suction catheter passed easily through this nasotracheal tube into the stomach. The lungs are well aerated and clear.

tions were also present.

Comment. Passage of a suction catheter into the stomach through the tube that was providing respiration suggested to the attending physicians that they were dealing with some form of tracheal atresia. Complex congenital heart defects have been noted in several of the cases of tracheal atresia reported to date.[1,9,21,28,29,48]

Case IV. (W.A.; Buffalo Children's Hospital —No. 272746.) This child was born in an outside hospital and obstetrical history is unavailable. Mild hydramnios was observed, and the full-term male infant was in immediate respiratory distress. Intubation relieved the distress, but it recurred when the tube was removed, and with each successive intubation it was thought that the tube had been removed from the esophagus and placed in the trachea. A chest roentgenogram (Fig. 8, *A* and *B*) after instillation of contrast medium into the esophagus showed the endotracheal tube to be in the esophagus, but no fistula to the tracheobronchial tree was demonstrated. Shortly after admission to the Buffalo Children's Hospital the infant died. At autopsy the larynx was normal but the trachea was absent to the carina (Fig. 9). The main bronchi joined in the midline and a fistula connected the esophagus to the posterior membranous portion of the carina. The lungs were firmly consolidated and intraalveolar and intralobular hemorrhages were present. The right upper and middle lobes were incompletely separated. There was a ventricular septal defect and the left atrium and ventricle were hypoplastic. Additional findings included complete cleft palate, moderate stenosis of the second portion of the duodenum, a Meckel's diverticulum, minor form anomalies of the liver and spleen, a bifid tenth left rib and an inguinal hernia.

Comment. Several other reported cases of tracheal agenesis have had obstructing lesions of the upper gastrointestinal tract.[1,29,48]

Case V. (B.C.; The Children's Mercy Hos-

FIG. 7. Case III. Photograph of autopsy specimen. The esophagus has been opened and the lungs removed. The arrow marks the esophageal orifice of the bronchial communication.

pital—No. A97149.) This 1,860 gram male infant was born to a primiparous mother after a 32 to 34 week gestation complicated by hydramnios. Labor and delivery were uncomplicated but the infant was limp, cyanotic, and without respirations until he was intubated and given oxygen by bag.

Roentgenograms showed extensive parenchymal density thought to represent hyaline membrane disease and an abnormal position of the endotracheal tube which appeared to be located in the esophagus. Because of copious oral and pharyngeal secretions, the diagnosis of esophageal atresia was considered but was excluded by successful passage of a nasogastric tube into the stomach. Removal of the endotracheal tube from the esophagus resulted in deep cyanosis and marked deterioration in the infant's clinical condition. Endoscopic examination showed normal pharynx and vocal cords but it was not possible to pass a tube more than a short distance beyond the cords. A small bronchoscope was passed into the esophagus and a connection to the respiratory tree was seen to extend as a single channel taking origin from the ventral wall of the esophagus at approximately the junction of its middle and lower third. The infant was unable to sustain respirations and expired at approximately 24 hours of age. Postmortem roentgenographic barium study demonstrated the blind upper airway immediately below the larynx and a communication between the esophagus and the carina (Fig. 10, *A* and *B*).

The larynx was normal at autopsy but the airway ended blindly immediately below it. The main bronchi joined in the midline and from that point communicated with the esophagus through a fistulous connection which was easily probed (Fig. 11). The lungs were otherwise anatomically normal but showed congestion, poor aeration, aspiration pneumonia, and barium within the airways. A subarachnoid hemorrhage was found and associated congenital cardiac abnormalities included mitral and aortic valvular atresia with aplasia of the left ventricle, and a large patent ductus arteriosus.

Comment. Again the endotracheal tube provided improved aeration despite its position in the esophagus. In this case the diagnosis was made with certainty before death by endoscopic studies, and this infant and our Case 1 represent the fourth and fifth times the esophagobronchial communication has been demonstrated antemortem.

DISCUSSION

Classification and Incidence. Most recent reports of tracheal agenesis have utilized a division into three types as proposed by Floyd *et al.*[9] (Fig. 12). In Type I there is agenesis of the upper trachea with an esophagotracheal communication to a normal distal trachea. Type II is agenesis of the entire trachea with normal main bronchi fused in the midline at the carina. This is the most common form and all but two[30,33] have had an associated communication from the esophagus to the fused bronchi. Complete tracheal agenesis with both of the bronchi connecting independently to the

esophagus constitutes Type III. In all types the lungs are developmentally normal or show only minor form anomalies.

Although all other reviews have included the case of Potter and Bohlender,[35] this actually represents a severe short segmental atresia with a malformed cricoid cartilage. Therefore, this case and a recent similar one[34] are not included in our review, but they are discussed as related anomalies. Technically, the two reported cases of total tracheopulmonary agenesis reported to date[8,31] qualify as cases of tracheal agenesis. However, they are also excluded in this review since they clearly represent an earlier developmental derangement.

Our report of five cases of tracheal agenesis brings the total number to 26 (Table I). Three infants have had Type I, seventeen have had Type II (15 with a communication to the esophagus and two without) and six have had Type III.

Clinical Findings. Information concerning the mother's pregnancy was available in 19 of the 26 cases. In seven cases the gestation was uncomplicated. Hydramnios was present in nine cases and is the most frequent complication. Four of these nine had associated anomalies of the upper gastrointestinal tract which may produce hydramnios (Table I). Two of the pregnancies were complicated by urinary tract infection (Joshi's case[21] and our Case III) and one by antepartum hemorrhage.[26] Sixteen of the case reports specified birth weight, gestational age, or both: eleven infants weighed less than 2,500 grams and only five were full term and of normal birth weight. Fourteen of the 20 cases in which the infant's sex was reported were male.

Information regarding resuscitation attempts was available in most of the cases. All of the infants were born alive although several showed no spontaneous respirations. Intubation attempts failed in nine cases, and in four cases tracheostomy was also unsuccessful. Seven cases, including four of our five, improved with the placement of an endotracheal tube in the

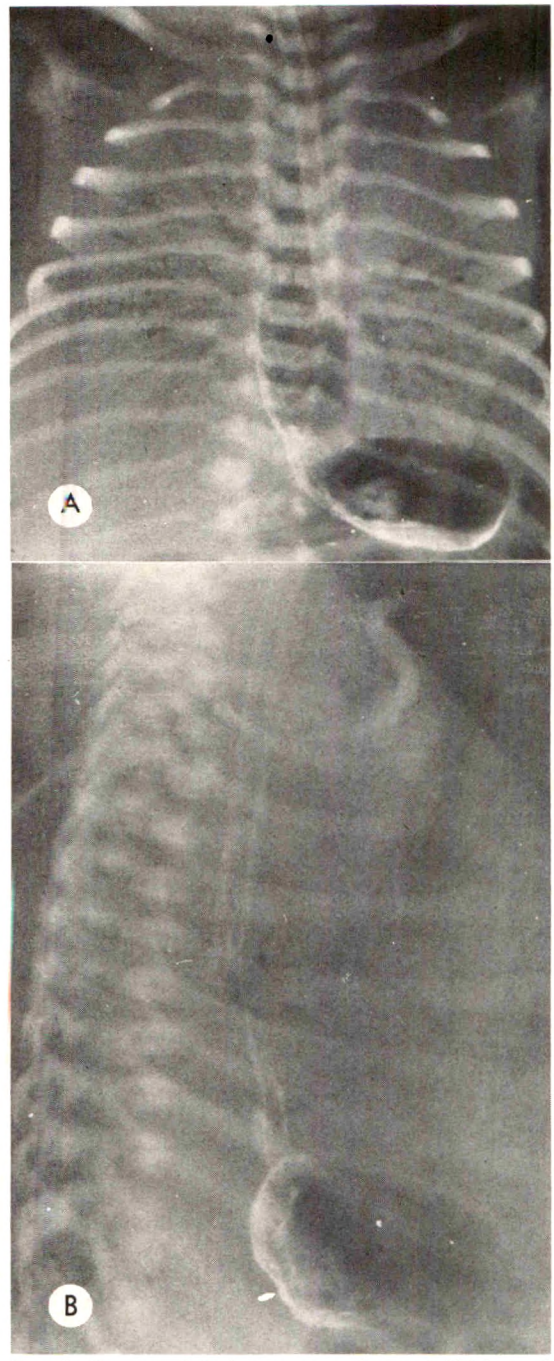

Fig. 8. Case IV. (*A*) Anteroposterior and (*B*) lateral chest roentgenograms obtained after contrast medium was instilled in the esophagus. This confirms the intraesophageal location of the endotracheal tube but no communication with the tracheobronchial tree was demonstrated by roentgenography. Air is present only in the larger airways. Note the degree of distention of the distal esophagus in (*A*).

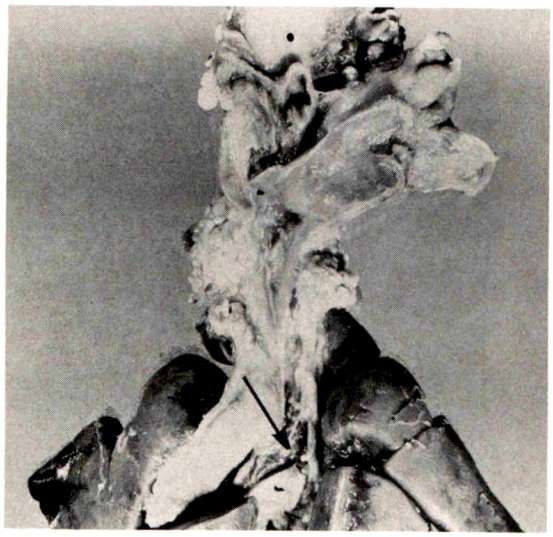

FIG. 9. Case IV. Photograph of autopsy specimen. The epiglottis is shown near the top. The upper esophagus has been placed to the right and the trachea is absent. The lungs (below) attach to the esophagus *via* a small communication (arrow).

esophagus. In each, extubation rapidly resulted in cyanosis and replacement of the tube improved the infant's condition. The rigid tube appears to provide a splint for the collapsible esophagus. Only three infants survived longer than 24 hours, and none lived longer than six weeks. Two of these[1,11] had surgical procedures aimed at supporting the upper esophagus and isolating it from gastrointestinal secretions.

Roentgenographic Findings. Roentgenographic examinations have been mentioned in only three previous case reports.[1,9,11] In the case of Floyd *et al.*[9] contrast material was utilized to demonstrate the blind tracheal sac and an esophagopulmonary fistula. Fonkalsrud *et al.*[11] instilled contrast material in the tracheostomy tube of their infant, demonstrated the intraesophageal position of the tube, and outlined a fistulous connection to a common bronchus. In the case of Altman *et al.*[1] a chest roentgenogram shortly after birth was normal. On the third day of life, contrast material was instilled into the pharyngeal airway and demonstrated an esophagobronchial communication.

In four of our five cases the tracheal tube was thought to be within the esophagus but removal led to increased respiratory distress which necessitated replacement. Marked posterior displacement of the tube was noted on the plain lateral roentgenogram in Case I. The intraesophageal location of the tube in Cases III and IV was proved with the passage of a suction catheter and the instillation of contrast material, and in Case V the anomaly was demonstrated by esophagoscopy. The suggestion of intraesophageal location of the nasotracheal or endotracheal tube in an infant whose respiration improves with such intubation should suggest the diagnosis of tracheal agenesis with an esophagobronchial communication. Case I and Case IV showed a dramatic fluctuation in esophageal caliber during ventilation, a clue to the presence of a communication between the esophagus and the airways. Case I is the fourth and Case V is the fifth time in which an esophagobronchial communication was demonstrated while the infant was still alive. Positive contrast material failed to show the fistula in Case IV, but the lungs were airless and the communication may have been occluded.

Associated Anomalies and Pathology. Seventeen of 22 cases with complete reports had other anomalies in addition to tracheal agenesis (Table I), and in several cases severe multi-system anomalies were present. In 14 infants with cardiac abnormalities, incomplete septum formation was the most common finding, but several had complex defects. Six of the eight infants with malformations of the gastrointestinal tract had incomplete development of the foregut or midgut. Five infants had major malformations of the urinary tract and three showed genital anomalies. Limb anomalies were present in three cases and one had hemivertebrae. Witzleben's[48] infant had hydrocephalus and microphthalmia and represented the only case with neurological defects.

The state of the lungs at autopsy was documented in 19 cases. Incomplete lobulation was present in two cases[29,48] and

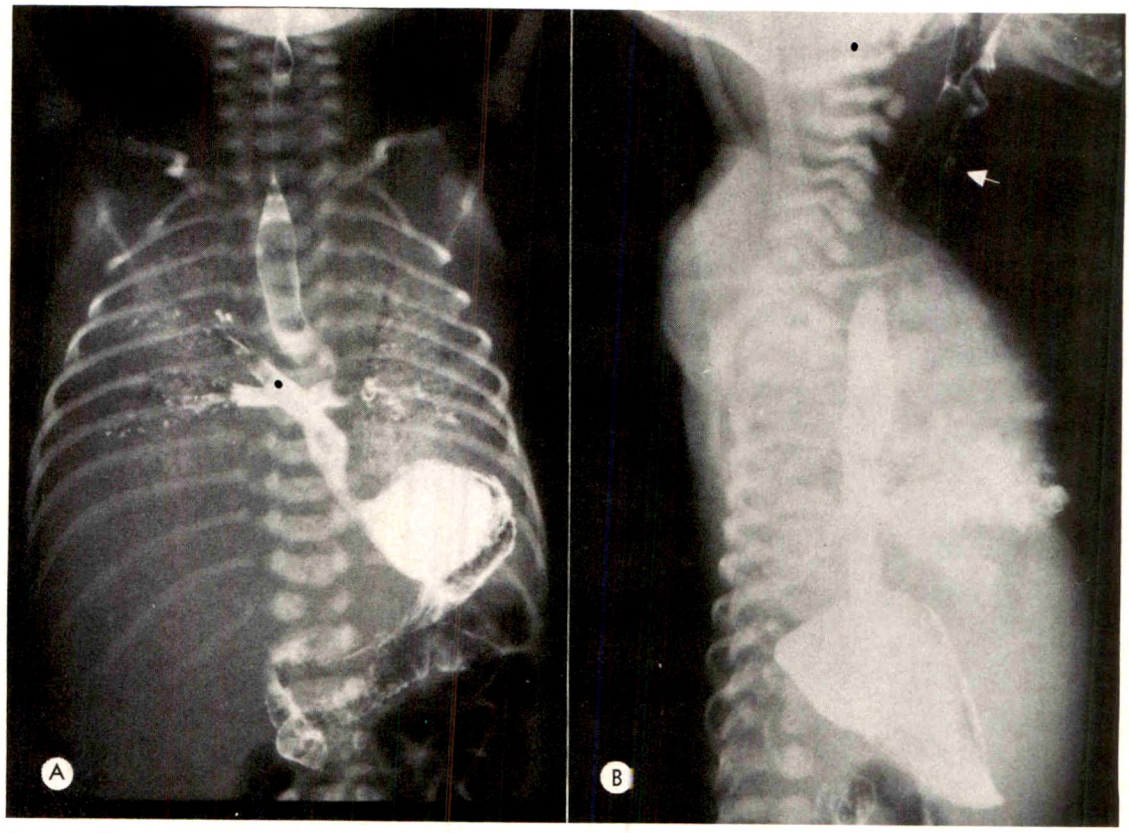

Fig. 10. Case v. (*A*) Anteroposterior and (*B*) lateral chest roentgenograms after postmortem instillation of barium showing the blind upper airway (arrow on lateral film) and filling of the bronchi from the esophagus.

our Case iv, and our second case had bilobed right and trilobed left lungs. Three cases including our Cases iii and iv showed evidence of intra-alveolar hemorrhage.[7] Vascular congestion was noted in three cases[7,24] including our Case v; and interlobular septal edema was present in one case.[30] Those infants surviving the immediate postnatal period showed evidence of aspiration or hemorrhagic pneumonia[1,9,11] (Cases i and v). Four cases[38,48] including our Cases ii and iii showed squamous epithelial cells within the alveoli, attesting to patency of the communications *in utero*.

Embryology. To understand the numerous theories concerning the etiology of tracheal agenesis and related malformations, a knowledge of the formation of the trachea is necessary. The respiratory system arises as a midline ventral diverticulum from the proximal foregut during the 21–24 day stage of development,[40,43,44] and this is associated with formation of a groove on the internal surface of the foregut (Fig. 13*A*). During the next ten days, rapid longitudinal growth of the foregut and the laryngotracheal groove takes place. At approximately 25–27 days, development of the tracheoesophageal septum begins (Fig. 13*B*). Lateral ridges develop separating the tracheal groove from the remaining foregut, and these eventually meet in the midline (Fig. 13*C*). This process of division of the respiratory and digestive system begins caudally and extends cranially and is almost complete by the 32–34 day stage.[40] Laryngeal differentiation begins as the tracheoesophageal septum nears completion.

Embryologic discussions in the cases of tracheal agenesis reported to date have relied heavily upon existing theories pro-

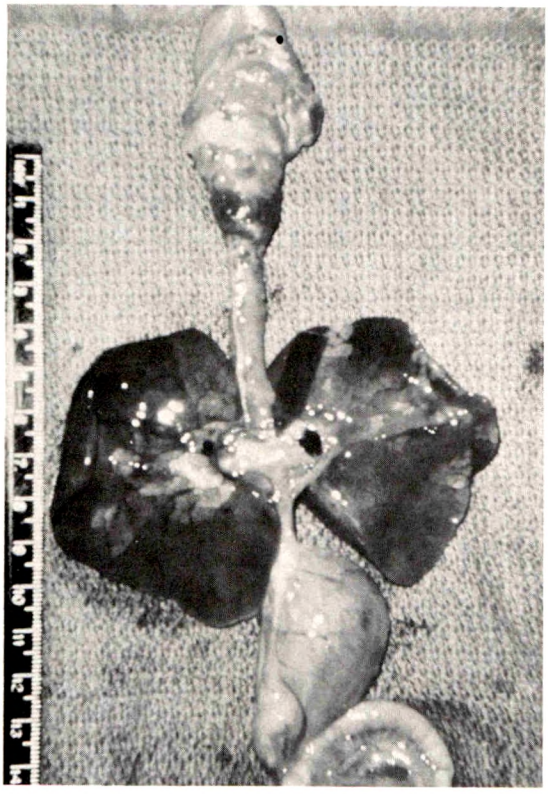

FIG. 11. Case v. Photograph of the autopsy specimen showing the esophagus with the larynx above and the stomach below. The trachea is absent and the two main bronchi connect with the esophagus by a fistula from the carina.

posed to explain esophageal atresia and tracheoesophageal fistula. Epithelial overgrowth with resultant occlusion and subsequent recanalization has been a popular explanation for atresias and stenoses of tubular structures. However, complete epithelial occlusion of the esophagus does not occur normally in humans,[13] and it would not, of course, account for the presence of a fistula to the trachea. The trachea does not undergo an epithelial occlusion stage.

Fluss and Poppen[10] reported three cases of tracheoesophageal fistula and esophageal atresia that were associated with anomalies of the arterial system and speculated that the vessels which crossed behind the esophagus at the site of the defects had exerted external pressure and had produced the defects. These anomalous vessels probably represented associated rather than causal anomalies since this is an inconsistent association.

Keith and Spicer,[23] Rosenthal,[37] Gruenwald,[15] Smith,[40] and Bremer[6] have all proposed abnormalities in the formation of the tracheoesophageal septum, or the rapid growth and elongation of the foregut, or both, as instrumental in producing esophageal atresia and tracheoesophageal fistula. Rosenthal,[37] reviewing eight cases of esophageal atresia with tracheoesophageal fistula, emphasized the failure of the endodermal lateral ridges to completely separate the trachea from the esophagus. Gruenwald[15] stressed that a comprehensive theory must explain not only the fistula (as Rosenthal's theory had done) but also the atresia of the esophagus. He postulated an interaction of two relatively independent processes, division of the foregut into trachea and esophagus and the rapid elongation of those structures, as fundamental in understanding this complex anomaly. If separation of the trachea and esophagus does not precede a phase of rapid growth, the esophagus might be "stretched out" by the faster growing laryngotracheal portion and esophageal atresia would result. Smith,[40] utilizing Streeter's work,[43,44] supported Gruenwald's theory and proposed that an imbalance of these two independent phenomena results in a spectrum of anomalies ranging from isolated esophageal atresia to isolated tracheoesophageal fistula. Devenis and Otis[7] have postulated that tracheal agenesis may result from a similar stretching of the trachea, but this seems to be an oversimplification since the tracheal portion of the common foregut develops at a faster rate than the esophageal portion.[13]

Bremer[6] developed a comprehensive theory that perhaps best explains tracheal atresia and its relation to tracheoesophageal fistula. He based his theory on deviation of the tracheoesophageal septum from its normal position. Dorsal and ventral displacement of the folds would result in esophageal or tracheal atresia, respectively,

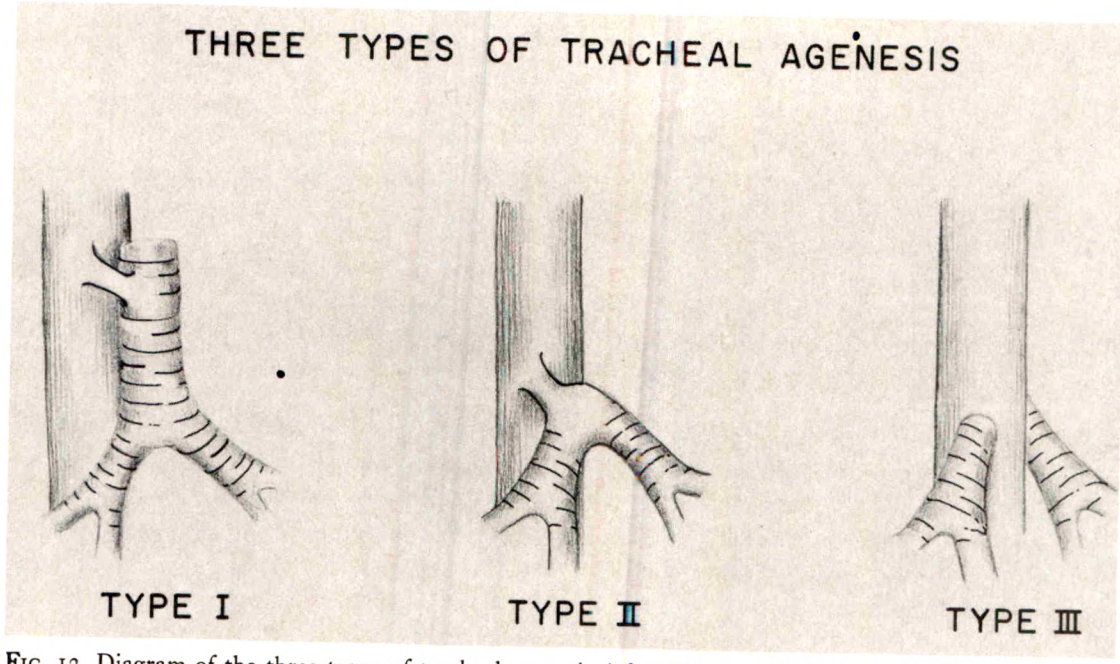

FIG. 12. Diagram of the three types of tracheal agenesis (after Floyd *et al.*[9]). Type II, with absence of the trachea to the level of the carina, is the most common.

and oblique development of the folds might result in rarer forms of anomalous bronchi such as one from the trachea and one from the esophagus. Early ventral deviation of the lateral folds followed by lengthening of the foregut would result in bronchi and lungs appearing to develop from the distal esophagus as in our cases.

According to Spencer,[41] cases of "total" sequestration of the lung represent a closely related anomaly. In eight cases reported to date,[2,16,17,22,25,32,45,47] the right lung was supplied solely by a bronchus arising from the esophagus, and the left lung arose from a normal trachea. As noted above, Bremer would attribute such a malformation to oblique development of the tracheoesophageal septum. The relationship of this rare anomaly to the more common forms of bronchopulmonary sequestration is unclear. Halasz *et al.*[16] reported one case of such "total" sequestration and postulated that intralobar sequestration, extralobar sequestration, esophagobronchial fistulas, bronchogenic cysts, and esophageal cysts all represent a spectrum of anomalies of the primitive foregut.

Several related tracheoesophageal septal anomalies deserve mention. Potter and Bohlender[35] reported a case with stenosis of the subglottic portion of the trachea and malformation of the cricoid cartilage. Similar degrees of subglottic stenosis were noted by Sayre and Hall[39] in two infants with tracheoesophageal fistula. Peison *et al.*[34] recently reported a case in which there was a fistula from the trachea to the esophagus 0.3 cm above the tracheal bifurcation. Above this, the tracheal cartilages gradually enlarged and finally fused, and in the subglottic area the lumen was totally obliterated by connective tissue. This case is thus related to the subglottic stenosis cases as well as to the cases of Type I tracheal agenesis.

Another rare anomaly involving the tracheoesophageal septum is the laryngotracheoesophageal cleft.[5,14,20] This may vary in severity from a small cleft in the larynx to persistent esophagotrachea, which is an almost total union of the trachea and esophagus into a single tube. This anomaly is explained as an arrest in the cranial movement of the tracheoesophageal sep-

TABLE I

SUMMARY OF REPORTED CASES OF TRACHEAL AGENESIS AND THEIR ASSOCIATED ANOMALIES

Case No.	Authors	Type	Cardiovascular Anomaly	Other Anomaly
1	Payne[33]	II (s̄)	—	—
2	Beneke[3]	II	?	?
3	Walcher[46]	III	—	—
4	Fritz[12]	I	?	?
5	Benesova and Peter[4]	III	—	—
6	Marek[27]	II	—	Deformed thumb
7	Milles and Dorsey[30]	II (s̄)	Coarctation of aorta PDA	Bilateral undescended testes
8	Kessel and Smith[24]	II	—	—
9	Sandison[38]	I	—	Hypoplastic right kidney and ureter
10	Hempel[19]	II	?	?
11	Devenis and Otis[7]	II	—	Meckel's diverticulum
12	Stowens[42]	III	?	?
13	Floyd *et al.*[9]	III	PDA, ASD, VSD Tricuspid stenosis Bicuspid pulmonary valve Aberrant portal vein	Imperforate anus Single aplastic kidney
14	Fonkalsrud *et al.*[11]	II	VSD	Cystic right kidney
15	Witzleben[48]	II	Total anomalous pulmonary venous connection ASD Persistent left SVC	Gastric hypoplasia Duodenal atresia Absent gallbladder Meckel's diverticulum Bicornuate uterus Atrophic fourth digit Hydrocephalus Microphthalmia o.s.
16	McCracken *et al.*[28]	II	Cor biloculare Transposition of the great vessels	—
17	Lyons and Bruce[26]	I	Congenital heart lesion—not specified	Other anomalies—not specified
18	Lyons and Bruce[26]	II	—	—
19	Joshi[21]	II	Tetralogy of Fallot	Imperforate anus Bilateral atresia of lower ureters
20	McNie and Pryse-Davies[29]	III	Truncus arteriosus ASD, VSD	Annular pancreas occluding duodenum
21	Altman *et al.*[1]	II	Total anomalous pulmonary venous connection Atrioventricular canal	Duodenal web
	Present Report			
22	Case I	II	VSD, PDA	Dysplastic kidney Hypoplastic penis Undescended testis Absent right radius and thumb
23	Case II	III	VSD	Right lung bilobed Left lung trilobed

II (s̄) = complete tracheal atresia without esophagotracheal or esophagobronchial communication.
PDA = patent ductus arteriosus.
ASD = atrial septal defect.
VSD = ventricular septal defect.
SVC = superior vena cava.

Table I (*Continued*)

Case No.	Authors	Type	Cardiovascular Anomaly	Other Anomaly
24	Case III	II	Complex	Ectopic pancreatic tissue in jejunum Multiple diaphragmatic eventrations
25	Case IV	II	VSD Hypoplasia of left ventricle and left atrium	Cleft palate Duodenal stenosis Meckel's diverticulum Anomalies of liver and spleen Inguinal hernia Hemivertebra
26	Case V	II	PDA Hypoplastic (aplastic) left ventricle	—

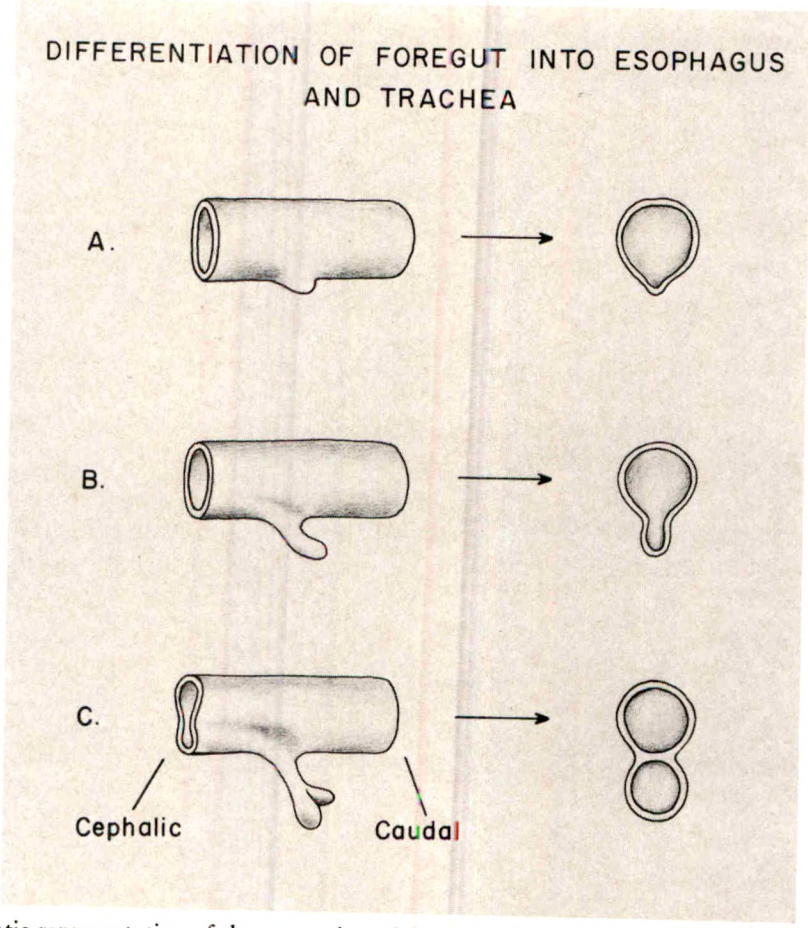

Fig. 13. Schematic representation of the separation of the trachea and esophagus in normal embryogenesis. (*A*) Development of the laryngotracheal groove on the ventral aspect of foregut; (*B*) evolution into a lung bud and beginning formation of the tracheoesophageal septum; (*C*) bifurcation of the single lung bud into main bronchi and partial construction of the tracheoesophageal septum.

tum.[6,13] If this arrest is early, then a persistent esophagotrachea results. If the arrest in cranial movement of the septum is late, then the dorsal fusion of the cricoid cartilage is prevented, resulting in a cleft larynx.

In the small number of cases of tracheal agenesis reported to date, no genetic pattern has been noted. McNie and Pryse-Davies'[29] case was a twin, and the other sibling was normal. The embryologic time course of several of the associated anomalies present in affected infants closely corresponds to that of the developing trachea. The fourth to sixth weeks are crucial phases in cardiac septal formation, distal foregut and midgut differentiation, renal development, and limb growth. The frequent presence of multisystem anomalies, most of which have similar developmental schedules, suggests a common timing of these malformations. The purely random association of these rare anomalies would be highly improbable.

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REFERENCES

1. ALTMAN, R. P., RANDOLPH, J. G., and SHEARIN, R. B. Tracheal agenesis: recognition and management. *J. Pediat. Surg.*, 1972, *7*, 112–118.
2. BATES, M. Total unilateral pulmonary sequestration. *Thorax*, 1968, *23*, 311–315.
3. BENEKE, R. Über Bauchlunge und hernia diaphragmatica spuria. *Verhandl. deutsch. Gesellsch. Pathol.*, 1905, *9*, 202–211.
4. BENESOVA, D., and PETER, R. Agenesie trachey. *Casop. lek. cesk.*, 1934, *73*, 356–358.
5. BLUMBERG, J. B., STEVENSON, J. K., LEMIRE, R. J., and BOYDEN, E. A. Laryngotracheoesophageal cleft, embryologic implications: review of literature. *Surgery*, 1965, *57*, 559–566.
6. BREMER, J. L. Congenital Anomalies of the Viscera: Their Embryological Basis. Harvard University Press, Cambridge, 1957, pp. 27–31.
7. DEVENIS, A. M., and OTIS, R. D. Tracheal atresia associated with tracheoesophageal fistula. *J. Thoracic Surg.*, 1957, *34*, 405–408.
8. DEVI, B., and MORE, J. R. S. Total tracheopulmonary agenesis associated with asplenia, agenesis of umbilical artery and other anomalies. *Acta paediat. scandinav.*, 1966, *55*, 107–116.
9. FLOYD, J., CAMPBELL, D. C., and DOMINY, D. E. Agenesis of trachea. *Am. Rev. Resp. Dis.*, 1962 *86*, 557–560.
10. FLUSS, Z., and POPPEN, K. J. Embryogenesis of tracheoesophageal fistula and esophageal atresia. *Arch. Path.*, 1951, *52*, 168–181.
11. FONKALSRUD, E. W., MARTELLE, R. R., and MALONEY, J. V. Surgical treatment of tracheal agenesis. *J. Thoracic & Cardiovasc. Surg.*, 1963, *45*, 520–525.
12. FRITZ, E. Eine seltene Missbildung der oberen Luftwege. *Virchows Arch. path. Anat.*, 1933, *289*, 264–269.
13. GRAY, S. W., and SKANDALAKIS, J. E. Embryology for Surgeons. W. B. Saunders Company, Philadelphia, 1972, pp. 65, 290.
14. GRISCOM, N. T. Persistent esophagotrachea: most severe degree of laryngotracheo-esophageal cleft. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *97*, 211–215.
15. GRUENWALD, P. Case of atresia of esophagus combined with tracheo-esophageal fistula in 9 mm. human embryo, and its embryological explanation. *Anat. Record*, 1940, *78*, 293–302.
16. HALASZ, N. A., LINDSKOG, G. E., and LIEBOW, A. A. Esophagobronchial fistula and bronchopulmonary sequestration. *Ann. Surg.*, 1962, *155*, 215–220.
17. HANNA, E. A. Broncho-esophageal fistula with total sequestration of right lung. *Ann. Surg.*, 1964, *159*, 599–603.
18. HARRIS, L. C., and OSBORNE, W. P. Congenital absence or hypoplasia of radius with ventricular septal defect: ventriculoradial dysplasia. *J. Peditat.*, 1966, *68*, 265–272.
19. HEMPEL, K. J. Beitrag zur kongenitalen Atresie des Larynx und Aplasie der Trachea. *Zentralbl. f. Alg. Path. u Path. Anat.*, 1956, *95*, 226–237.
20. IMBRIE, J. D., and DOYLE, P. J. Laryngotracheoesophageal cleft. *Laryngoscope*, 1969, *79*, 1252–1274.
21. JOSHI, V. V. Tracheal agenesis. *Am. J. Dis. Child.*, 1969, *117*, 341–343.
22. KEELEY, J. L., and SCHAIRER, A. E. Anomalous origin of right main bronchus from esophagus. *Ann. Surg.*, 1960, *152*, 871–874.
23. KEITH, A., and SPICER, J. E. Three cases of malformation of tracheo-esophageal septum. *J. Anat. & Physiol.*, 1907, *41*, 52–55.
24. KESSEL, I., and SMITH, J. N. Congenital absence of trachea. *Thorax*, 1953, *8*, 266–268.
25. LEWIS, J. E., and MURRAY, R. E. Pulmonary sequestration with bronchoesophageal fistula. *J. Pediat. Surg.*, 1968, *3*, 575–579.
26. LYONS, S. M., and BRUCE, A. E. G. Tracheal

agenesis: report of two cases. *Anaesthesia*, 1968, *23*, 98–102.

27. Marek, J. J. Congenital deformity of trachea. *Ohio M. J.*, 1940, *36*, 1308.
28. McCracken, A. W., Flanagan, N., and Flanagan, P. Tracheal atresia associated with cor biloculare. *Thorax*, 1964, *19*, 530–532.
29. McNie, D. J. M., and Pryse-Davies, J. Tracheal agenesis. *Arch. Dis. Childhood*, 1970, *45*, 143–144.
30. Milles, G., and Dorsey, D. B. Intra-uterine respiration-like movements in relation to development of fetal vascular system: discussion of intra-uterine physiology based upon cases of congenital absence of trachea, abnormal vascular development, and other anomalies. *Am. J. Path.*, 1950, *26*, 411–425.
31. Morison, J. E. Congenital malformations in one of monozygotic twins. *Arch. Dis. Childhood*, 1949, *24*, 214–218.
32. Nikaidoh, H., and Swenson, O. Ectopic origin of right main bronchus from esophagus. *J. Thoracic & Cardiovasc. Surg.*, 1971, *62*, 151–160.
33. Payne, W. A. Congenital absence of trachea, specimen (illustrated). *Brooklyn M. J.*, 1900, *14*, 568.
34. Peison, B., Levitzky, E., and Sprowls, J. J. Tracheoesophageal fistula associated with tracheal atresia and malformation of larynx. *J. Pediat. Surg.*, 1970, *5*, 464–467.
35. Potter, E. L., and Bohlender, G. P. Intra-uterine respiration in relation to development of fetal lung: with report of two unusual anomalies of respiratory system. *Am. J. Obst. & Gynec.*, 1941, *42*, 14–22.
36. Poznanski, A. K., Stern, A. M., and Gall, J. C. Skeletal anomalies in genetically determined congenital heart disease. *Rad. Clin. North America*, 1971, *9*, 435–458.
37. Rosenthal, A. H. Congenital atresia of esophagus with tracheo-esophageal fistula. *Arch. Path.*, 1931, *12*, 756–772.
38. Sandison, A. T. Partial absence of trachea with live birth. *Arch. Dis. Childhood*, 1955, *30*, 475–477.
39. Sayre, J. W., and Hall, E. G. Anomalies of larynx associated with tracheo-esophageal fistula. *Pediatrics*, 1954, *13*, 150–155.
40. Smith, E. I. Early development of trachea and esophagus in relation to atresia of esophagus and tracheo-esophageal fistula. *Contrib. Embryol. Carnegie Inst.*, 1957, *36*, 41–57.
41. Spencer, H. Pathology of the Lung. Second edition. Permagon Press, Oxford, 1968, p. 76.
42. Stowens, D. Pediatric Pathology. Williams & Wilkins Company, Baltimore, 1961, p. 379.
43. Streeter, G. L. Developmental horizons in human embryos: description of age group XI, 13 to 20 somites, and age group XII, 21 to 29 somites. *Contrib. Embryol. Carnegie Inst.*, 1942, *30*, 211–245.
44. Streeter, G. L. Developmental horizons in human embryos: description of age group XIII, embryos about 4 to 5 millimeters long, and age group XIV, period of indentation of lens vesicle. *Contrib. Embryol. Carnegie Inst.*, 1945, *31*, 27–63.
45. Thompson, N. B., and Aquino, T. Anomalous origin of right main-stem bronchus. *Surgery*, 1962, *51*, 668–676.
46. Walcher. Angeborener Mangel der Trachea. *Deutsche Ztschr. ges. gerichtl. Med.*, 1928, *12*, 292–296.
47. Warner, F. S. McGraw, C. T., Peterson, H. G., Cleland, R. S., and Meyer, B. W. Lung ectopia and agenesis with heart dextrorotation. *Am. J. Dis. Child.*, 1961, *101*, 514–518.
48. Witzleben, C. L. Aplasia of trachea. *Pediatrics*, 1963, *32*, 31–35.

ROENTGENOGRAPHIC FEATURES OF MUSHROOM (*Amanita*) POISONING*

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ABSTRACT:

The clinical and roentgenographic features of six cases of *Amanita* mushroom poisoning were reviewed. The roentgenographic manifestations included adynamic ileus (three patients) and small, irregularly shaped kidneys secondary to the healing process of acute tubular necrosis (one patient). Intestinal pseudo-obstruction can result from many medical problems and mushroom poisoning should be considered in its differential diagnosis.[6]

IT is well known that poisoning from mushrooms of the genus *Amanita* can produce complications of the kidney, liver, and central nervous system. Severe, adynamic ileus is usually not included in discussions of this uncommon and often fatal condition. A recent case of *Amanita phalloides* poisoning in which prolonged intestinal pseudo-obstruction occurred prompted a 15 year retrospective review of our medical records that disclosed five additional cases of *Amanita* poisoning. Two of the patients had severe intestinal ileus. The roentgenographic features of mushroom poisoning have not been described in the roentgenologic literature. We wish to present these features and to offer possible explanations for the ileus caused by poisoning from the *Amanita* mushroom.

REPORT OF CASES

CASE 1. An 87 year old Italian man ingested several white-capped wild mushrooms on December 15, 1974. Twenty-two hours later he developed severe nausea, vomiting, abdominal cramps, and profuse diarrhea. He was admitted to a local hospital on December 16, 1974. Physical findings were unremarkable. Laboratory studies showed blood urea nitrogen to be 28 mg/dl, creatinine 3.0 mg/dl, sodium 137 mEq/L, potassium 6.6 mEq/L, chloride 103 mEq/L, and serum glutamic oxaloacetic transaminase 160 IU/L. The patient became oliguric and was transferred on December 17, 1974, to our hospital for thioctic acid therapy.

On arrival, the patient was alert but vomiting blood-tinged material. The physical findings were still normal. A laboratory profile showed blood urea nitrogen to be 43 mg/dl, creatinine 4.4 mg/dl, serum glutamic oxaloacetic transaminase 1,440 IU/L, total bilirubin 1.6 mg/dl, and hematocrit 49.5 percent. Nasogastric aspirate and stool specimens were guaiac-positive. Roentgenograms of the chest and supine and upright views of the abdomen demonstrated elevated diaphragm leaves and marked gaseous distention of the stomach, small bowel, and entire colon. Several, small, air-fluid levels were present within the colon, but there was no free air nor obvious ascites (Fig. 1). The patient was treated with intravenous fluids, thioctic acid, and neomycin. Thin-layer chromatography performed on a stool specimen disclosed high levels of *Amanita* toxin and confirmed the clinical impression of *Amanita phalloides* poisoning. Gradually, over the next six days, there was considerable improvement in the renal function and repair of hepatocellular damage. The serum liver enzyme values became normal, the blood urea nitrogen fell to 29 mg/dl, and the creatinine fell to 2.1 ml/dl. The adynamic ileus disappeared along with the improvement in renal and hepatic conditions.

On December 22, 1974, the patient developed an enterococcal infection of the urinary tract, which was followed by a rise in the blood urea nitrogen to 36 mg/dl and creatinine to 6 mg/dl. An excretory urogram demonstrated poor function bilaterally but normal-sized kidneys and no distention of the pyelocalyceal systems.

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Two days later the patient's abdomen became distended, and roentgenograms of the abdomen disclosed recurrence of the adynamic ileus. This condition progressed and became severe by December 28. On the same date, the creatinine level rose to a level of 14.8 mg/dl, but the liver enzyme values remained normal. Hemodialysis, using a Scribner shunt, was instituted and performed intermittently for the next two weeks. During this time, the serum creatinine stabilized in the 7 to 8 mg/dl range and persisted at this level for the remainder of the hospital course. The adynamic ileus gradually resolved during the period of dialysis. The patient was discharged on the forty-third hospital day with mild residual renal failure. He was totally asymptomatic.

Case II. A 43 year old, Caucasian male alcoholic was in good health until November 13, 1962, when he ingested wild mushrooms. Five hours later, he developed severe nausea, vomiting, and diarrhea. He was admitted to a local hospital. Over the next three days he was treated with intravenous fluids. The gastrointestinal symptoms gradually abated but he became oliguric and, on November 16, 1962, was transferred to the University of California, San Francisco, for treatment of his renal failure.

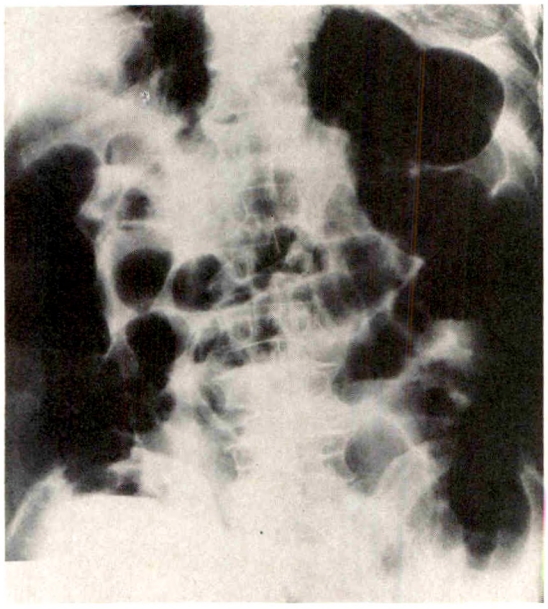

Fig. 1. Case I. Upright view of the abdomen. There is marked distention of the small bowel and colon by gas and several, short, air-fluid levels can be seen.

Physical examination disclosed signs of dehydration, abdominal distention, and hypoactive bowel sounds. At admission, the stool was guaiac-positive and laboratory studies showed sodium to be 112 mEq/L, potassium 6.2 mEq/L, bicarbonate 8.3 mEq/L, chloride 72 mEq/L, creatinine 13.6 mg/dl, and blood urea nitrogen 202 mg/dl. Liver function tests demonstrated 50 percent Bromsulphalein retention at 45 minutes, serum glutamic oxaloacetic transaminase 86 IU/L, prothrombin time 47 percent, and total bilirubin 2.2 mg/dl. Initial roentgenographic examination of the chest and abdomen demonstrated elevated diaphragm leaves and gaseous distention of multiple loops of the small bowel and colon but no evidence of free air or ascites. This pattern was compatible with severe adynamic ileus. It persisted throughout the hospital course. Peritoneal dialysis was begun on the day of admission and continued for seven days. On the fourth hospital day, the patient developed septicemia from *Escherichia coli* and *Pseudomonas*. Three days later, after becoming disoriented and then comatose, he died.

Autopsy disclosed marked dilatation of the small bowel. The mucosa of the stomach and small bowel was edematous and hyperemic. Histologic evaluation of the kidneys revealed acute tubular necrosis. There were also centrilobular necrosis of the liver, myocarditis, and several pulmonary abscesses.

Case III. A 43 year old American Indian woman was in good health until October 4, 1962, when she ingested several wild mushrooms. Three to four hours later she developed nausea, vomiting, and slight lacrimation. She was admitted to a local hospital and was given atropine to control her vomiting. During the next three days she became oliguric and, on October 8, 1962, she was transferred to the University of California San Francisco for treatment of renal failure.

At physical examination the patient was dyspneic and lethargic. Her blood pressure was 150/110 mm of Hg and pulse 88/min. There were generalized edema, diminished breath sounds at both lung bases, an abdominal fluid wave, and bilateral costovertebral angle tenderness. Initial laboratory values included serum glutamic oxaloacetic transaminase 21 IU/L, total bilirubin 0.3 mg/dl, sodium 108 mEq/L, potassium 5.6 mEq/L, chloride 78.5

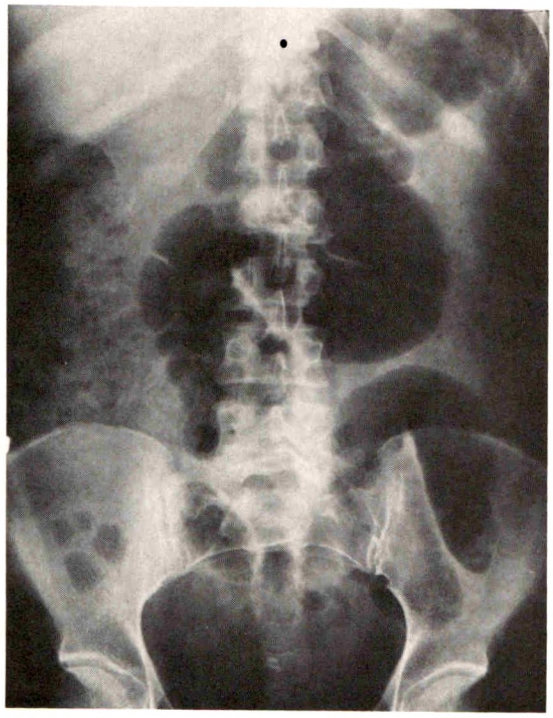

FIG. 2. Case III. Supine view of the abdomen. The transverse colon and several loops of small bowel in the lower abdomen are dilated.

mEq/L, bicarbonate 18.5 mEq/L, and blood urea nitrogen 94 mg/dl. Chest roentgenograms demonstrated bilateral lower lobe, discoid atelectasis. Supine and upright views of the abdomen disclosed dilated loops of small and large bowel (Fig. 2) with multiple, short, air-fluid levels in the colon. The patient was treated intermittently with peritoneal dialysis for acute tubular necrosis. Her hospital course was characterized by a slow, uncomplicated return of renal function. No additional abdominal roentgenograms were obtained, and physical examination during the hospitalization did not indicate persisting ileus. An excretory urogram performed on the thirty-fourth hospital day demonstrated poor renal function bilaterally but no evidence of obstruction. The renal contours were smooth with the right and left kidneys measuring 12.6 and 13.9 cm in length respectively (Fig. 3*A*). A left renal biopsy performed on the same day revealed necrosis of the proximal and distal convoluted tubules and diffuse interstitial fibrosis. The patient was discharged on the forty-eighth hospital day with mild, residual renal dysfunction. The creatinine was 4.9 mg/dl.

She was subsequently seen as an out-patient because of mild progressive renal failure secondary to the mushroom poisoning. A repeat excretory urogram in 1969 showed contraction of both kidneys, the right kidney measuring 10.7 cm and the left 11.9 cm in length. The renal outlines had become lobulated, but the pyelocalyceal systems were normal (Fig. 3*B*). The patient is currently asymptomatic and has persistent chemical evidence of mild renal failure.

No roentgenograms were available of Cases IV through VI. These cases are presented in Table 1. Two of these patients (Cases IV and V) had liver damage without renal impairment and, on physical examination, exhibited no evidence of an adynamic ileus. They had mild and uncomplicated clinical courses. Case VI is an example of severe mushroom poisoning causing

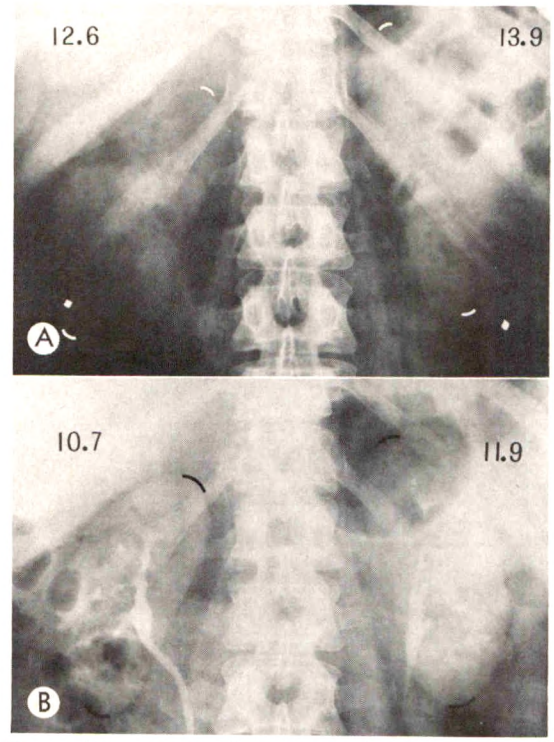

FIG. 3. Case III. (*A*) Five minute film from excretory urogram (1962). The pyelocalyceal systems are not seen because of acute tubular necrosis. The kidneys are of normal size, and the renal contours are smooth. (*B*) Five minute film (1969) demonstrates small and lobulated kidneys with normal pyelocalyceal systems.

TABLE I

PERTINENT FEATURES OF MUSHROOM (*AMANITA*) POISONING IN 6 PATIENTS

Case	Age, Sex	Presenting Symptoms	Days after Ingestion	SGOT (IU/L)	Creatinine (mg/dl)	Roentgenographic Findings	Clinical Course
I	87, M	Nausea; emesis; diarrhea; abdominal pain	1	160	3	Severe ileus (resolved on day 7)	Hepatocellular damage and renal failure with apparent resolution
			2	1440	4.4		
			4	2160	3.3		
			6	418	2.1		
			7	160	2.1		
			9	80	5.2	Recurrence of ileus	Recurrence of renal failure; hemodialysis
			14		14.8	Severe ileus with resolution on day 28	
			44		7.7	Poor renal opacification but normal kidney size and pyelocalyceal systems	Discharged 43rd hospital day with stable creatinine
II	43, M	Nausea; emesis; diarrhea; gastrointestinal bleeding	3	86	13.6	Severe adynamic ileus throughout hospitalization	Mild hepatocellular damage; renal failure; peritoneal dialysis complication—septicemia; died 7th hospital day
			10	>9.0			
III	43, F	Nausea; emesis	4	21	8.8	Moderate adynamic ileus; poor renal opacification; kidneys normal size and contour. Seven years later, small, irregular kidneys and normal pyelocalyceal systems	Acute tubular necrosis; peritoneal dialysis—no complications; discharged 43rd hospital day with mild renal failure
			38		7.0		
			47		4.6		
IV	32, M	Nausea; emesis; diarrhea	1	2580	1.9	None	Hepatic impairment without renal complications; no clinical evidence of ileus; discharged on 9th hospital day
			2	2790	1.2		
			11	40			
V	35, M	Nausea; emesis; diarrhea; abdominal pain; shock	1	260		None	Hepatic impairment without renal complications; no clinical evidence of ileus; discharged on 4th hospital day
			2	410			
			4	245	1.3		
VI	21 mo., M	Nausea; emesis; diarrhea; abdominal pain; gastrointestinal bleeding; central nervous system complication	1	750		None	Progressive hepatic and renal failure; died on day of admission; autopsy—focal gastric erosion, normal small and large bowel, fatty infiltration of the liver, acute tubular necrosis, and cerebral edema
			3		1.7		

rapid deterioration of renal, hepatic, and central nervous system function ending in death four days after ingestion of the mushrooms. This patient also showed no clinical evidence of an adynamic ileus, and autopsy revealed normal small and large bowel.

DISCUSSION

The clinical pattern of poisoning from the *Amanita* mushroom can be divided into three separate stages.[2] The first stage is characterized by a period without symptoms of six to 24 hours, followed by severe nausea, vomiting, abdominal cramps, and profuse watery diarrhea. Electrolyte disturbances occur, and without adequate fluid replacement the patient may succumb at this stage. In the second stage, which may last 48 hours, some patients appear to be recovering but often this is a false remission followed by the critical third stage. The third stage is characterized to varying degrees by acute renal tubular necrosis, hepatocellular damage, and signs of central nervous system and myocardial dysfunction. The patient usually dies four to eight days after the onset of symptoms; mortality rates vary between 45 percent and 90 percent.

The toxins responsible for the initial gastrointestinal symptoms and subsequent third stage abnormalities are known as amanitines.[5] They are cyclopeptides that act on the liver by inhibiting nucleolar ribonucleic acid polymerase. This leads to nucleolar and, later, nuclear and cytoplasmic break up.[5] In the kidney, the amanitine complex is filtered by the glomeruli and directly damages the convoluted tubules, producing acute tubular necrosis and, with healing, an interstitial fibrosis.[7]

The gastrointestinal symptoms of *Amanita* mushroom poisoning have been experimentally produced in dogs by intraperitoneal injection of amanitine.[3] Vomiting and diarrhea developed nine to eleven hours after injection, and all animals died within 20 to 24 hours. Histologic examination demonstrated a generalized hemorrhagic gastroenteritis.

In humans, histologic findings from autopsy material usually demonstrate gastrointestinal edema and congestion; hemorrhagic gastroenteritis; hyperplasia of intestinal lymphoid tissue and of deep abdominal lymph nodes and; rarely, small bowel dilatation.[1]

Severe adynamic ileus secondary to *Amanita* mushroom ingestion is considered rare.[2] It has also been reported secondary to poisoning from the *Galerina* mushroom, which is known to contain amanitine.[4] In this case, as in our patients, the ileus developed during the third clinical phase after initial gastrointestinal symptoms had abated.

The cause of the adynamic ileus is not clear, but several possibilities exist: (1) Although the mechanism is unknown, uremia and accompanying complex disturbances of electrolyte balance have been associated with intestinal pseudo-obstruction. This may be the explanation in mushroom poisoning. In our three cases and in the one in which it was secondary to *Galerina* mushroom ingestion, ileus developed with the onset of renal failure. In one patient (Case 1) the ileus resolved as renal function improved, only to reappear with the recurrence of uremia. In the other two patients, the ileus resolved during dialysis. (2) As Grossman and Malbin[4] suggested, the ileus may result from the initial, severe gastroenteritis. (3) The ileus can also be considered the result of a direct toxic effect on the bowel wall. As with other organ impairment, a critical period of time elapses before the visceral damage becomes apparent. The resolution of the ileus might also occur independent of other organ recovery.[4] In this regard, chromatography of the bowel may identify the amanitines.[4] (4) These mushrooms may contain additional substances which have anticholinergic properties. Other possible causes of adynamic ileus, such as hypokalemia and peritonitis, were not encountered in our patients.

In our series, the adynamic ileus was not confused clinically or roentgenographically with colonic obstruction. Although of concern, the ileus was overshadowed by the other critical medical problems. It was treated conservatively by use of intravenous fluids, nasogastric suction, and restriction of oral intake.

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We wish to acknowledge the helpful suggestions and materials provided by Dr. Theodore G. Tong and Dr. Charles E. Becker of San Francisco General Hospital.

REFERENCES

1. ABUL-HAJ, S. K., EWALD, R. A., and KAZYAK, L. Fatal mushroom poisoning: report of case confirmed by toxicologic analysis of tissue. *New England J. Med.*, 1963, *269*, 223–227.
2. ARMSTRONG, C. D. Mushroom poisoning: report of two fatal cases of poisoning by Amanita phalloides. *Stanford M. Bull.*, 1955, *13*, 111–116.
3. FIUME, L., DERENZINI, M., MARINOZZI, V., PETAZZI, F., and TESTONI, A. Pathogenesis of

gastro-intestinal symptomatology during poisoning by Amanita phalloides. *Experientia*, 1973, *29*, 1520–1521.

4. Grossman, C. M., and Malbin, B. Mushroom poisoning: review of literature and report of two cases by previously undescribed species. *Ann. Int. Med.*, 1954, *40*, 249–259.
5. Litten, W. Most poisonous mushrooms. *Scient. Am.*, 1975, *232*, 91–101.
6. Moss, A. A., and Goldberg, H. I. Intestinal pseudo-obstruction. *CRC. Crit. Rev. Radiol. Sc.*, 1972, *3*, 363–387.
7. Myler, R. K., Lee, J. C., and Hopper, J. Renal tubular necrosis caused by mushroom poisoning: renal biopsy findings by electron microscopy and use of peritoneal dialysis in treatment. *A.M.A. Arch. Int. Med.*, 1964, *114*, 196–204.

RENAL HEMANGIOMAS*

AN ANALYSIS OF 13 CASES DIAGNOSED BY ANGIOGRAPHY

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ABSTRACT:

The angiographic findings in 13 patients with renal hemangiomas (one pararenal) are presented. The material suggests, contrary to earlier reports, a predominance for the female sex and the right kidney. Nine of the patients had macroscopic hematuria, of which five had renal colic. Obstruction was found at urography in seven cases. Renal angiography is the definitive diagnostic procedure and should be performed in all cases of unexplained macroscopic hematuria. Diagnosis may eventually be improved by pharmacoangiography or by hemodynamic studies using a dye dilution technique, as small arteriovenous shunts may remain undetected at angiography. Cardiac decompensation was not noted in our material, even though arteriovenous shunting within the hemangioma was seen in eight cases, suggesting that the shunt flow in renal hemangiomas usually does not reach levels high enough to impair the general circulation. Hypertension was present in only one patient, attributed to chronic pyelonephritis. Hematuria disappeared in all nine patients operated upon.

HEMANGIOMAS of the kidney are considered rare. Only scattered reports exist of hemangiomas diagnosed *in vivo* and preoperatively.[1,3,5,7,8,13,14,16,19,20] As hemangiomas often cause hematuria, a correct preoperative diagnosis is fundamental for the planning of surgery, which may be limited to renal resection. We therefore thought it worthwhile to report our experience with 13 cases diagnosed by angiography, six of which have been presented earlier by this institution.[1,3,14,16] We thus add seven new cases to the literature.

MATERIAL AND METHOD

The material was comprised of 13 patients, two males and eleven females, aged from 21 to 60 years. Urography and bilateral selective angiography of the kidneys in at least two projections were performed in all cases. In reviewing the angiograms special attention was paid to kidney size, caliber of main renal artery, size of hemangioma, possible arteriovenous shunting, and evidence of local ischemia peripheral to the hemangioma. In two cases renal blood flow studies were performed with a dye dilution technique in connection with angiography.[14,15] Shunt flow was calculated according to Ekelund and associates.[9] In one case renal phlebography was also performed.

RESULTS

(Table 1)

Five of the patients were examined because of renal colic and macroscopic hematuria (Cases I–III, X and XIII) and in all of them obstruction was found at urography. Further, four patients with macroscopic hematuria without pain were referred for urography, and in two cases obstruction was demonstrable. In one of them urography revealed infundibular displacement; in the other patient, with painless macroscopic hematuria, the examination was normal. Urography was also normal in two patients with painless microscopic hematuria (Cases V and VII). In one patient examined because of hypertension and recurring urinary infections, pyelonephritic changes were found at urography (Case XII), while in the last patient, investigated for suspected malignancy, it was normal (Case XI).

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Table I

CLINICAL AND ROENTGENOLOGICAL FINDINGS IN 13 PATIENTS WITH RENAL HEMANGIOMAS

Case	Age (years)	Sex	Renal Colic	Hematuria	Urography	Angiography	
						Size of Hemangioma	Arteriovenous Shunting
I	30	F	+	Macroscopic	Obstruction	20×30 mm	+
II	26	M	+	Macroscopic	Obstruction	10×20 mm	−
III	28	F	+	Macroscopic	Obstruction	25×15 mm	+
IV	52	F	−	Macroscopic	Obstruction	30×40 mm	+
V	60	F	−	Microscopic	Normal	30×20 mm	+
VI	36	F	−	Macroscopic	Infundibular displacement	20×40 mm	+
VII	31	F	−	Microscopic	Normal	7×12 mm	−
VIII	21	F	−	Macroscopic	Obstruction	Multiple	
						<5×5 mm	−
IX	30	F	−	Macroscopic	Normal	26×52 mm	+
X	54	F	+	Macroscopic	Obstruction	12×22 mm	−
XI	50	M	−	−	Normal	18×13 mm	−
XII	35	F	−	—	Pyelonephritic changes	30×25 mm	+
XIII	47	F	+	Macroscopic	Obstruction	35×35 mm	+

The angiographic findings in Cases I–VI have previously been reported by this clinic.[1,3,14,16] In twelve patients the hemangioma was solitary, the smallest measuring seven × twelve mm and the largest 26×52 mm. In a 21 year old female (Case VIII) multiple hemangiomas were found at angiography (Fig. 1). Surgery consisted of left-sided pyelolithotomy: postoperatively she developed serious hematuria, requiring blood transfusions. Ten days postoperatively, renal angiography was performed in order to reveal the bleeding source. During early childhood the patient had had multiple cavernous skin hemangiomas which had been irradiated and subsequently disappeared, therefore the diagnosis of renal hemangiomatosis was made and a nephrectomy performed. The diagnosis was verified at microscopic examination.

There was neither significant difference in the width of the renal artery on the side of the hemangioma as compared to the contralateral side, nor a significant difference in renal size except from Case VIII, with an enlarged left kidney because of postoperative edema.

In eight patients arteriovenous shunting of contrast medium within the hemangioma was evident at angiography. In Case VI this shunting was estimated by a dye-dilution technique at angiography.[14] A 20×40 mm hemangioma situated centrally in the upper pole of the right kidney was found at arteriography and was also partly visualized at renal phlebography. Renal blood flow of the right kidney was calculated to 629 ml/min (normally 450 ml/min) and the shunt flow was estimated to 209 ml/min. Blood flow studies were also performed in Case VII, where angiography revealed a seven × twelve mm hemangioma in the upper pole of the right kidney without demonstrable arteriovenous shunting. In spite of this a small shunt flow of twelve ml/min was shown by the dye dilution study.

One patient had hypertension (Case XII). This was a 35 year old woman with chronic pyelonephritis and a blood pressure of 165/110. Urography had demonstrated the kidneys to be of equal size with pyelonephritic changes noted in the right kidney. She had no hematuria, but angiography was performed to determine the degree of renal parenchymatous destruction. A 30

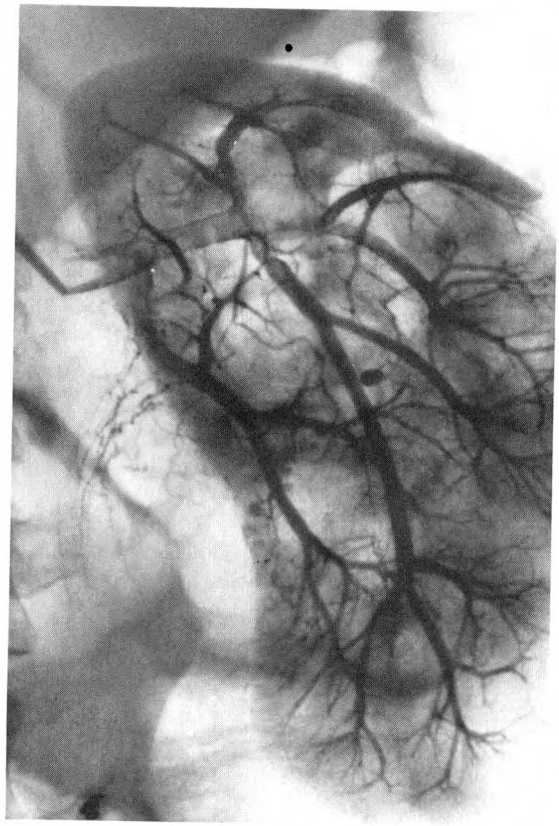

FIG. 1. Case VIII. Angiography ten days following pyelolithotomy. Swollen kidney. Multiple small hemangiomas. Irregular and tortuous arteries in wall of dilated kidney pelvis. Microscopy: renal hemangiomatosis.

×25 mm hemangioma was found in the upper part of the right kidney with early filling of draining veins. Peripheral to the hemangioma there were reduced vascularity and localized atrophy, which were taken as evidence of focal ischemia (Fig. 2). Because of rapidly progressing uremia this patient was not operated upon, but has been successfully treated with hemodialysis.

Signs of reduced perfusion distal to the hemangioma were also found in a 47 year old woman (Case XIII) with right-sided renal colic and macroscopic hematuria. Angiography revealed a 35×35 mm hemangioma in the upper half of the right kidney with heavy arteriovenous shunting (Fig. 3). In this case, nephrectomy had to be performed because at operation the surface of the lower pole of the kidney was also found to be involved—a finding that was not demonstrated at angiography.

Ten hemangiomas were located in the right kidney: five in the superior pole; two in the middle part; and three in the inferior pole. One of the three left-sided hemangiomas had a peculiar localization medial to the kidney (Case IX). This was a 30 year old female who was investigated because of macroscopic hematuria. The 26×52 mm hemangioma was fed from a widened inferior suprarenal artery and located medial and dorsal to the kidney but in close connection with the renal pelvis (Fig. 4). The patient was not operated upon because the hematuria was thought to be explained by a nephritis—verified at biopsy.

Nine of our patients were operated upon (Cases I–VI; VIII; X and XIII) and in four it was possible to perform a local resection only (Cases I; II; V and X), while in the others nephrectomy was necessary. In all operated cases hematuria disappeared following surgery.

DISCUSSION

Judging from autopsy materials, hemangiomas of the kidney are extremely rare. Bell[2] observed only one hemangioma in 30,000 autopsies. Andersson and associates[1] reviewing autopsy material of 12,765 cases from Malmö, Sweden, found three. Until today approximately 170 cases have been reported, a minority of which have been diagnosed *in vivo* by angiography. Romaniuk[22] after reviewing 1,500 renal angiographies found only one hemangioma. Recently, reports have appeared suggesting that this type of vascular lesion may be more common than previously expected.[16,21] As such lesions may be extremely small it is easy to understand that they may be overlooked at a routine autopsy without serial sections of the kidneys, especially if in a collapsed state. It is also well known that hemangiomas may be too small to be detected at angiography. Andersson *et al.*[1] reported five cases of histologically verified hemangiomas smaller than ten mm in di-

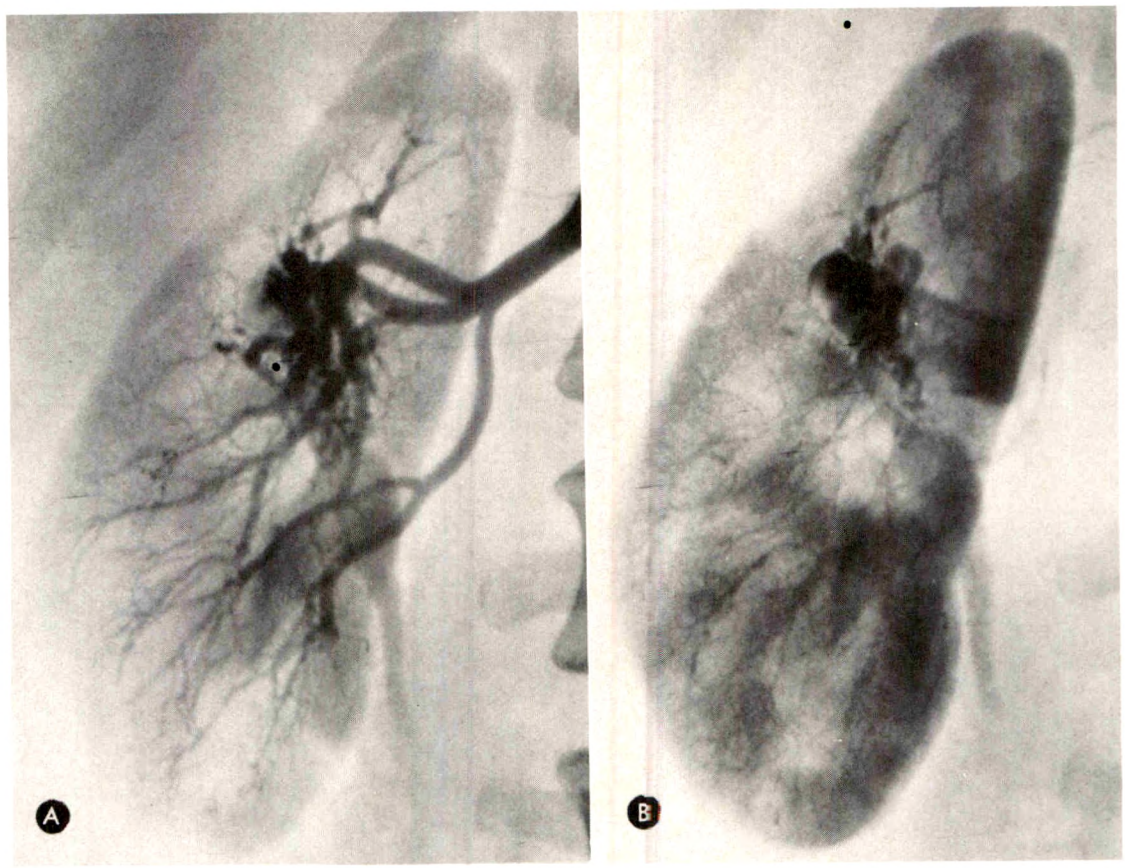

Fig. 2. Case xii. Hemangioma in upper half of the right kidney with early filling of the renal vein. (*A*) Early and (*B*) late arterial phases. Reduced perfusion and localized atrophy peripherally are noted.

ameter where angiography failed to reveal any pathological changes. These authors stressed the importance of selective examinations with high quality angiograms for demonstration of this type of lesion. It is possible that magnification angiography (with a small focal spot) could increase the diagnostic accuracy in such lesions.

Hemangiomas are composed of various-sized, endothelium-lined spaces (small, capillary; large, cavernous) lacking contractile elements, and in this regard are similar to other types of neoplastic vasculature. Such vessels do not react on vasoactive stimuli and therefore pharmacoangiography with vasoconstricting drugs might improve diagnosis as suggested by Andersson and associates.[1] In this connection it is of interest to note that improved visualization of hemangiomas in the liver has been reported after pharmacoangiography with angiotensin[11] and vasopressin.[4]

Ekelund *et al.*[9] studied arteriovenous shunts in rabbit kidneys by a dye-dilution technique at angiography and were able to show that minute shunts, not demonstrable by angiography, could be detected by dye-dilution technique. They suggested that a combination of (pharmaco-) angiography and hemodynamic studies using dye-dilution technique should be valuable in the search of small renal vascular shunts, especially if angiotensin was used. This possibility is illustrated by Case vii where a small shunt of twelve ml/min was diagnosed at the blood flow studies. This shunt could not be seen at angiography, yet the hemangioma was visualized.

A large shunt in the kidney may lead to cardiomegaly and other signs of heart

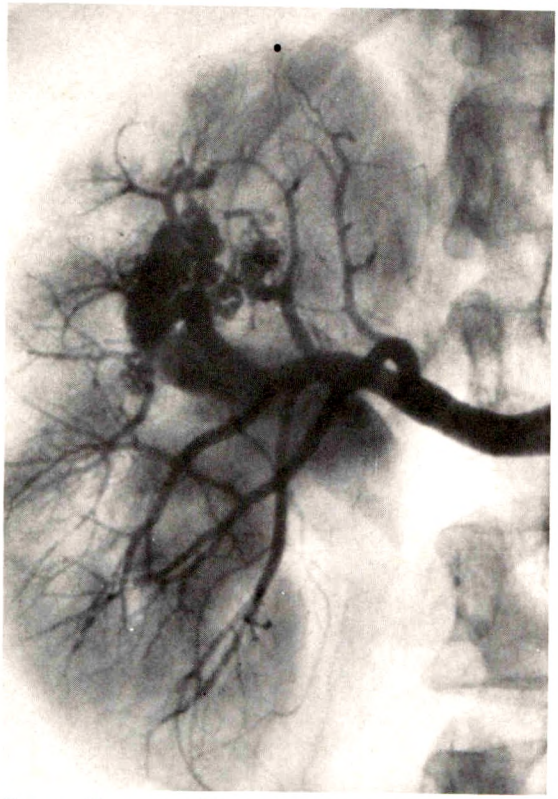

FIG. 3. Case XIII. Extensive hemangioma in upper half of right kidney with heavy arteriovenous shunting. Reduced cortical perfusion peripheral to hemangioma.

failure.[6,17] This is probably rare in hemangiomas where the shunt flow does not reach values as high as in cases with a direct fistula between renal arteries and veins. An example of such a congenital arteriovenous fistula is given by Christensson and associates[6] concerning a 54 year old woman with heart failure, where the shunt flow was 2.8 ml/min. In this case the cardiac output preoperatively was 6.8 ml/min and after nephrectomy 4.3 ml/min. None of our 13 cases had signs of cardiac decompensation.

Decreased perfusion of the renal parenchyma peripheral to an arteriovenous shunt may lead to localized renal ischemia and ensuing hypertension.[10,13,17,18] Arteriovenous shunting within the hemangioma was evident at angiography in eight of our cases. In two cases (Cases XII and XIII) reduced vascularization was demonstrated peripheral to the shunt. One of these patients had hypertension which, however, was attributed to chronic pyelonephritis. All other patients in our material had normal blood pressures.

Peterson and Thompson[21] after reviewing the reports of renal hemangiomas through 1967 (more than 150 cases) found that the cases were nearly equally distributed between sexes and that the right and left kidneys were essentially equally involved. Our material, however, displays a heavy predominance for the female sex—eleven of 13 patients were women. The right kidney was also more often involved than the left —ten hemangiomas being situated on the right side.

FIG. 4. Case IX. Large extrarenal hemangioma in close relation to kidney pelvis fed from a widened inferior suprarenal artery.

From roentgenological methods available, urography may sometimes be helpful by showing pelvic displacement or defects.[12,13,19,21] Infundibular displacement was evident at urography in one of our cases with a hemangioma 20×40 mm in size. Except for large hemangiomas, however, urography does not usually demonstrate pathological changes.[1] In cases of renal colic, macroscopic hematuria, or both, urography gives valuable information in demonstrating obstruction.

A "segmental nephrogram effect" at retrograde pyelography has been claimed as an important diagnostic sign of hemangioma of the kidney.[23] Andersson *et al.*[1] also observed a pyelointerstitial reflux of contrast medium in one of their cases at a place where the hemangioma was later found at surgery. As stated by these authors, reflux during retrograde pyelography is, however, such a common phenomenon that this finding cannot be attributed real value.

The ultimate diagnostic tool is selective renal angiography, a procedure which is especially indicated in cases where unilateral bleeding has been established at cystoscopy. The diagnostic information may eventually be increased by means of pharmacoangiography and hemodynamic studies using a dye-dilution technique in connection with angiography. The angiographic mapping of the renal vascular anatomy is also of major importance for the planning of surgery, as parts of the kidney may be saved by resection of the hemangioma, as documented in four of our cases. Hematuria, the most common complication of renal hemangiomas, disappeared in all nine patients treated by renal resection or nephrectomy in this series.

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REFERENCES

1. Andersson, L., Boijsen, E., and Henrikson, H. Hämangiom und andere Gefässabnormitäten der Niere als Ursachen der Hämaturie. *Der Urologe*, 1967, *6*, 119–126.
2. Bell, E. T. Classification of renal tumors with observations of frequency of various types. *J. Urol.*, 1938, *39*, 238–243.
3. Boijsen, E., and Köhler, R. Renal arteriovenous fistulae. *Acta radiol.*, 1962, *57*, 433–445.
4. Boijsen, E., and Göthlin, J. To be published.
5. Boné Andersen, J., and Rasmussen, Th. Renal hemangioma diagnosed preoperatively by selective renal angiography. *Acta radiol.*, 1964, *2*, 201–204.
6. Christensson, B., Ekelund, L., Lindstedt, E., Nordenfelt, I., and Olin, T. Renal arteriovenous fistula—cause of heart failure. *Acta med. scandinav.*, 1974, *196*, 513–516.
7. Crummy, A. B., Jr., Atkinson, R. J., and Caruthers, S. B., Jr. Congenital renal arteriovenous fistulas. *J. Urol.*, 1965, *93*, 24–26.
8. Desai, S. G., and Desantels, R. E. Congenital arteriovenous malformation of kidney. *J. Urol.*, 1973, *110*, 17–19.
9. Ekelund, L., Göthlin, J., and Olin, T. Arteriovenous fistulae in rabbit kidney studied by dye-dilution technique and by angiography. *Scandinav. J. Urol. Nephrol.*, 1972, *6*, 84–90.
10. Ekelund, L., Göthlin, J., Lindholm, T., Lindstedt, E., and Mattsson, K. Arteriovenous fistulas following renal biopsy with hypertension and hemodynamic changes: report of case studied by dye-dilution technique. *J. Urol.*, 1972, *108*, 373–376.
11. Ekelund, L., and Lunderquist, A. Pharmacoangiography with angiotensin. *Radiology*, 1974, *110*, 533–540.
12. Feinberg, S. B., and Goldberg, M. E. Arteriovenous communication of kidney in patient with hypertension. *Radiology*, 1963, *81*, 601–603.
13. Gunterberg, B. Renal arteriovenous malformation. *Acta radiol.* (Diag.), 1968, *7*, 425–430.
14. Göthlin, J. Dye dilution technique with angiography in investigation of renal arteriovenous shunting. *Acta radiol.* (Diag.), 1973, *14*, 305–314.
15. Göthlin, J., and Olin, T. Dye-dilution technique with nephroangiography for determination of renal blood flow and related parameters. *Acta radiol.* (Diag.), 1973, *14*, 113–137.
16. Jonsson, K. Renal angiography in patients with hematuria. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *66*, 758–765.
17. Maldonado, J. E., Sheps, S. G., Bernatz, P. E., De Weerd, J. H., and Harrison, E. G. Renal arteriovenous fistula: reversible cause of hypertension and heart failure. *Am. J. Med.*, 1964, *37*, 499–513.

18. MILLOY, F., JR., FELL, E. H., DILLON, R. F., and ZAYAS, A. M. Intrarenal arteriovenous fistula with hypertensive cardiovascular disease. *Am. J. Surg.*, 1958, *96*, 3–11.

19. OCHSENSCHLÄGER, A., and BÖTTGER, E. Angioma racemosum der Niere. *Ztschr. Urol. und Nephrol.*, 1973, *66*, 215–219.

20. PALMER, J. M., and CONNOLLY, J. E. Intrarenal arteriovenous fistula: surgical excision under selective renal hypothermia with kidney survival. *J. Urol.*, 1966, *96*, 599–605.

21. PETERSON, N. E., and THOMPSON, H. T. Renal hemangioma. *J. Urol.*, 1971, *105*, 27–31.

22. ROMANIUK, P. A. Zur Struktur der Nierenbeckenangioms im Renovasogram. Beitrag zum Problem der einseitigen Nierenblutung. *Fortschr. a. d. Geb. d. Röntgenstrahlen. u. d. Nuklearmedizin*, 1962, *110*, 457–462.

23. SUMMERS, J. L., and KEITZER, W. A. Radiographic clue to diagnosis of hemangioma of kidney. *J. Urol.*, 1972, *108*, 852–853.

MEDULLARY RAYS*

VISUALIZATION DURING EXCRETORY UROGRAPHY

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ABSTRACT:

The medullary rays are well-defined anatomic structures consisting of bundles of renal tubules which form in the renal cortex and continue through the renal medulla as the medullary striations. These structures are occasionally visualized during excretory urography in abnormal physiologic states characterized by tubular stasis and hyperconcentration of contrast material. Under these conditions, fine striations are visualized which correspond in position and orientation to these structures.

The work of others has shown that in tubular stasis, contrast material is hyperconcentrated in the tubules. On microradiography in normal and obstructed kidneys, the highest concentrations of contrast material are seen in the lumen of collecting ducts. Lastly, retrograde injection of contrast material into the medullary rays produces a roentgen image similar to our illustrated cases.

THE medullary rays are distinct anatomic structures which can be seen on excretory urography in conditions producing hyperconcentration of contrast medium. They have been illustrated several times in the recent literature. When their visualization was first described in the English speaking literature in 1942,[17] they were not identified. Descriptions have existed in standard histology[16] and pathology[18] textbooks for some time; but, to our knowledge, none exists in the radiology literature. Since anatomic correlation is the foundation of radiology, we wish to describe and identify the medullary rays anatomically and roentgenographically.

In several specific situations, we have noted on excretory urography a fine linear striation in the renal cortex radiating from the hilus. Our investigation led us to conclude that the alternating radiolucent and radiodense stripes were produced by the medullary rays. We then sought to prove that the linear radiodensities produced in such circumstances are due to contrast medium in the medullary rays.

NORMAL ANATOMY

The cut surface of a kidney shows the medullary rays in the cortex. The faint striations are projections of light colored, striated, medullary substance into the cortex from the base of the medullary pyramids[16] (Fig. 1, *A* and *B*). They are composed of loops of Henle and collecting ducts cut longitudinally.[16,18] The rest of the cortex appears finely granular because the highly convoluted proximal and distal tubules are cut randomly in all planes. The clear medullary striations in the renal pyramid are continuations of the loops of Henle and collecting ducts, and the dark striations are the adjoining blood vessels known as vasa recta.

The nephron is the basic functioning unit of the kidney, consisting of Malpighian corpuscle, proximal convoluted tubule, loop of Henle, and distal convoluted tubule. Many nephrons deliver to each collecting duct, and several collecting ducts join to form a duct of Bellini or papillary duct at the papilla. The maximum external diameter is up to 200μ in the papillary col-

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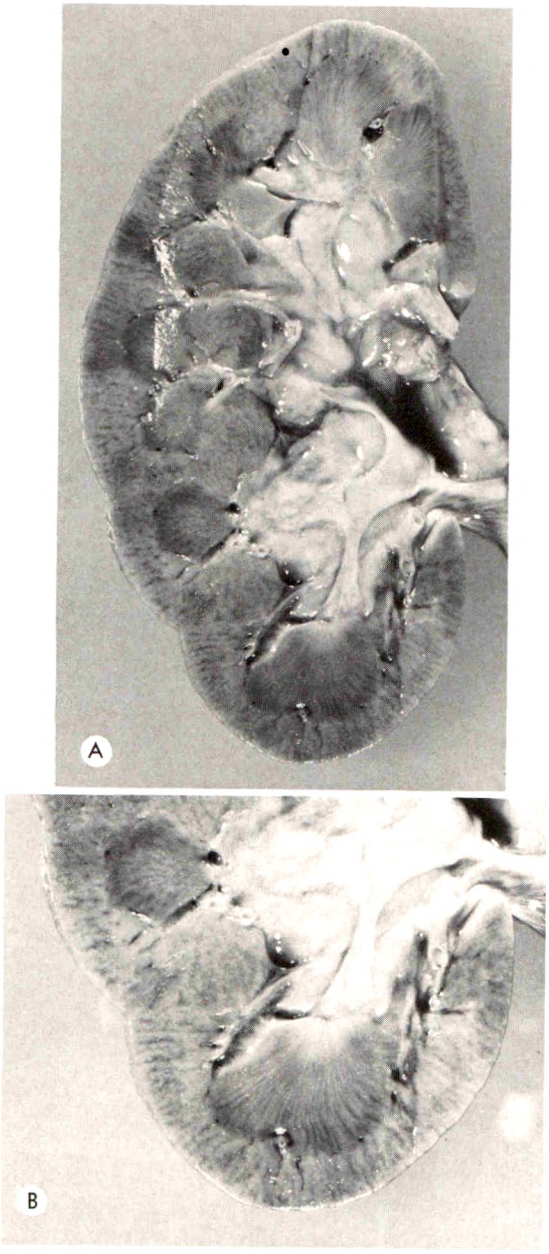

FIG. 1. (*A*) Cross section of fresh human kidney showing fine striations in the cortex. The dark striations are interlobular vessels. The light areas continuous with the medullary striations in the pyramid are medullary rays. (*B*) Enlargement of lower pole.

lecting duct.[23] A schematic representation of the relationships of various portions of the nephrons to the cut surface of the kidney is shown in Figure 2. As the pyramid is the center of a renal lobe, so the medullary ray is the center of a renal lobule[16] (Fig. 3).

NORMAL PHYSIOLOGY

The physiology of contrast medium excretion has been discussed in a number of recent communications.[8,9,24–26] Only the basic principles pertinent to our discussion will be reviewed. Modern contrast media are excreted by glomerular filtration with little or no tubular secretion. The evidence is summarized by Waes.[28] Therefore, the quantity of contrast medium that enters the kidney in the filtered load is the product of the plasma concentration and the glomerular filtration rate, since no more iodine will be added and none reabsorbed. The urinary system is seen on excretory urography because the iodine containing contrast material is concentrated by the kidney. The process of concentration is that of water reabsorption.[11,23,27] While several details of the mechanism are not yet known, many general conclusions have been drawn.

As summarized by Valtin,[27] water reabsorption from the renal tubules is a passive process that depends on osmotic gradients between the tubular lumen and some site within the tubular epithelium. Since Na^+ (and Cl^-) are normally the most abundant osmotically active particles in tubular fluid, water passively diffuses in response to an osmotic gradient set up by these ions. It has been said that 80 to 90 percent of the H_2O in the filtered load is reabsorbed in the proximal tubule,[9,23,24,26] that is, the proximal convoluted tubule (pars convoluta) and the thick portion (pars recta) of the proximal loop of Henle. The more current opinion[27] is that normally no more than 70 percent of the water is removed from the filtered load in this part of the nephron. Since contrast medium is an osmotic diuretic, the amount of water reabsorbed is less than this figure, certainly not more. Nevertheless, during excretory urography the largest volume of water is reabsorbed in the proximal tubule. The cells of the proximal tubule actively reabsorb sodium, and water follows by osmosis.

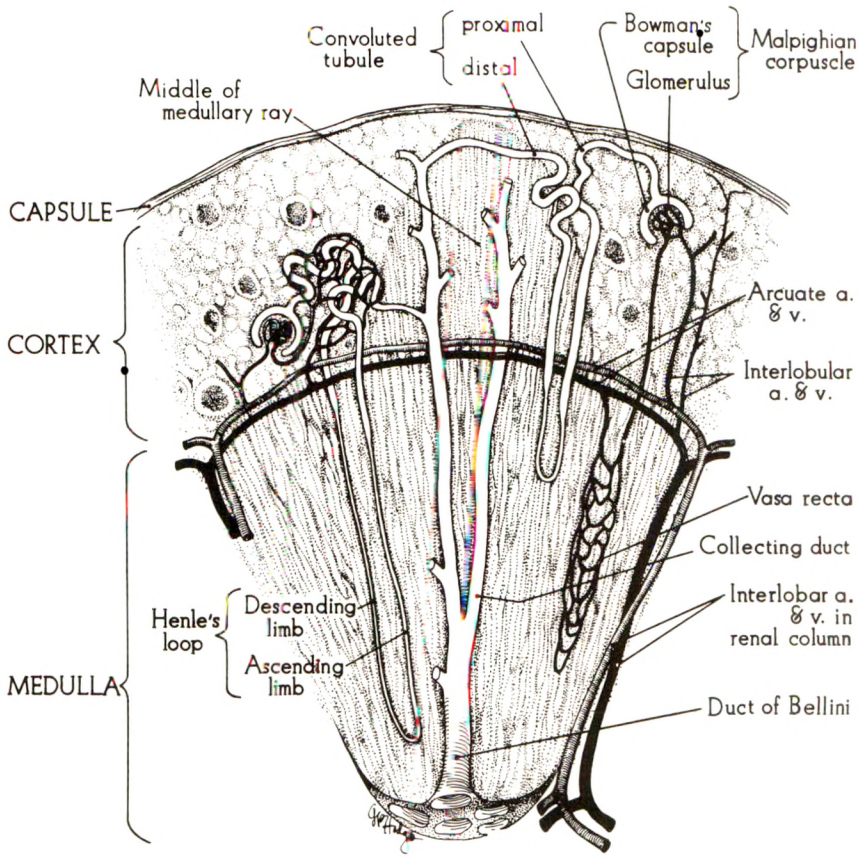

Fig. 2. Artist's sketch demonstrating relationship of nephrons to the cross sectional anatomy. A single lobule is drawn superimposed on a lobe. The medullary rays are bundles of loops of Henle and collecting ducts.

The milieu of the tubules becomes more hyperosmotic centrally because of the anatomic relationship of the loops of Henle with their hairpin turns in the medulla. Valtin[27] states that countercurrent multiplication of the single effect of reabsorbing Na^+ and Cl^-, virtually without water, from ascending loops of Henle results in progressive hyperosmolality of the medullary and papillary interstitium. The buildup of this osmotic gradient from cortex to papilla is aided by reabsorption of urea from the medullary portions of the collecting ducts. In the ascending loop of Henle, solute is reabsorbed to the virtual, but not complete, exclusion of H_2O. In this way, an osmotic gradient is created around the water-permeable descending loop of Henle, and since this gradient is greater centrally, a countercurrent multiplier system is formed. Water resorption up to here is obligatory, unaffected by the state of hydration. As long as the kidney is normal, the proximal tubule will reabsorb water.

Antidiuretic hormone (vasopressin) makes the distal nephron, that is, the distal convoluted tubule and collecting duct, more permeable to water, thereby again exposing the tubular contents to the corticopapillary osmotic gradient. More water is reabsorbed from the filtered load with consequent increase in concentration of the contrast medium in the urinary system. Water reabsorption in the distal nephron is dependent on the state of hydration of the patient. While the amount of water reabsorbed in the distal nephron is small, its proportion to the tubular contents is high and its effect on concentration is great.

Pitts[23] and Valtin[27] describe the balance

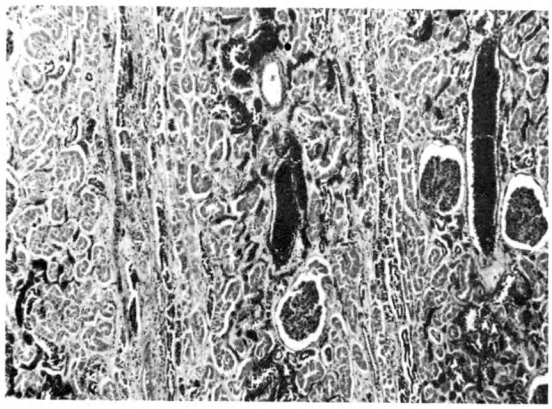

FIG. 3. Low power photomicrograph of inner cortex showing the medullary rays as bundles of tubular structures cut longitudinally. The renal lobules are separated by the interlobular vessels which in this specimen are packed with red blood cells.

of forces involved in glomerular filtration which separates the plasma water and non-protein constituents from the blood cells and protein macromolecules. The glomerular arteriolar pressure is approximately 70 percent of systemic arterial pressure. This pressure is opposed by the hydrostatic pressure in Bowman's space (within Bowman's capsule) and by the plasma osmotic pressure in the glomerular capillary. The latter pressure is exerted by the osmotically active particles in solution in the blood. In the steady state, the net result is positive pressure and flow of filtrate from capillary tuft to Bowman's space. This pressure is the driving force for movement of tubular fluid.

Alterations in this balance of forces can produce stagnation of tubular contents. Since the tubular contents are then exposed to the osmotic gradients for a longer period of time, more water than usual is reabsorbed and hyperconcentration of contrast results. The situations in which the radiating fine cortical striations have been observed on excretory urography have in common that they are associated with changes in function which can reduce the rate of tubular urine flow.

Since the medullary rays conform in location, shape, and distribution to these linear striations (Fig. 4–7), since the contrast medium is most concentrated in this structure, and since there is no known physiological reason for the visualized contrast medium to be anywhere else but in the lumen of the tubules which form the medullary rays, we conclude that the medullary rays were visualized.

MATERIAL AND METHOD

Having concluded from our investigation of anatomy and function that the radiating

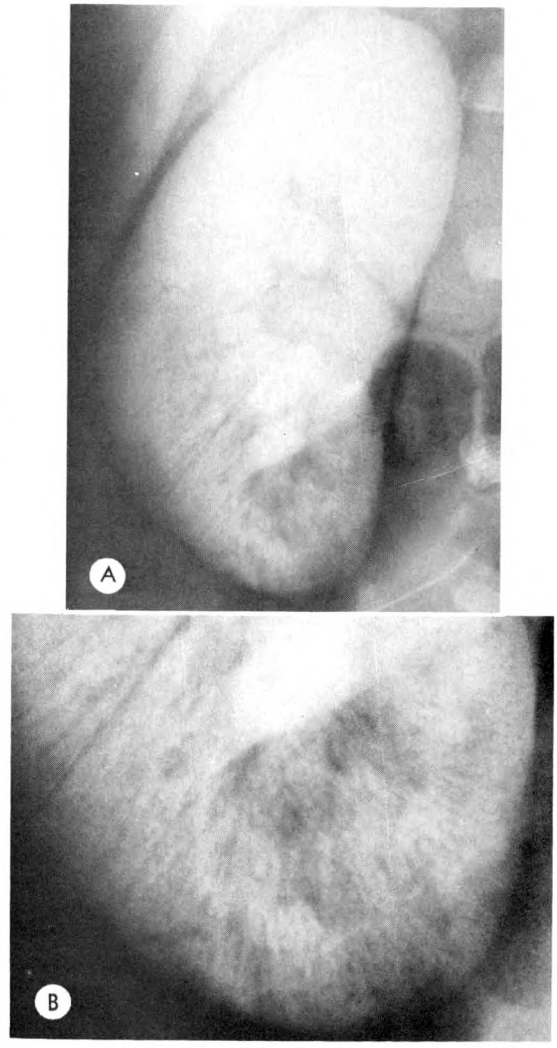

FIG. 4. (*A*) Stasis nephrogram at five hours in 25 year old male with ureteral calculus. (*B*) Enlargement of lower pole. The fine striations in the cortex are contrast-filled medullary rays. Some can be seen extending into the medulla as medullary striations. Compare with Figure 1.

fine cortical striations we observed were due to contrast medium in the bundles of tubules called medullary rays, we sought a practical, simple, and inexpensive method to reproduce this image in such a manner as to allow histologic study. Because it is unlikely that we would have the opportunity to obtain an anatomic specimen with contrast medium in the medullary rays, and because there is no simple way to identify iodinated contrast medium microscopically, we devised an indirect method.

We obtained a roentgenographic image similar to that seen in our patients by retrograde ureteral injection of a normal cadaver kidney. After the ureter was cannulated and the hilar blood vessels tied, a mixture of Conray 60 and several drops of India ink was injected, filling the renal pelvis. In various kidneys, further hand injection under fluoroscopic control obtained combinations of pyelosinus, pyelovenous, pyelotubular, and pyelointerstitial backflow. This result was expected from the work of others.[2,15] After several attempts, gentle hand injection fortuitously produced pyelotubular backflow demonstrating fine radiating striations in the cortex. Roentgenograms were obtained using a 0.6 mm focal spot tube and Kodak Industrex Type M2 Ready Pack.

The specimen was cut and appropriate sections were obtained both unstained and stained with hematoxyline and eosin. These were viewed microscopically to determine the location of the India ink.

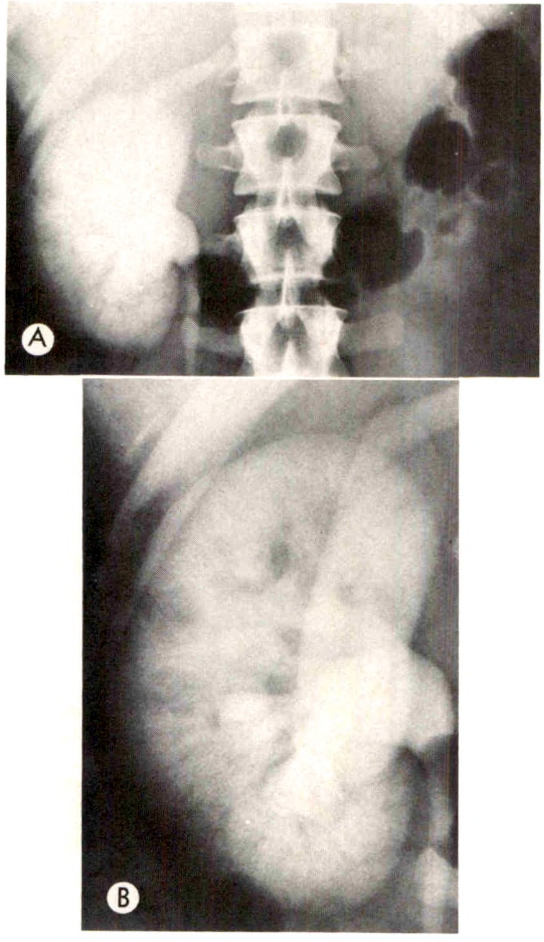

Fig. 5. (*A*) Stasis nephrogram at 24 hours in another 25 year old male with ureteral calculus. There has been vicarious excretion of contrast medium into the gallbladder. (*B*) Enlargement shows medullary rays more clearly.

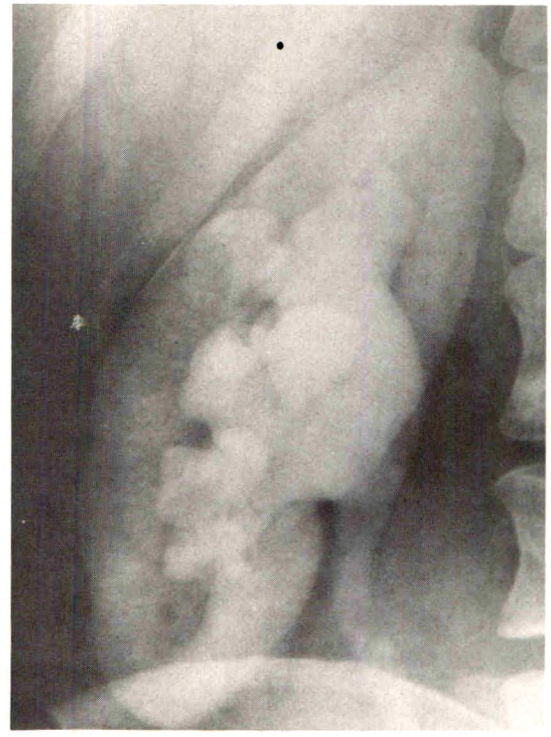

Fig. 6. Thirty-one year old female with segmental periureteral fibrosis secondary to Sansert therapy. The medullary rays are seen particularly along the lateral margin.

RESULTS

The roentgenogram is illustrated (Fig. 8),

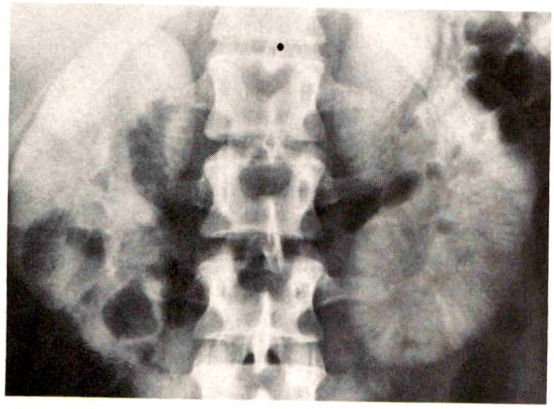

FIG. 7. The medullary rays are well seen in this 19 year old male who was being worked up for lymphoma. This 45 minute delayed roentgenogram was taken for evaluation of the bladder. An excretory urogram the next day was normal without demonstration of the medullary rays. He was on no medication and had no uricosuria or proteinuria. A transient hypotensive episode is postulated.

and the radiating fine cortical striation may be compared to the illustrated cases. Gross and microscopic examination of the cut specimen showed black stain in the medullary striations extending into the cortex (Fig. 9). Unstained (Fig. 10*A*) and stained sections (Fig. 10*B*) show India ink in the medullary rays. High power examination shows India ink in collecting ducts (Fig. 11).

DISCUSSION

As a generalization, the medullary rays are visualized during excretory urography in pathophysiological states wherein tubular stasis produces hyperconcentration of contrast. Fry and Cattell[14] classify such events as producing an increasingly dense nephrogram. This phenomenon may occur in a number of conditions, all associated with changes in function which can reduce the tubular flow rate. Most common are urinary tract obstruction, hypotension, and renal vein thrombosis.

In 1954, Edling and others[12] in an experimental study with rabbits showed that with fall in blood pressure to 60–70 mm Hg, filling of the pelvis by contrast medium ceases and a dense nephrogram is obtained. This finding corresponded with earlier clinical observations.[29] A similar response occurred when the intrapelvic pressure was increased. Furthermore, increasing the blood pressure or removing the ureteral counter pressure diminished the nephrogram and filled the pelvis with contrast material. If the arterial pressure is too low or if the pressure within Bowman's capsule and the renal tubules too high, glomerular filtration will cease and no nephrogram will be produced.

Persky *et al.*[22] in 1959 using dogs showed that radioactive iodinated albumin placed in the pelvis of a kidney with an obstructed ureter would produce "radioautographs" demonstrating pyelotubular backflow. Kazmin *et al.*[19] in 1960 obtained similar results after 48 hours, 72 hours and seven days of obstruction. Elkin and associates[13] concluded that since marked increase in intrapelvic pressure causes renal damage, pelvic pressure is transmitted to the tubule. There is indirect evidence, at least, that elevated intrapelvic pressure is transmitted back to the tubules.

But why does the nephrogram become progressively dense? Brenes and co-workers[5] in 1966 showed that during acute ureteral obstruction with and without diuresis using both Diodrast and Hypaque,

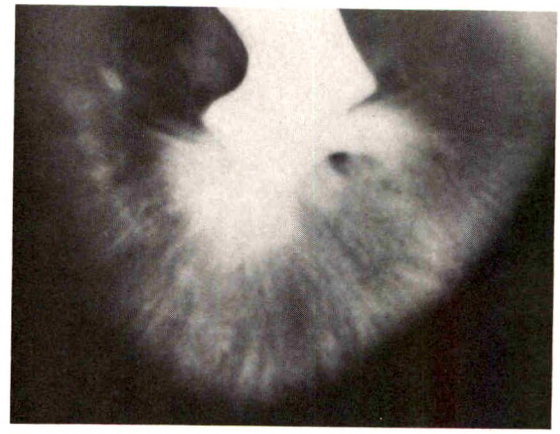

FIG. 8. Pyelotubular backflow produced in an autopsy specimen duplicates fine striation. A mixture of Conray 60 and India ink was injected. The specimen is lying in a puddle of contrast medium on the roentgenogram.

glomerular filtration continued. It was suggested that tubular reabsorption of water continues in the obstructed kidney decreasing the tubular hydrostatic pressure and allowing more contrast medium to enter the tubule *via* the glomerulus. The net effect is a steadily increasing concentration of contrast medium in the renal tubule producing a dense nephrogram. Tubular secretion of contrast medium is not necessary for this effect, since Diodrast, a contrast medium known to be excreted by tubular secretion is as effective as Hypaque, a contrast medium known to have little or no tubular secretion. Korobkin *et al.*[20] suggested that a similar mechanism occurs in the nephrogram of hypotension. The edema produced by renal vein thrombosis may also produce tubular stasis.

The study of Anigstein *et al.*[1] in 1972 using microradiographic techniques on the kidneys of rabbits and rats indicated that Hypaque is excreted in the same manner by both the obstructed and nonobstructed kidney. Direct microscopic analysis of microradiographic sections showed the same relative distribution of contrast material in both normal and obstructed kidneys. However, the obstructed kidneys contained a greater total amount of contrast material. There was no significant amount of contrast material outside the tubular lumen in either group. The highest concentrations of Hypaque were in the lumens of collecting ducts. In both groups the concentrations were not equal in all the collecting ducts. Some showed large amounts and others little if any. This study corroborates Brenes' hypothesis and provides anatomic correlation for our cases.

Demonstration of the medullary rays on excretory urography is uncommon.

Fig. 9. Cut section of specimen demonstrating India ink in the medullary striations and rays.

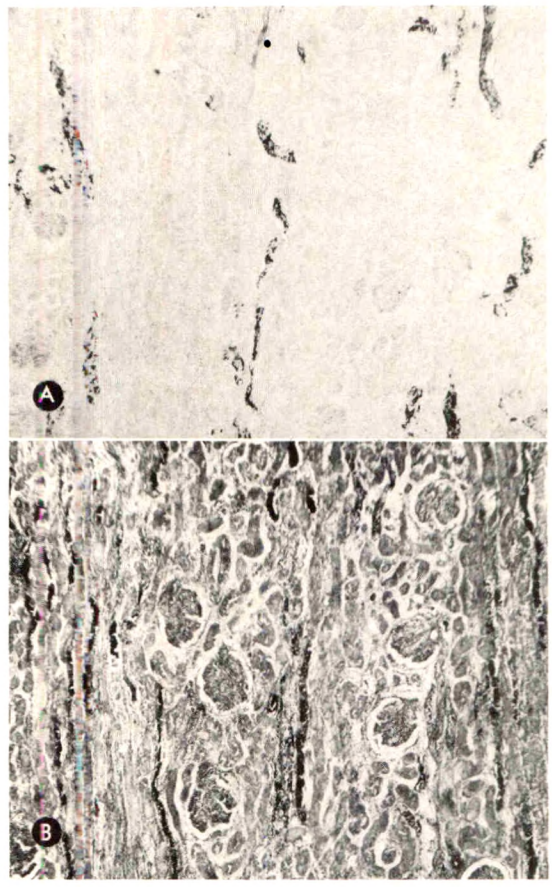

Fig. 10. (*A*) Low power view of unstained section showing India ink in the medullary rays. (*B*) Low power view of stained section showing India ink in the medullary rays.

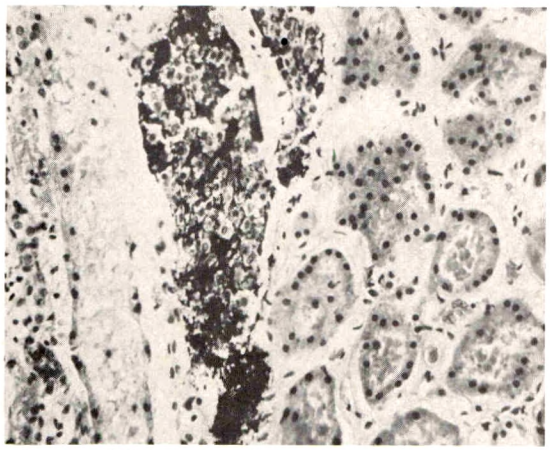

FIG. 11. High power examination showing India ink in a collecting duct.

Whether or not they are visualized depends on the degree and extent of tubular stasis. This phenomenon probably occurs more often than is realized, since it usually occurs after the standard examination is completed. Bretland[6] noted alternate striation in six of his 86 cases of acute ureteric obstruction followed closely until the affected kidney was shown to excrete. He also noted the similarity of the distribution to the medullary rays. The striation appeared after the delayed nephrogram had persisted for some hours. He stated that the pattern of alternate striation is due to reflux of nonopacified urine back up some of the collecting ducts as a function of their individual lengths.[6,7] He has demonstrated how and explained why such an event is possible.[7] We believe that reflux of nonopacified urine may be part of the entire explanation.

The medullary rays appear as linear densities in the cortex corresponding to their known anatomic distribution (Fig. 4–7). These striations are either groups of ducts or individual dilated ducts, since the size of the normal collecting duct is well below the resolving power of conventional roentgenography. In this way the lobular anatomy is revealed. In some instances, further visualization of the tubular anatomy is obtained. The medullary portion of some collecting ducts in the medullary striations may be seen radiating toward the hilus (Fig. 4*B*).

In his classic article on the nephrogram, Hellmer[17] briefly noted fine striations at the periphery of the renal shadow corresponding to the appearance of the cut surface of the kidney. Our experiments show these to be the medullary rays. A more complete correlation has not appeared in the literature since that time. This fine striation can also be seen in an illustration to an article by Berdon *et al.*[4] on Tamm-Horsfall proteinuria. It was probably caused by tubular obstruction by casts. Coel and Talner[10] illustrated an obstructive nephrogram in renal vein thrombosis which showed a fine striation they believed to be opacified collecting ducts. Their illustration bears a striking resemblance to our Figure 4.

A similar radial fine cortical striation corresponding in location, arrangement, and distribution to the medullary rays was seen in six of 14 patients with infantile polycystic disease reported by Lieberman *et al.*[21] The autopsy specimen of their illustrated case shows radially orientated slender cysts where medullary rays are expected in a normal kidney. The microdissection studies of Baxter[3] have shown that the collecting ducts are dilated in this disease. Since we know that medullary rays are bundles of collecting ducts, we feel it appropriate to say the medullary rays were visualized. Hyperconcentration may have played some role, but visualization was due primarily to dilatation of the ducts.

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The radiographic expertise of Meredith Burr, R.T., Linda Bandy, R.T., and Jane E. Boyles, R.T. contributed greatly to the successful outcome of the experimental studies.

REFERENCES

1. Anigstein, R., Elkin, M., Roland, P., and Schulz, R. J. Obstructive nephrogram: micro-

radiographic studies. *Invest. Radiol.*, 1972, *7*, 24–32.
2. Bauer, D. Pyelorenal backflow. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *78*, 296–316.
3. Baxter, T. J. Polycystic kidney of infants and children: morphology, distribution and relation of cysts. *Nephron.*, 1965, *2*, 15–31.
4. Berdon, W. E., Schwartz, R. H., Becker, J., and Baker, D. H. Tamm-Horsfall proteinuria. *Radiology*, 1969, *92*, 714–722.
5. Brenes, L. G., Forlano, H., Koutouratsas, N., and Stauffer, H. M. Mechanism of nephrographic effect during urinary stasis. *Acta radiol.*, 1966, *4*, 14–20.
6. Bretland, P. M. Acute Ureteric Obstruction: a Clinical and Radiological Study. Appleton-Century-Crofts, Inc., New York, 1972.
7. Bretland, P. M. Acute ureteric obstruction: clinical and radiological aspects. *Proc. Roy. Soc. Med.*, 1974, *67*, 1215–1217.
8. Cattell, W. R. Excretory pathways for contrast media. *Invest. Radiol.*, 1970, *5*, 473–497.
9. Cattell, W. R., Fry, I. K., Spencer, A. G., and Purkiss, P. Excretion urography. *Brit. J. Radiol.*, 1967, *40*, 561–580.
10. Coel, M. N., and Talner, L. B. Obstructive nephrogram due to renal vein thrombosis. *Radiology*, 1971, *101*, 573–574.
11. Dicker, S. E. Mechanisms of Urine Concentration and Dilution in Mammals. Williams & Wilkins Company, Baltimore, 1970.
12. Edling, N. P. G., Helander, C. G., and Renck, L. Correlation between contrast excretion and arterial and intrapelvic pressures in urography: experimental study in rabbits. *Acta radiol.*, 1954, *42*, 422–450.
13. Elkin, M., Boyarsky, S., Martinez, J., and Kaplan, N. Physiology of ureteral obstruction as determined by roentgenologic studies. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 291–301.
14. Fry, I. K., and Cattell, W. R. Nephrographic pattern during excretion urography. *Brit. M. Bull.*, 1972, *28*, 227–232.
15. Green, N., Fingerhut, A. G., and French, S. Mechanism of renovascular backflow. *Radiology*, 1969, *92*, 531–536.
16. Ham, A. W., and Leeson, T. S. Histology. Fourth edition. J. B. Lippincott, Philadelphia, 1970.
17. Hellmer, H. Nephrography. *Acta radiol.*, 1942, *23*, 233–254.
18. Heptinstall, R. H. Pathology of the Kidney. Little, Brown & Company, Boston, 1966.
19. Kazmin, M., Persky, L., and Storaasli, J. P. Backflow patterns in experimental chronic hydronephrosis. *J. Urol.*, 1960, *84*, 10–13.
20. Kolobkin, M. T., Kirkwood, R., and Minagi, H. Nephrogram of hypotension. *Radiology*, 1971, *98*, 129–133.
21. Lieberman, E., Salinas-Madrigal, L., Gwinn, J. L., Brennan, L. P., Fine, R. N., and Landing, B. H. Infantile polycystic disease of kidneys and liver: clinical, pathological, and radiological correlations and comparison with congenital hepatic fibrosis. *Medicine*, 1971, *50*, 277–318.
22. Persky, L., Bonte, F. J., and Austen, G. Mechanisms of hydronephrosis: radioautographic backflow patterns. *J. Urol.*, 1959, *75*, 190–193.
23. Pitts, R. F. Physiology of the Kidney and Body Fluids. Year Book Publishers, Inc., Chicago, 1963.
24. Saxton, H. M. Urography. *Brit. J. Radiol.*, 1969, *42*, 321–346.
25. Sherwood, T. Physiology of intravenous urography. *Scient. Basis Med. A. Rev.*, 1971, 336–348.
26. Talner, L. B. Urographic contrast media in uremia. *Radiol. Clin. North America*, 1972, *10*, 421–432.
27. Valtin, H. Renal Function: Mechanisms Preserving Fluid and Solute Balance in Health. Little, Brown & Company, Boston, 1973.
28. Waes, P. F. G. M. van. High dose urography in oliguric and anuric patients. *Excerpta Med.*, 1972.
29. Wickbom, I. Influence of blood pressure in urographic examination. *Acta radiol.*, 1950, *34*, 1–5.

THE NATURAL HISTORY OF THE UPPER RENAL TRACTS IN ADULTS FOLLOWING URETERO-ILEAL DIVERSION (BRICKER PROCEDURE)*

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ABSTRACT:

The natural history of the upper tracts (170 renal units) of 87 patients undergoing total cystectomy and ileal loop diversion has been followed roentgenologically for up to seven years. The complications inherent in the procedure are illustrated and the appearances of new upper tract tumors demonstrated. Of 126 renal units which were normal preoperatively, 87 remained normal. The most common complication was obstruction of the anastomosis, seen in 29 units. The need for regular contrast material examination of the surgically modified upper tracts is emphasized.

TOTAL cystectomy with insertion of the ureters into an isolated loop of ileum is a well-established surgical procedure, one use of which is in the treatment of certain stages of bladder cancer. Because of the type of urinary diversion and the nature of the underlying disease process, these patients require frequent postoperative contrast material examinations of the surgically modified urinary tract. It is the purpose of this communication to report our experiences, paying particular attention to the natural history of the kidneys subjected to the invariable reflux from the ileal loop, and also to document the complications inherent in the procedure with emphasis on their roentgenological features.

MATERIAL AND METHOD

Eighty-seven patients who have undergone urinary diversion using an isolated loop of ileum form the basis of this report. All had transitional cell carcinoma of the bladder. The degree of hydronephrosis demonstrated by intravenous urography in the individual collecting system (or renal unit) was graded according to the criteria shown in Table I.[3,17] Two patients had congenital absence of one kidney, and one nonfunctioning kidney was removed at the time of the original surgery, as was one kidney of a fourth patient with a concomitant renal pelvis transitional cell carcinoma. Thus, 170 postoperative renal units were available for assessment and comparison with their preoperative appearance. In addition to assigning a degree of hydronephrosis to the individual collecting systems, the renal length and substance thickness were also measured, and the roentgenological features of complications were recorded.

Some of the patients had "ileal loopograms," and the presence or absence of reflux was noted. In those patients in whom progressive postoperative obstruction was

TABLE I

CRITERION FOR GRADING

Individual Collecting System	Grade
Normal	0
Minimal or questionable hydronephrosis	1
Definite hydronephrosis	2
Massive hydronephrosis	3
Nonvisualization	4

* Presented at the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, Quebec, Canada, September 25–28, 1973.

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TABLE II

COMPARISON OF PREOPERATIVE AND POSTOPERATIVE APPEARANCES

Preoperative Urogram	Postoperative Urogram					
	Normal	Abnormal but Improved	Unchanged	Deterioration†	New Tumor Formation	Post-obstruction Atrophy
Normal 126	87			36*	3	
Mild hydronephrosis 7	2		2	3		
Moderate hydronephrosis 9	3	3*	1	2		
Severe hydronephrosis 16	1	9*	3*		1	2
Nonvisualization 7		4				3
Chronic pyelonephritis 5			5			
Total—170	93	16	11	41	4	5

* Indicates calculus formation.
† See text.

suspected, antegrade pyelograms were performed. Two of the four patients who developed new upper tract tumors had selective renal arteriograms.

RESULTS

Table II compares the preoperative and postoperative urographic appearances of the individual renal units and demonstrates the following noteworthy features. Of the 126 preoperative normal renal units, 87 remained normal. These patients have been followed for periods varying from six months to seven years to determine if any roentgenographic change could be observed in the upper tracts which are subjected to the almost inevitable reflux from the ileal loop.[4,18,22] In not one was there a generalized or localized loss of renal substance nor was there an alteration in the calyceal architecture. In 13 patients who had loopograms with normal postoperative upper tracts, there was failure to reflux in only one system. In the presence of a normal urogram this reaction indicates that a fortuitous flap-like valve may be present at the uretero-ileal anastomosis.[13] It was also noted that in the three units which showed roentgenographic evidence of chronic pyelonephritis preoperatively, there was no change following radical bladder surgery.

Thirty-six of the 126 normal preoperative units exhibited increasing hydronephrosis postoperatively as did five of the preoperative hydronephrotic units. The causes of deterioration in these 41 units are shown in Table III. The most common cause of deterioration was obstruction at or very close to the uretero-ileal anastomosis,

TABLE III

CAUSE OF DETERIORATION IN 41 PATIENTS

Obstruction at uretero-ileal anastomosis	29
Obstruction by calculus	2
Disseminated tumor	8
Cause undetermined	2
	41

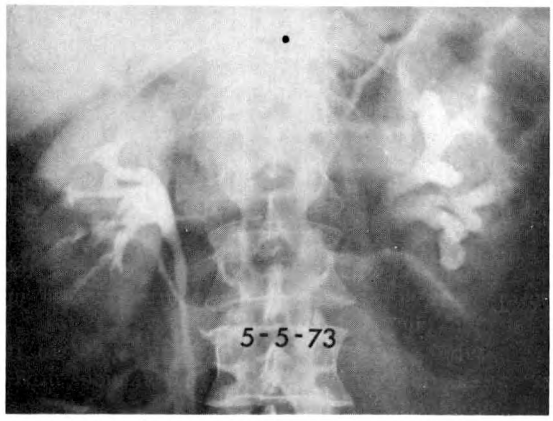

FIG. 1. Urogram performed one month after surgery showing some dilatation of the left collecting system.

demonstrated by postoperative hydronephrosis with dilatation of the ureter down to the ileal insertion. The obstruction was more common on the left side (19 *vs.* ten), and this left-sided preponderance was almost certainly related to the increased technical difficulty associated with the anastomosis on the left, where the ureter must course around the base of the sigmoid mesentery.[2,23]

It was in this type of patient that roentgenology was most helpful, with loopograms and antegrade pyelograms confirming the findings on intravenous urography (Fig. 1–4). Antegrade pyelography has the added advantage of providing a specimen of urine for culture and cytology, two factors of importance in the subsequent surgical management. Of the 29 units with obstruction at the uretero-ileal anastomosis, the sequential intravenous urograms of 18 units showed complete recovery over a period of three to twelve months. This figure also included one patient who had a postoperative nonfunctioning upper tract. An additional operation was required in seven units. In five, the cause of the obstruction was shown to be fibrosis and not recurrent tumor, and two other units showed leakage at the site of the anastomosis. As sequential intravenous urograms showed nonprogressive hydronephrosis in two units with uretero-ileal anastomosis obstruction, further surgery has not been performed, and the fate of two other units is unknown.

In eight units with progressive hydronephrosis appearing after surgery in patients with disseminated malignant disease, pelvic recurrences were found at autopsy. It should be noted, however, that this paper is

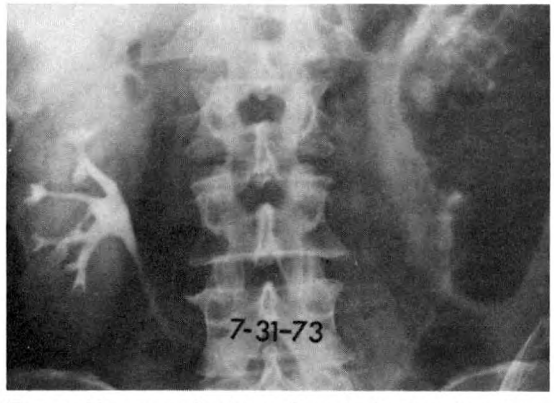

FIG. 2. Two months later there is further deterioration with massive dilatation of the left side and poor opacification.

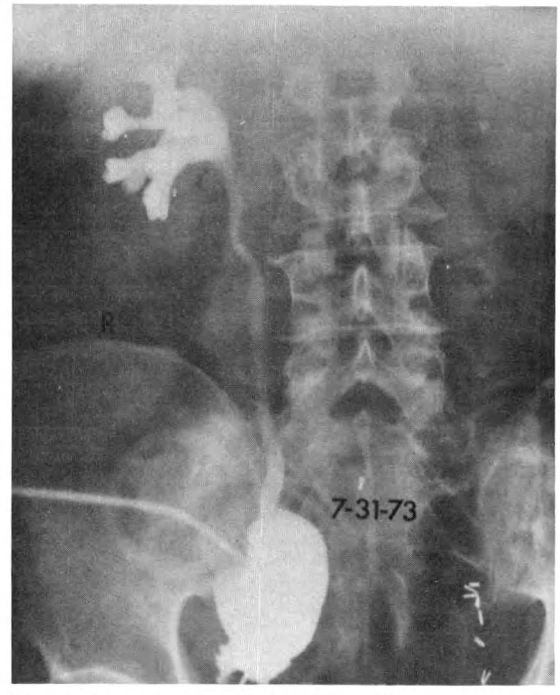

FIG. 3. A loopogram performed at the same time as Figure 2 demonstrates reflux up the right side only.

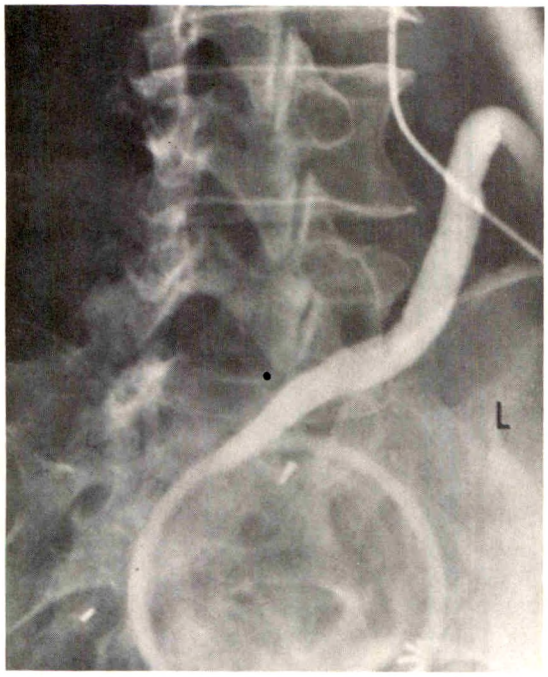

Fig. 4. An antegrade pyelogram confirms the presence of narrowing at the lower end of the left ureter. A fibrous stricture at the anastomosis site was found at surgery.

a survey of the roentgenological findings, and as such will give a falsely low incidence of recurrent tumor because some patients with a clinically obvious recurrence at the site of the original surgery may not have additional contrast material examinations of the urinary tract.

None of the units had stones prior to surgery. In two normal preoperative units that showed increasing hydronephrosis following surgery there was ureteric obstruction due to calculus formation. Calculi

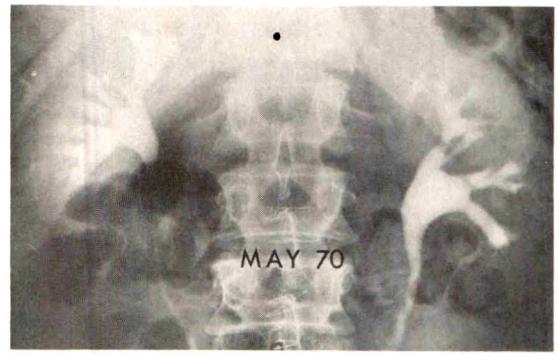

Fig. 5. An intravenous urogram performed one month following surgery shows minimal dilatation of the upper tracts but no evidence of tumor.

developed in five other systems which were hydronephrotic prior to surgery. These calculi appeared between one to four years following the Bricker procedure. All the calculi were radiopaque.

Of the 39 preoperative hydronephrotic units 28 improved following radical surgery. In this group of patients it is important, when assessing functioning in a previously nonfunctioning system, to avoid being misled by contrast medium which has been excreted by the other system and which refluxes from the ileal loop.[17] When the seven systems that were nonfunctioning preoperatively were examined following surgery all showed visualization on the early roentgenograms before contrast medium had entered the ileal loop. Incidentally noted were five units which were either nonfunctioning or showed massive dilatation before operation and following the procedure exhibited the classical features of postobstructive atrophy.[11]

Table IV

CLINICAL DETAILS

Initials	Age at Time of Total Cystectomy	Site of New Tumor	Interval Following Total Cystectomy
J.D.	59	Right renal pelvis	2 years
I.C.	57	Right upper calyx	3 years
Z.I.	66	Right renal pelvis and upper ureter	1.5 years
		Left lower ureter, multiple tumors	4.5 years
R.P.	71	Right ureter, multiple tumors	2 years

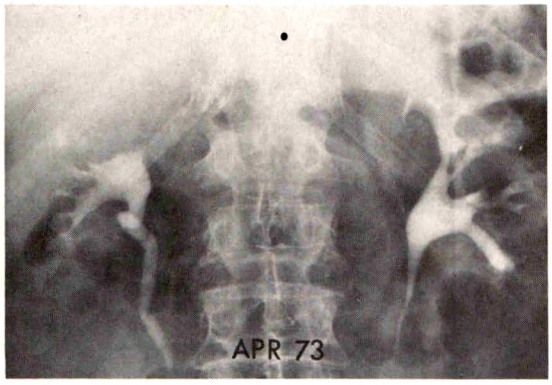

FIG. 6. An intravenous urogram three years later shows obliteration of the major calyx of the upper pole of the right kidney with irregularity of the upper pelvis.

The upper tracts of four patients developed new tumors between 18 to 36 months after total cystectomy. There was no roentgenological evidence of tumor elsewhere at the time of surgery, although all had evidence of carcinoma *in situ* within the distal ureter removed at the time of original surgery. The clinical details are shown in Table IV. The new tumor manifested itself in two patients by progressive narrowing of a major calyx (Fig. 5–7) and in a third by two irregularly surfaced radiolucent filling defects in the right pelvis and upper right ureter which could be misinterpreted as calculi. However, renal arteriography provided conclusive evidence that there was a new tumor (Fig. 8–10). This patient had a right nephroureterectomy, and three years later developed multiple ureteric tumors (Fig. 11) on the left similar to the multiple ureteric tumors found in the fourth patient (Fig. 12).

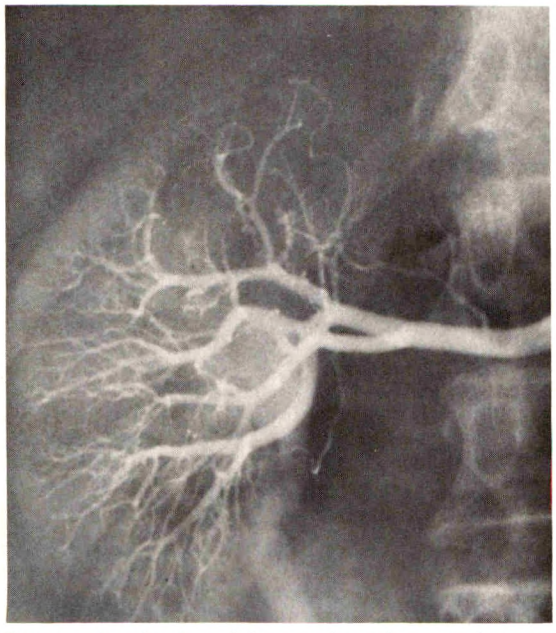

FIG. 7. A selective right renal angiogram demonstrates pruning of some of the interlobar arteries to the upper pole with irregularity and beading due to tumor encasement. Surgery confirmed the presence of a new tumor in the upper pole.

DISCUSSION

Following Bricker's original description of the uretero-ileal conduit in 1950,[1] several large series in adults have established its adequacy, and the complications are well documented.[3,6,7,10,12,16,18]

The most common postoperative complication was obstruction at the uretero-ileal anastomosis. Intravenous urograms, usually performed within a month of surgery, showed deterioration in 29 systems due to anastomotic narrowing. In this situation the surgeon is interested in the patency of the uretero-ileal anastomosis, and failure to reflux from the ileal loop with a hydronephrotic collecting system indicates obstruction at the anastomosis.[13,22] Further information was obtained in some cases by

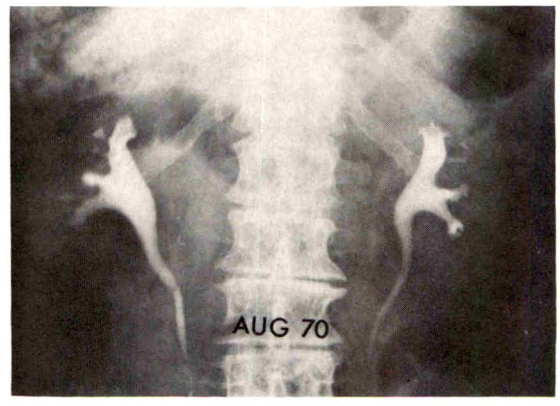

FIG. 8. Intravenous urogram before total cystectomy shows minimal dilatation of the upper tracts but no evidence of new tumor formation.

antegrade pyelography which, in addition to confirming the site of the obstruction, supplied a sample of urine from the closed system. In the majority of cases the obstruction was due to edema which resolved over a period of three to six months, similar findings having been reported by other workers.[3,12,13] In eight units progressive hydronephrosis appeared later in the postoperative period, and at autopsy extensive pelvic recurrence was found. That the later appearance of hydronephrosis is very suggestive of recurrence at the site of original surgery has been recorded previously.[3]

In this series calculi appeared one to four years following surgery. A higher incidence of calculous disease may become apparent upon longer follow-up, calculi having been reported five to 15 years following the Bricker procedure.[14] Of the seven units which showed calculus formation following surgery, in only two were the systems not dilated. Dretler[5] covered the subject of stone formation very extensively, and the precipitating factors included a large volume or an obstructed system.

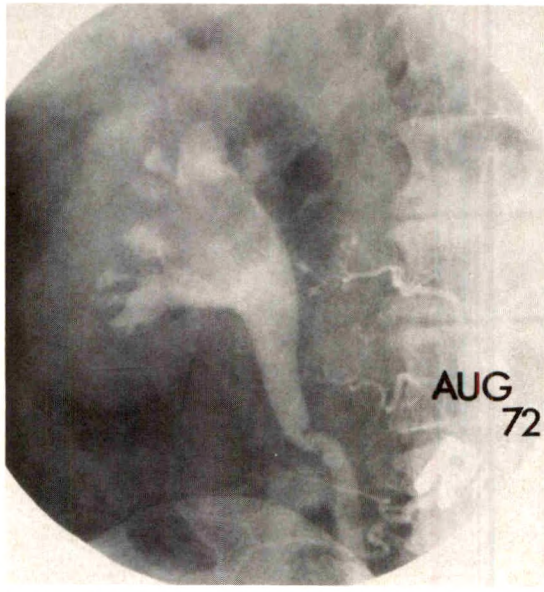

Fig. 9. Urogram at the time of angiography 18 months following surgery demonstrates filling defects with irregular surfaces within the right renal pelvis and in the ureter at the level of L-4.

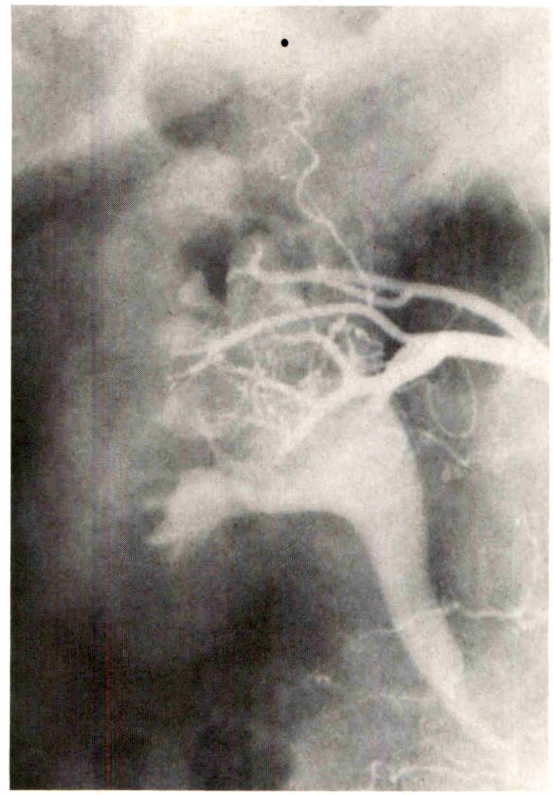

Fig. 10. A right selective arteriogram demonstrates neovascularity within the pelvic tumor. Surgery confirmed the presence of transitional cell carcinomata in the pelvis and ureter.

Speculation on the source of upper tract tumors occurring after total cystectomy has moved away from the tumor implantation theory toward the hypothesis of a generalized instability of the urothelium perhaps caused by a biochemical carcinogen.[19] Further evidence for this latter hypothesis is provided by the four patients in our series. The long time interval of 18 to 36 months between total cystectomy and the appearance of new upper tract tumors makes the implantation theory less acceptable. Instability of the urothelium was seen in all four patients in whom there were changes of carcinoma *in situ* at the level of the ureteric orifice distant from the primary tumor at the time of original surgery. These four patients are from a larger series recently reported[21] in which it

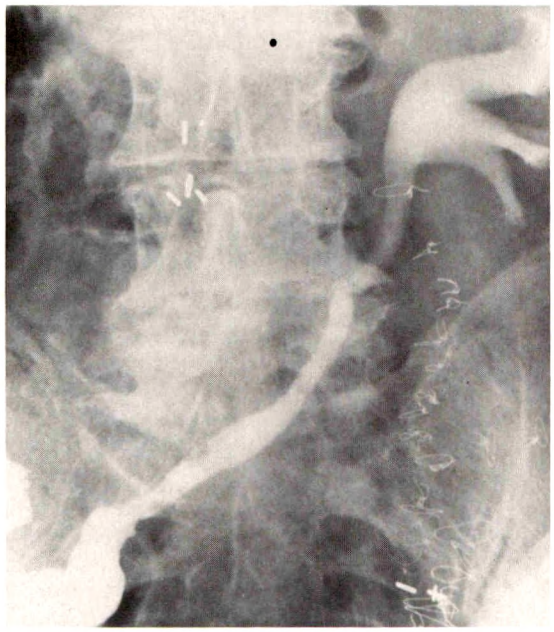

FIG. 11. A loopogram performed four and one-half years following total cystectomy shows multiple filling defects in the left lower ureter presumably due to new transitional cell carcinomas.

was noted that one-third of the patients with histological changes of carcinoma *in situ* developed another transitional cell tumor within the remaining pelvis and ureter within 18 months to three years following total cystectomy.

These cases are important, for not only do they add to our knowledge of urothelial neoplastic disease but make regular follow-up contrast material examination of the remainder of the urinary tract mandatory, particularly if at the time of cystectomy premalignant changes are found elsewhere. In addition, the unwary could misinterpret roentgenological features due to new tumor as those of calculous disease or inflammation and thus delay further surgery.

In this series no deterioration was observed in the kidney which was normal preoperatively for as long as seven years, although the majority were followed for only one to three years. These kidneys are subjected to invariable reflux from the ileal conduit which contains pathogenic bacteria in a high proportion of patients.[4]

The question of whether the reflux of infected urine can be followed by pyelonephritic scarring of the growing kidney excites controversy and is incompletely solved.[20] In adults the evidence is also inconclusive. Nonspecific roentgenological deterioration has been reported in some adults with ureteric reflux following bladder irradiation,[24] and roentgenological pyelonephritis has been recorded in three patients following surgery in whom there was reflux of infected urine.[8] However, in this series there was no deterioration, and in addition a group of adults with ileal "ureters" where there is inevitable reflux of infected urine showed no loss of renal substance or alteration in the calyceal architecture.[9] None of the patients in our series had an episode of acute pyelone-

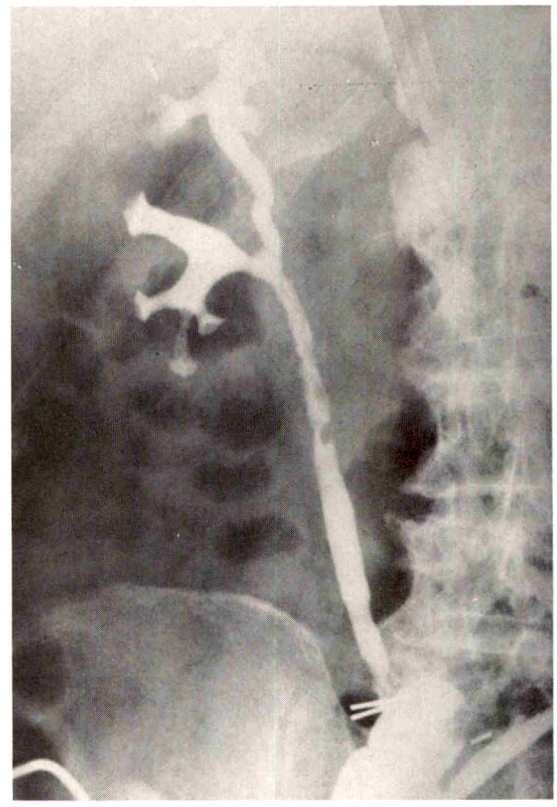

FIG. 12. A loopogram demonstrates bilateral reflux. There are many nodular filling defects in the right ureter due to multiple transitional cell carcinomata.

phritis clinically, although even in this situation roentgenological signs are seen infrequently in the follow-up pyelogram.[15]

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REFERENCES

1. Bricker, E. M. Bladder substitution after pelvic evisceration. *Surg. Clin. North America*, 1950, *30*, 1511–1521.
2. Burnham, J. P., and Farrer, J. Group experience with ureteroilial cutaneous anastomosis for urinary diversion. *J. Urol.*, 1970, *83*, 622–629.
3. Butcher, H. R., Sugg, W. L., McAfee, C. A., and Bricker, E. M. Ileal conduit method of ureteral urinary diversion. *Ann. Surg.*, 1962, *156*, 682–691.
4. Campbell, J. E., Oliver, J. A., and McKay, D. E. Dynamics of ileal conduits. *Radiology*, 1965, *85*, 338–342.
5. Dretler, S. P. Pathogenesis of urinary tract calculi occurring after ileal conduit diversion. *J. Urol.*, 1973, *109*, 204–209.
6. Ellis, L. R., Udall, D. A., and Hodges, C. R. Further clinical experiences with intestinal segment for urinary diversion. *J. Urol.*, 1971, *105*, 354–357.
7. Engle, R. M. Complications of bilateral uretero-ileal cutaneous urinary diversion: review of 208 cases. *J. Urol.*, 1969, *101*, 508–512.
8. Fritjofsson, A., and Sundin, T. Studies of renal function in vesico-ureteric reflux. *Brit. J. Urol.*, 1966, *38*, 445–452.
9. Fritzsche, P. Goodwin, W. E., Skinner, D. G., Craven, J. D., and Cahill, P. Long-term radiographic changes of kidney following "ileal ureter operation." To be published.
10. Harbach, L. B., Hall, R. L., Cockett, A. T. K., Kaufman, J. J., Martin, D. C., Mims, M. M., and Goodwin, W. E. Ileal-loop cutaneous urinary diversion: critical review. *J. Urol.*, 1971, *105*, 511–515.
11. Hodson, C. J., and Craven, J. D. Radiology of obstructive atrophy of kidney. *Clin. Radiol.*, 1966, *17*, 305–320.
12. Jaffe, B. M., Bricker, E. M., and Butcher, H. R. Surgical complications of ileal segment urinary diversion. *Ann. Surg.*, 1968, *167*, 367–376.
13. Jude, J. R., Lusted, L. B., and Smith, R. R. Radiographic evaluation of urinary tract following diversion to ileal bladder. *Cancer*, 1959, *12*, 1134–1141.
14. Koehler, P. R., Bowles, W. T., and McAlister, W. H. Roentgenographic evaluation of late results of ileal-loop urinary diversion in infants and children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 177–185.
15. Little, P. J., McPherson, D. R., and De Wardner, H. E. Appearance of intravenous pyelogram during and after acute pyelonephritis. *Lancet*, 1965, *1*, 1186–1188.
16. Parkhurst, E. C. Experiences with more than 500 ileal conduit diversions in a 12-year period. *J. Urol.*, 1968, *99*, 434–436.
17. Retik, A. B., Perlmutter, A. D., and Gross, R. E. Cutaneous ureteroileostomy in children. *New England J. Med.*, 1967, *277*, 217–222.
18. Schmidt, J. D., Hawtrey, C. E., Flocks, R. H., and Culp, D. A. Complications, results, and problems of ileal conduit diversions. *J. Urol.*, 1973, *109*, 210–216.
19. Sherwood, T. Upper urinary tract tumors following on bladder carcinoma. *Brit. J. Radiol.*, 1971, *44*, 137–141.
20. Sherwood, T. Ureteric reflux: chronic pyelonephritis vs. reflux nephropathy. *Brit. J. Radiol.*, 1973, *46*, 653–654.
21. Skinner, D. G., Richie, J. P., Cooper, P. M., Waisman, J., and Kaufman, J. J. Clinical significance of carcinoma *in situ* of bladder and its association with overt carcinoma. *J. Urol.*, 1974, *112*, 68–71.
22. Smith, E. D. Follow-up studies on 150 ileal conduits in children. *J. Pediat. Surg.*, 1972, *7*, 1–10.
23. Stamey, T. A., and Scott, W. W. Uretero-ileal anastomosis. *Surg., Gynec. & Obst.*, 1957, *104*, 11–24.
24. Williams, G., Wallace, D. M., Bloom, M. J. G., and Stevenson, J. J. Vesico-ureteric reflux following irradiation of urinary bladder. *Proc. Roy. Soc. Med.*, 1971, *64*, 64–66.

THE BLEEDING MARGINAL ULCER*

CATHETERIZATION DIAGNOSIS AND THERAPY

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ABSTRACT:

Superior mesenteric arteriography can often demonstrate actively bleeding marginal ulcers. Five cases diagnosed by angiography are reported.

Pitressin infusions of the superior mesenteric artery stopped bleeding permanently in two cases, transiently in two cases, and was not attempted in one case. Pitressin infusions of the superior mesenteric artery should be attempted before surgery is performed for bleeding marginal ulcers.

THE diagnosis and therapy of gastrointestinal bleeding by catheterization techniques have made great advances since their introduction in the beginning of the last decade. However, no previous studies have specifically focused on the evaluation and treatment of bleeding marginal ulcers.

We have had experience with five cases in which angiography was successfully employed in visualizing the bleeding site in the jejunum.

Pitressin infusion of the superior mesenteric artery was utilized in four cases, with permanent cessation of bleeding in two cases and with transient control of bleeding in two cases.

MATERIAL AND METHOD

The patients were studied by the standard Seldinger technique. The bleeding site in the proximal jejunum was identified *via* selective superior mesenteric artery injection. The catheter was left in place for pitressin infusion in four of the five patients. The rate of infusion initially was 0.2 units per minute and was increased to 0.4 units on one occasion.

REPORT OF CASES

CASE I. A 92 year old man was admitted to Montefiore Hospital and Medical Center (MHMC) with melena and epigastric pain. His hematocrit was 13 percent. A partial gastrectomy with gastrojejunostomy had been performed for duodenal ulcer disease ten years prior to admission. The patient also had a history of myocardial infarction two years previously.

Emergency angiography with a selective superior mesenteric injection revealed a bleeding site in the upper abdomen considered to represent a marginal ulcer (Fig. 1). A pitressin infusion was started and bleeding stopped abruptly. Follow-up for two months revealed no evidence of subsequent bleeding.

CASE II. This 55 year old white male with widespread metastatic disease from bladder carcinoma was admitted to MHMC with black, tarry stools for two days. He had had a partial gastrectomy and gastrojejunostomy 20 years earlier for duodenal ulcer disease.

The hematocrit on admission was 24 percent. Nasogastric aspiration revealed bright red blood and coffee ground like material. Endoscopy showed active bleeding which appeared to be originating from the efferent loop and refluxing into the stomach. No definite ulcer niche was identified. A superior mesenteric arteriogram (Fig. 2, *A* and *B*) demonstrated a bleeding site in the upper abdomen in the region of the gastrojejunostomy with contrast

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.

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material extravasating into the jejunum and from the jejunum into the stomach.

A pitressin infusion of the superior mesenteric artery was instituted. For twelve hours pitressin was infused at a rate of 0.2 units per minute. No significant change in the bleeding occurred and the pitressin infusion was increased to 0.4 units per minute with the infusion continued at this rate for an additional six hours. The bleeding stopped and the infusion was maintained at a rate of 0.1 units per minute for an additional twelve hours, after which the catheter was withdrawn. Standard oral medical therapy for peptic ulcer disease was continued. No further gastrointestinal bleeding occurred. During the bleeding episode the patient had received four units of packed red blood cells and one unit of fresh frozen plasma.

The patient died one month after admission from his bladder carcinoma. Autopsy showed no evidence of an active mucosal ulceration at the gastrojejunostomy. Slight telangiectasia was noted at the efferent loop.

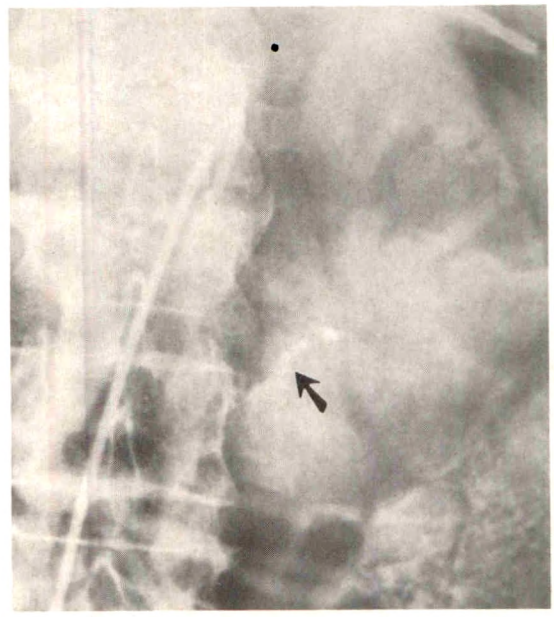

Fig. 1. Case I. Late venous phase of superior mesenteric arteriogram demonstrates extravasation of contrast material (arrow) in the upper abdomen representing a bleeding marginal ulcer.

Case III. A 40 year old woman was admitted to MHMC for severe epigastric pain. A subtotal gastrectomy with gastrojejunostomy for carcinoid tumor of the stomach had been performed one year prior to admission. One week following admission the patient developed rectal bleeding with a fall in hematocrit from 37 percent to 19 percent. Emergency superior mesenteric angiography revealed a bleeding site in the proximal jejunum at the level of the gastrojejunostomy. A pitressin infusion was started and the bleeding subsided. Twenty-four hours later, when the pitressin infusion was stopped, the patient started to bleed abruptly. The pitressin infusion was started again and the bleeding stopped. At this time the patient was operated upon because of her stable condition and the considered likelihood of rebleeding. A

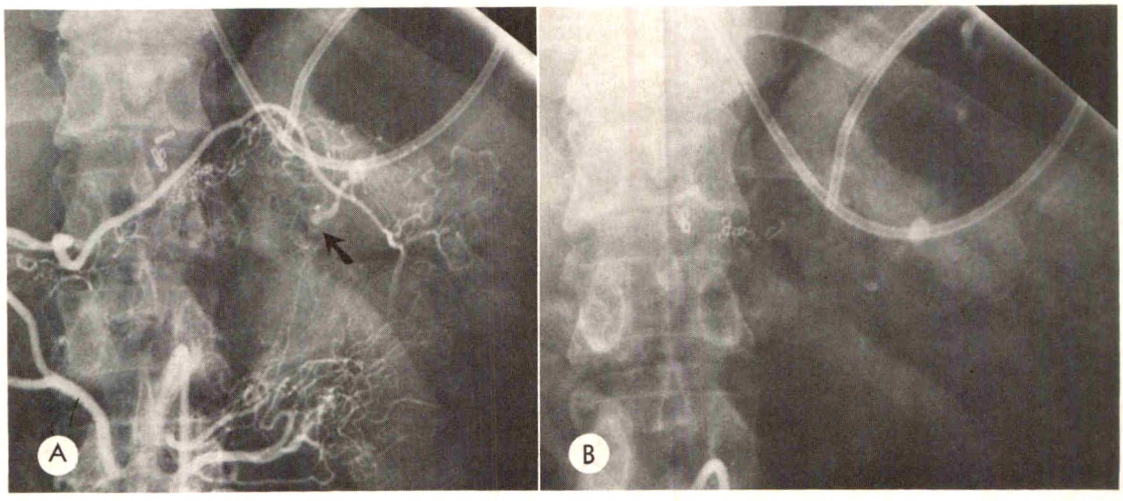

Fig. 2. Case II. (*A*) Mid arterial phase of superior mesenteric arteriogram shows contrast material extravasation in the left upper quadrant from a jejunal artery (arrow). (*B*) Late venous phase of superior mesenteric arteriogram shows several distinct areas of pooled contrast material in the stomach and jejunum.

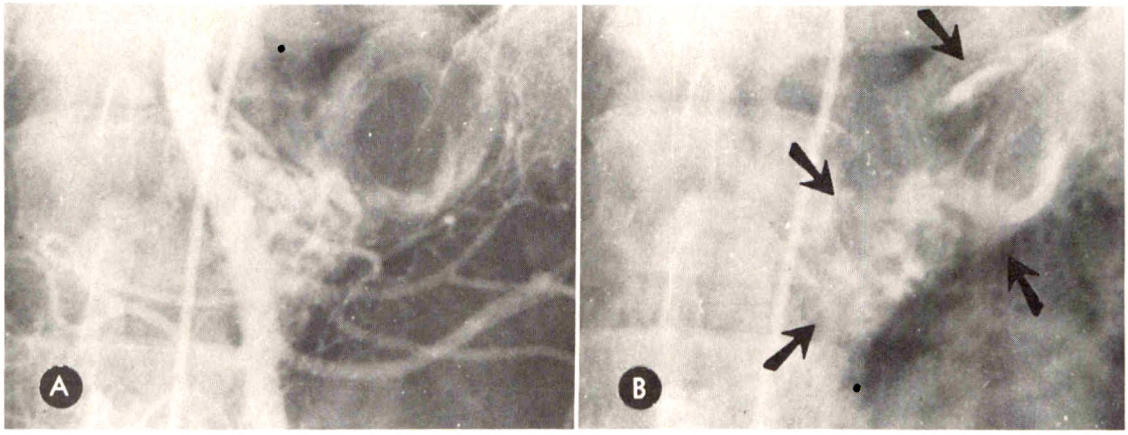

FIG. 3. Case IV. Mid arterial (*A*) and (*B*) late venous phase of an emergency superior mesenteric arteriogram demonstrate massive extravasation of contrast material in the region of the proximal jejunum (arrows in *B*).

marginal ulcer was found at operation.

CASE IV. A 62 year old man was admitted to MHMC for massive rectal bleeding. The patient underwent partial gastrectomy and gastrojejunostomy 14 months previously for duodenal ulcer disease.

On the day of admission the hematocrit dropped from 33 percent to 16 percent. Emergency superior mesenteric angiography revealed contrast material extravasation in the proximal jejunum (Fig. 3, *A* and *B*). A pitressin infusion was started and the bleeding stopped transiently. Several hours later hemorrhage increased while the pitressin infusion was running. The pitressin dose was not increased above 0.2 units per minute. Operation revealed a bleeding marginal ulcer. The hematocrit at the time of surgery was 29 percent.

CASE V. A 65 year old woman was admitted to MHMC for hematemesis. The patient had undergone a gastrojejunostomy for carcinoma of the head of the pancreas seven months prior to admission. The patient's hematocrit on admission was 28 percent. Emergency angiography with selective superior mesenteric injection demonstrated a bleeding site in the proximal jejunum. No attempt to stop the bleeding *via* pitressin infusion was made. The patient underwent surgery and a marginal ulcer was resected.

DISCUSSION

The introduction of emergency angiography for the diagnosis of acute gastrointestinal bleeding was first described by Baum and Nusbaum in 1963 and 1965.[4,7,8] Subsequent articles [1–3,5,6,9,11,12] attest to the value of angiography in the diagnosis of lesions causing upper gastrointestinal bleeding, many of which could not be detected by barium studies nor at operation.[3,6] Few cases of marginal ulcers diagnosed by angiography are to be found in the literature.[3,11] In 1968 Nusbaum *et al.*[9] described pitressin infusion into the superior mesenteric artery as a successful means of stopping variceal bleeding in the majority of patients. Following this, the use of vasoconstricting drugs (epinephrine and pitressin) was attempted in the management of additional causes of upper gastrointestinal bleeding.[1–3,5,6,10–12]

Good results were obtained in treatment of bleeding due to certain superficial lesions such as are found in gastritis and Mallory-Weiss syndrome.[10,11] The treatment of peptic ulcer disease was, however, disappointing. Rösch and associates[10] failed to achieve complete control of bleeding in eleven of thirteen cases.

In the clinical experience with catheter directed drug treatment of upper gastrointestinal bleeding, the marginal ulcer has received little comment.[11] To date, no study has focused on the angiographic diagnosis and catheter directed therapy of marginal

ulcers, which are characteristically located on the jejunal side of the anastomosis. To emphasize the importance of this lesion, one has to consider that the most common operation for peptic ulcer disease is gastrojejunostomy. Up to eight percent of these patients develop marginal ulcers,[15,16] and almost 50 percent of these ulcers produce gastrointestinal hemorrhage.[14]

As already emphasized by Rösch and associates,[10,11] the selectivity of the infusion is of great importance in determining success or failure of the therapy by vasoconstrictors. Marginal ulcers are particularly suited for such a direct infusion because of the ease of catheterization of the superior mesenteric artery which directly feeds the bleeding site. The favorable response of bleeding marginal ulcers to pitressin infusion as demonstrated in our series is also most likely related to the tendency of the ulcers to be superficial and, as such, to be amenable to induction of coagulation.

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REFERENCES

1. Baum, S., and Nusbaum, M. Control of gastrointestinal hemorrhage by selective mesenteric arterial infusion of vasopressin. *Radiology*, 1971, *98*, 497–505.
2. Baum, S., Nusbaum, M., Blakemore, W. A., and Finkelstein, A. K. Preoperative radiographic demonstration of intra-abdominal bleeding from undetermined sites by percutaneous selective celiac and superior mesenteric arteriography. *Surgery*, 1965, *58*, 797–805.
3. Baum, S., Nusbaum, M., and Turner, H. J. Control of gastrointestinal hemorrhage by selective mesenteric arterial infusions of pitressin. *Gastroenterology*, 1970, *58*, 926–933.
4. Baum, S., Roy, R., Finkelstein, A. K., and Blakemore, W. S. Clinical application of selective celiac and superior mesenteric arteriography. *Radiology*, 1965, *84*, 279–294.
5. Frey, C. F., Renter, S. R., and Bookstein, J. J. Localization of gastrointestinal hemorrhage by selective angiography. *Surgery*, 1970, *67*, 548–555.
6. Koehler, P. R., and Salmon, R. B. Angiographic localization of unknown acute gastrointestinal bleeding sites. *Radiology*, 1967, *89*, 244–249.
7. Nusbaum, M., and Baum, S. Radiographic demonstration of unknown sites of gastrointestinal bleeding. *S. Forum*, 1963, *14*, 374–375.
8. Nusbaum, M., Baum, S., Blakemore, W. S., and Finkelstein, A. K. Demonstration of intra-abdominal bleeding by selective arteriography: visualization of celiac and superior mesenteric arteries. *J.A.M.A.*, 1965, *191*, 389–390.
9. Nusbaum, M., Baum, S., Kuroda, K., and Blakemore, W. S. Control of portal hypertension by selective mesenteric arterial drug infusion. *A.M.A. Arch. Surg.*, 1968, *97*, 10005–10012.
10. Rösch, J., Dotter, C. T., and Antonovic, R. Selective vasoconstrictor infusion in management of arterio-capillary gastrointestinal hemorrhage. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 279–288.
11. Rösch, J., Dotter, C. T., and Rose, R. W. Selective arterial infusions of vasoconstrictors in acute gastrointestinal bleeding. *Radiology*, 1971, *99*, 27–36.
12. Rösch, J., Gray, R. K., and Grollman, J. W., Jr. Selective arterial drug infusions in treatment of acute gastrointestinal bleeding. *Gastroenterology*, 1970, *59*, 341–349.
13. Vargha, J. Funktionelle Roentgenuntersuchungen der Anastomose bei Anastomosenkarzinomen. *Radiol. Diagn.* (Berlin), 1962, *7*, 215–227.
14. Walters, W. Six to ten-year follow-up of surgical treatment of duodenal gastric and gastrojejunal ulcers. *Gastroenterologia* (Basel), 1960, *93*, 15–23.
15. Walters, W., Priestley, J. T., and Balding, H. H. Vagotomy in treatment of gastrojejunal ulcerations: postoperative clinical and laboratory study. *J.A.M.A.*, 1952, *148*, 803–811.
16. Weir, J. F., and Bennet, H. S. Peptic ulcer: follow-up study after partial gastrectomy. *Proc. Staff Meet. Mayo Clin.*, 1956, *31*, 632–641.

THE SIGNIFICANCE OF THE DURAL SUPPLY FROM THE CAROTID SIPHON*

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ABSTRACT:

The dural branches from the cavernous portion of the internal carotid consists of the meningohypophyseal trunk, the inferior cavernous, and the capsular arteries. These vessels have extensive anastomoses in and around the sellar area. These arteries are not usually demonstrated angiographically; therefore, when they are enlarged, a pathological lesion should be suspected. There are numerous lesions other than meningiomas associated with hypertrophy of these dural branches and the corresponding vessels involved depends on the anatomic location of the associated lesion.

IN 1956 Bernasconi and Cassinari[1] described a tentorial meningioma supplied by dural branches, which they considered to originate from the external carotid artery. At that time they angiographically demonstrated tentorial arteries in five of seven tentorial meningiomas and considered the visualization of these tentorial vessels specific for meningiomas. In 1958 Wickbom and Stattin[12] demonstrated that these arteries are actually dural branches of the cavernous portion of the internal carotid arteries. Since that time there has been demonstration of lesions other than tentorial meningiomas associated with hypertrophy of these dural arteries.[2,4,8–11] Some of these are arteriovenous malformations, angiomas, gliomas invading the tentorium, trigeminal neurinomas, pituitary tumors, hypothalamic tumors, and occlusive vascular lesions. The vascular anatomic aspects of the carotid siphon have been discussed by Parkinson.[7] Our intention in this presentation is to discuss further the anatomy of the dural supply of the carotid siphon (Fig. 1, *A* and *B*) and to illustrate the significance of angiographic visualization of its branches.

ANATOMIC FEATURES

The first branch originating from the cavernous portion of the internal carotid

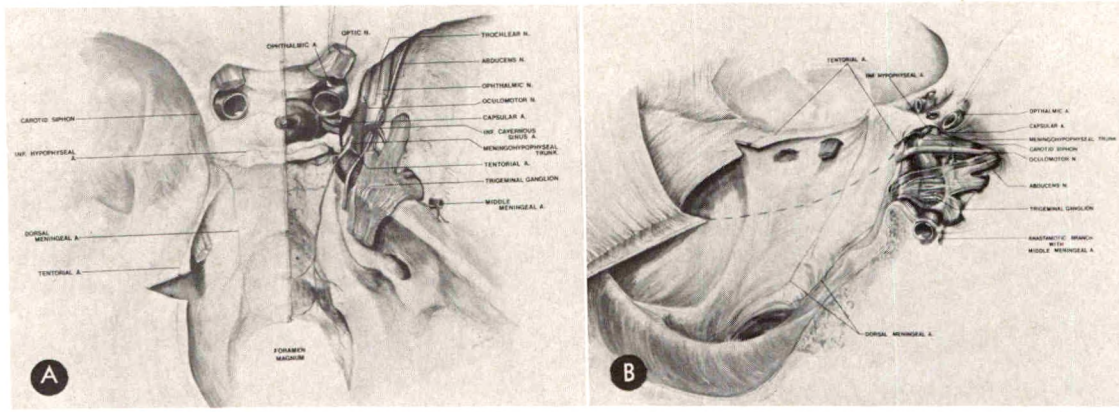

FIG. 1. (*A* and *B*) Anatomic presentation of the dural branches from the cavernous portion of the internal carotid with demonstration of their anastomoses and areas of supply.

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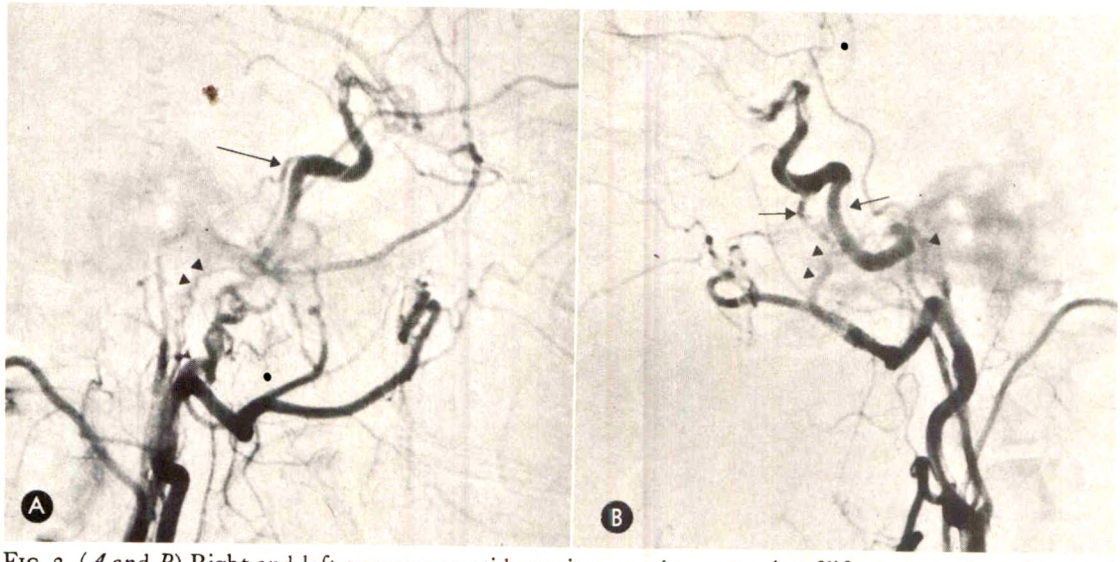

Fig. 2. (*A* and *B*) Right and left common carotid arteriograms demonstrating filiform narrowing of internal carotids from their origin to foramen lacerum. Note multiple dural branches anastomosing the internal (arrows) and external carotid (arrowheads) systems.

artery is the meningohypophyseal trunk which usually trifurcates into three branches: the tentorial, the dorsal meningeal, and the inferior hypophyseal arteries. The meningohypophyseal trunk arises from the vertical segment of the cavernous portion of the internal carotid artery, shortly after it enters the cavernous sinus. It courses in a posterolateral direction, gives off branches which supply the third and fourth cranial nerves, and anastomoses with ophthalmic meningeal and ascending pharyngeal branches of the external carotid artery. The tentorial artery courses posteriorly giving off branches to the tentorium and posterior portion of the falx cerebri, branching to join with the tentorial artery from the opposite side. The second branch of the meningohypophyseal trunk, the dorsal meningeal, leaves the trunk in a medial posteroinferior direction to supply the dura of the clivus anastomosing with its counterpart from the opposite side. It may anastomose with branches of the vertebral and cervical arteries. The third branch of the meningohypophyseal trunk is the inferior hypophyseal artery. This vessel leaves in an anteromedial direction and supplies portions of the pituitary gland, the contents of the cavernous sinus, and the diaphragm and floor of the sella. It also anastomoses with its counterpart from the opposite side.

A second branch, from the cavernous portion of the internal carotid artery, is the artery of the inferior cavernous sinus which originates from the inferolateral aspect. It supplies the dura of the inferior portion of the cavernous sinus and the Gasserian

Fig. 3. A 50 year old female with a recurrent sphenoidal wing meningioma with extension to the anterior and middle cranial fossae. Note hypertrophy of the dural branches from the siphon (small arrows) and the ophthalmic artery (large arrows) supplying the lesion.

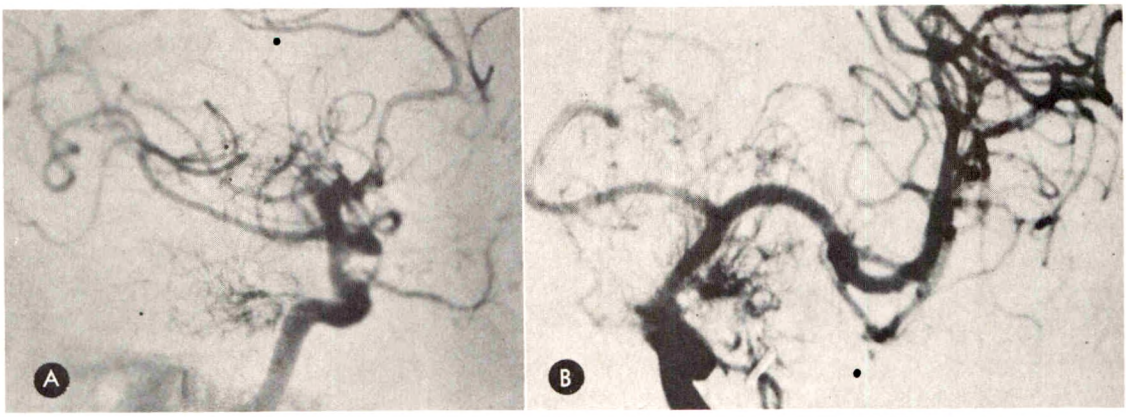

FIG. 4. (*A*, and *B*) This demonstrates a meningioma of the clivus in a 47 year old male. Note the numerous dural branches extending posteriorly from the siphon.

FIG. 5. Demonstration of *en plaque* meningioma involving the entire petrous bone extending into the tympanic cavity in a 43 year old female with left sixth nerve palsy and hearing loss. Note dural branches (arrows).

FIG. 6. A 50 year old female with a large parasellar meningioma with extension to the cavernous sinus, and middle and posterior fossae. Note the extensive dural supply (arrows).

ganglion. Near the foramen spinosum it may anastomose with the middle meningeal and the accessory middle meningeal branches.

The third branch to arise from the cavernous portion of the internal carotid is the capsular artery of McConnel[5] which has an inferomedial origin and anastamoses with its counterpart from the opposite side. The existence of the capsular artery is variable as well as its origin which may be from the cavernous portion of the internal carotid or from the inferior hypophyseal artery.

DISCUSSION

These vessels are normally present but are seldom demonstrated angiographically.

FIG. 7. Hypertrophy of the dorsal meningeal and tentorial arteries (arrows) supplying a cerebellopontine angle meningioma.

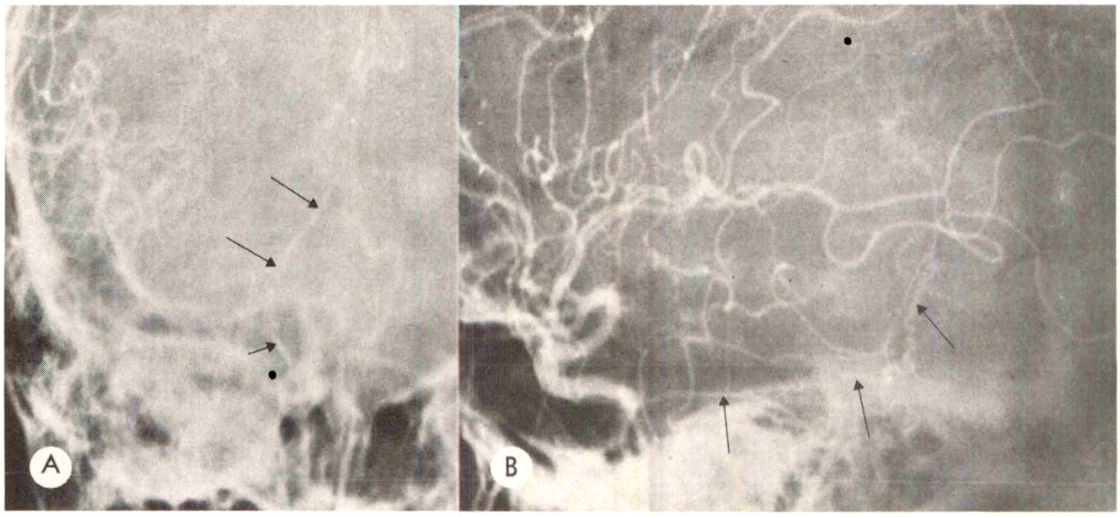

Fig. 8. (*A* and *B*) This shows the typical appearance of the artery of Bernasconi and Cassinari (arrows) in a tentorial meningioma.

In a study by Hatam[3] the incidence of filling of the cavernous branches in 100 normal angiograms was distributed as follows: meningohypophyseal trunk 12 percent; inferior cavernous sinus artery four percent and the capsular artery zero percent. However, the incidence of visualization may vary considerably with selective carotid angiography and with the aid of subtraction technique. Since the vessels may be visualized in normal angiograms, certain criteria should be used to evaluate their significance. Hypertrophy and elonga-

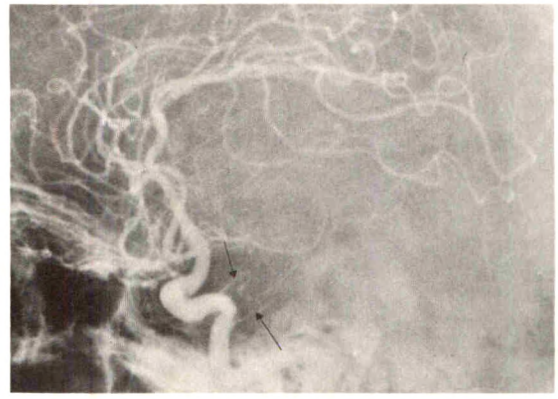

Fig. 9. Metastatic temporal lobe mass with parasellar extension supplied by dural branches (arrows).

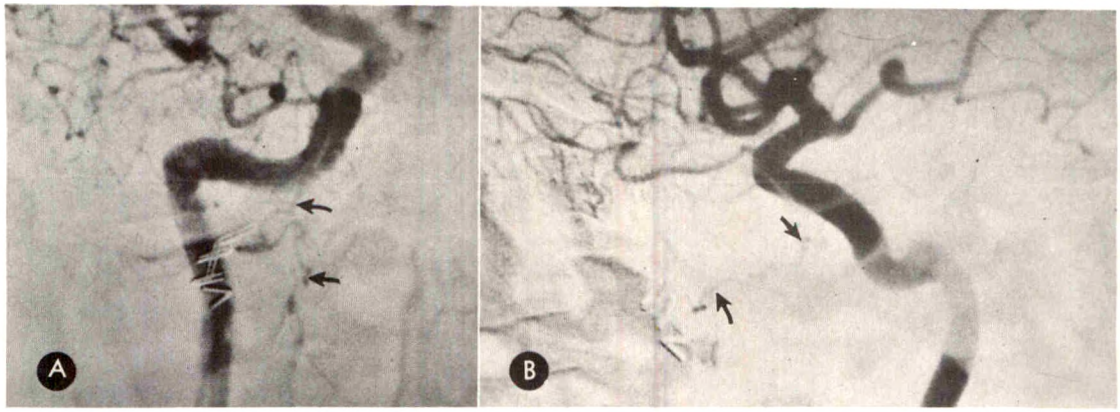

Fig. 10. (*A* and *B*) Dural branches (arrows) supplying large recurrent nasopharyngeal carcinoma with massive extension into the base of the skull.

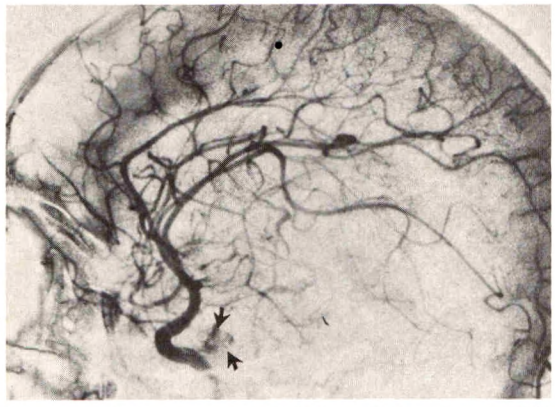

FIG. 11. Large temporal lobe glioblastoma with parasellar extension which is indicated by hypertrophy of dural branches (arrows).

FIG. 12. Dural branches from the meningohypophyseal trunk (arrow) feeding a chromophobe adenoma with suprasellar extension.

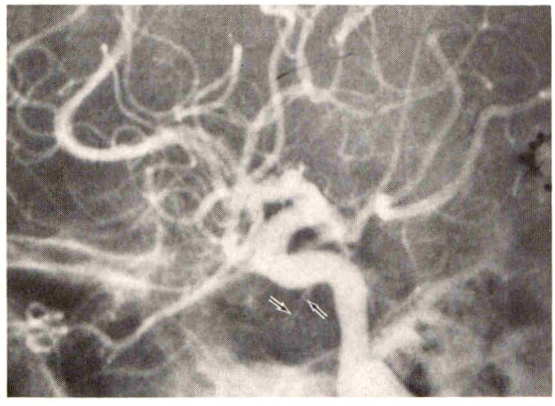

FIG. 13. Lateral extension of a large chromophobe adenoma. Note dural supply (arrows) by the inferior cavernous sinus artery and superior and posterior displacement of siphon.

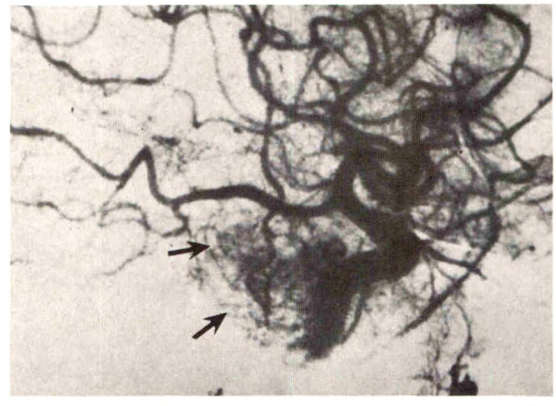

FIG. 14. Trigeminal neurinoma profusely supplied by dural branches (arrows) from the carotid siphon.

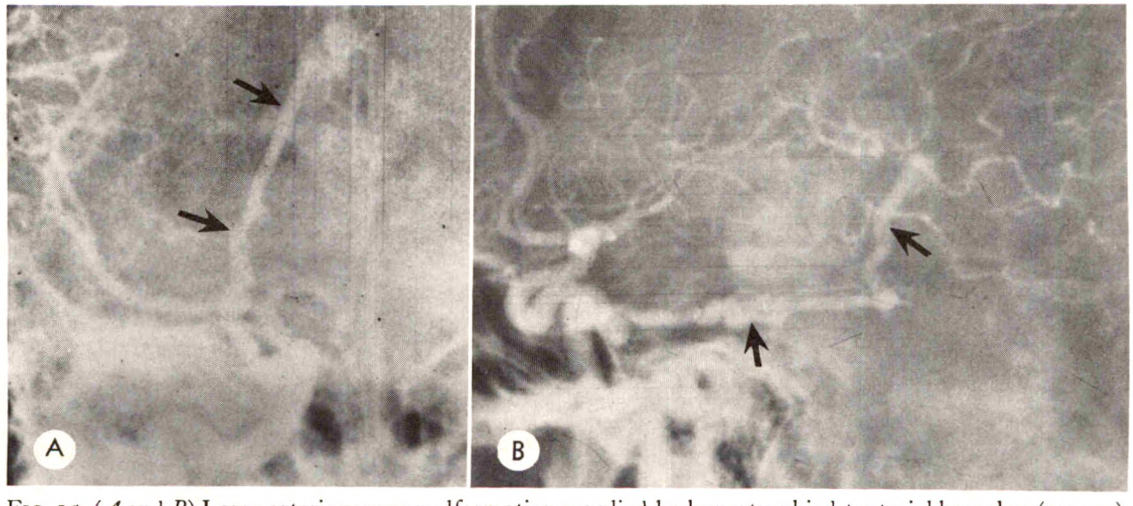

FIG. 15. (*A* and *B*) Large arteriovenous malformation supplied by hypertrophied tentorial branches (arrows). Similar changes were noted on opposite side. Note drainage into the great vein of Galen.

tion of the vessel[9] and association with tumor vessels, tumor blushes, and early venous filling are important criteria for evaluation. These dural branches from the carotid siphon may become hypertrophied in lesions which are anatomically located in the region which these vessels supply. Due to their extensive anastomoses, these vessels may also become hypertrophied as a result of collateral circulation. There has been reference to their importance in collateral circulation by Stattin[10] and Kramer and Newton[4] in cases of thrombosis and berry aneurysm of the middle cerebral and also in occlusive disease of the carotid arteries; however, no examples were given. Figure 2, *A* and *B*, demonstrates a filiform narrowing of both internal carotid arteries resulting in a collateral circulation *via* the external carotid to the dural branches of the cavernous portion of the internal carotid artery.

In the literature, most cases that have demonstrated hypertrophy of the dural arteries have been meningiomas and indeed this should be the first consideration when there is hypertrophy of the vessels. We have demonstrated seven cases of meningiomas in different locations relative to the origin of the dural branches of the internal carotid artery. Examples include a sphenoidal wing meningioma with extension to the anterior and middle cranial fossae (Fig. 3), a meningioma of the clivus (Fig. 4, *A* and *B*), a meningioma *en plaque* extending into the tympanic cavity (Fig. 5), a meningioma with extension to the middle and posterior fossae and cavernous sinus (Fig. 6), a cerebellopontine angle meningioma (Fig. 7), and a tentorial meningioma (Fig. 8, *A* and *B*).

Hypertrophy of the dural vessels may also occur in association with lesions that secondarily invade the dura. Examples included here are metastatic carcinoma (Fig. 9), a nasopharyngeal carcinoma (Fig. 10, *A* and *B*), and a glioblastoma (Fig. 11). These examples of secondary involvement of the meninges and those of primary meningeal involvement demonstrate that pathological lesions involving

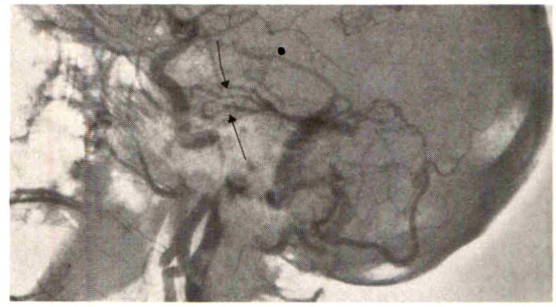

Fig. 16. Arteriovenous malformation draining into transverse and sigmoid sinuses. Note dural branches (arrows).

an area normally supplied by the dural arteries alter flow to that area and cause hypertrophy of the supplying vessels.

As demonstrated in Figure 1*A*, and *B*, there are other structures, besides the dura, supplied by dural branches of the internal carotid artery. When these structures become pathological so as to require increased blood supply, the flow to those areas increases and hypertrophy of the supplying vessels occurs. Examples of such lesions may include chromophobe adenoma (Fig. 12; and 13) and trigeminal neurinoma (Fig. 14).

Vascular malformations have been discussed extensively by Newton and Hoyt[6] and are another cause of hypertrophy of the dural arteries. Figure 15, *A* and *B*, is an example of an arteriovenous malformation extending to the great vein of Galen, and Figure 16 shows a posterior fossa arteriovenous malformation fed by multiple dural branches.

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REFERENCES

1. Bernasconi, V., and Cassinari, V. Un segno carotidografico tipico di meningioma del tentorio. *Chirurgra*, (Milan), 1956, *11*, 586–588.
2. Cortes, O., Chase, N. E., and Leeds, N. Visualization of tentorial branches of internal carotid artery in intracranial lesions other than meningiomas. *Radiology*, 1964, *82*, 1024–1028.
3. Hatam, A. Vascular supply of eosinophilic and mixed pituitary adenomas in acromegalic

patients. *Neuradiology*, 1971, *3*, 4–7.
4. KRAMER, R., and NEWTON, T. H. Tentorial branches of internal carotid artery. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *95*, 826–830.
5. MCCONNELL, E. M. Arterial blood supply of human hypophysis cerebri. *Acta radiol.*, 1961, *55*, 329–336.
6. NEWTON, T. H., and HOYT, W. F. Dural arteriovenous shunts in region of cavernous sinus. *Neuroradiology*, 1970, *1*, 71–81.
7. PARKINSON, D. Collateral circulation of cavernous carotid artery: anatomy. *Canad. J. Surg.*, 1964, *7*, 151–158.
8. PRIBRAM, H. F. W., BOULTER, T. R., and MCCORMICK, W. F. Roentgenology of meningohypophyseal trunk. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *98*, 583–594.
9. SCHNÜRER, L.-B., and STATTIN, S. Vascular supply of intracranial dura from internal carotid artery with special reference to its angiographic significance. *Acta radiol.* (Diag.), 1963, *1*, 441–450.
10. STATTIN, S. Memingeal vessel of internal carotid artery and their angiographic significance. *Acta radiol.*, 1961, *55*, 329–336.
11. WESTBERG, G. Angiographic changes in neurinoma of trigeminal nerve. *Acta radiol.* (Diag.), 1963, *1*, 513–520.
12. WICKBOM, I., and STATTIN, S. Roentgen examination of intracranial meningiomas. *Acta radiol.*, 1958, *50*, 175–186.

Vol. 125, No. 4

INTRACRANIAL HEMANGIOPERICYTOMAS*

ANGIOGRAPHY, PATHOLOGY AND DIFFERENTIAL DIAGNOSIS

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ABSTRACT:

Intracranial hemangiopericytomas are tumors which angiographically and histologically can mimic other vascular intracranial masses. The term angioblastic meningioma has been applied to this tumor and to other vascular tumors. However, hemangiopericytomas have a unique biological behavior and prognosis as they tend to recur or metastasize and are insensitive to radiation. Hence, the importance of their specific recognition is emphasized. This is a report of the roentgenologic and pathologic findings in five cases of histologically verified intracranial hemangiopericytomas. The angiographic findings common to these tumors include a myriad of tiny irregular feeding vessels springing from a main trunk, an intense fluffy type of stain, lack of early veins, and prolonged tumor circulation time. The major arterial supply is from the internal carotid or vertebral circulation rather than from external (meningeal) vessels. Differentiation from other vascular tumors particularly supratentorial hemangioblastoma, glioblastoma multiforme, as well as from vascular meningothelial meningioma is important from both the angiographic and histologic aspects.

WICKBOM[16] in 1953 stated "the possibility of establishing a differential diagnosis of tumors by angiography is mainly based on the fact that many tumors have a vascular supply and architecture different in appearance from the vascularity of the normal brain tissue."

Among the vascular intracranial neoplasms, hemangiopericytomas are sufficiently unique in their angiographic manifestations that a careful analysis of the findings should permit at least a high suspicion of their presence. The distinction of these tumors from other types of meningeal tumors is an important one as hemangiopericytomas have a high tendency to recur and sometimes to metastasize.[8,9,10] Angiographically, they can resemble supratentorial hemangioblastomas, glioblastoma multiforme or some metastatic tumors. Nevertheless, unlike glioblastomas and some metastatic lesions they are insensitive to radiation. Surgical extirpation is the definitive treatment, and long term follow-up should be the rule. These facts make obvious the importance of their recognition by both the radiologist and the pathologist. Their awareness of the entity will avoid fruitless radiation therapy and guard against delay in recognizing recurrences. The importance of recognizing these tumors along with our inability to find a reference in the English literature on the angiographic features of this neoplasm has prompted us to report our observations.

MATERIAL AND METHOD

Five cases of intracranial hemangiopericytomas which were histologically proved subsequent to the operation were

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.

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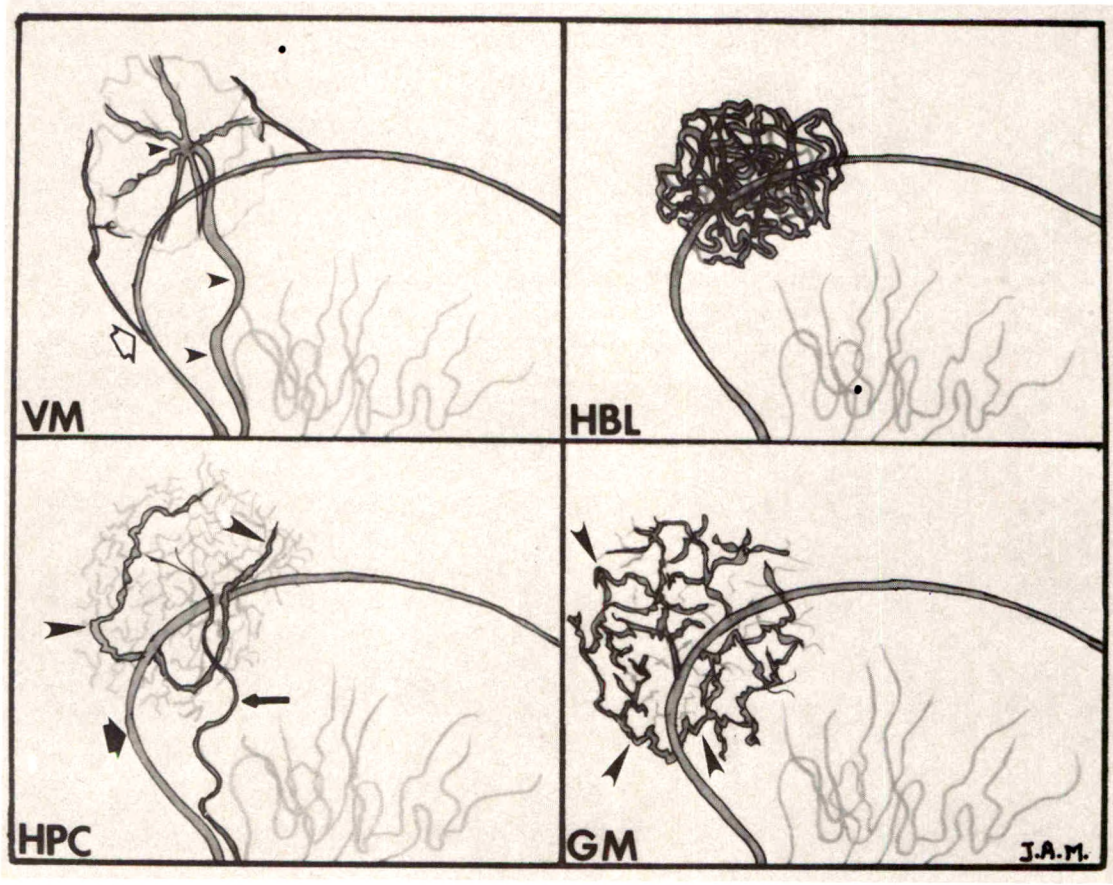

FIG. 1. A diagrammatic comparison of the arterial phases of four hypothetical tumors in the frontal area. In meningothelial, but vascular meningioma (VM), the major contribution is from the middle meningeal artery (arrowheads) which supplies the center of the tumor where it divides into several branches resembling the spokes of a wheel. A limited contribution may come from the internal carotid branches (open short arrows). In hemangiopericytoma (HPC) the major supply is from the anterior cerebral artery (short arrow) *via* a major feeder or feeders (arrowheads) from which a myriad of tiny feeders springs. Only a minor contribution from the middle meningeal occurs (arrow). In glioblastoma multiforme (GM) several large feeders (arrowheads) arise from internal carotid artery branches (anterior cerebral in this example) and there is no meningeal supply. In supratentorial hemangioblastoma (HBL) large main feeders from internal carotid artery branches are seen, but the feeders are tightly packed in the region of the tumor and microaneurysm formation is common. No meningeal artery branches supply to the tumor.

studied and analyzed angiographically. In each case, a percutaneous puncture of the carotid artery, or femoral artery catheterization, was performed. Ten cc of contrast material were used for the common carotid artery, and eight cc for either the internal carotid artery or the vertebral artery injection. Serial angiography was done in all cases on a rapid film changer at a rate of two films per second for three seconds followed by one film per second for six seconds.

RESULTS

There are similar angiographic features seen in each of our cases. These include (Fig. 1; and 2):

(1) A dual arterial supply. These tumors draw arterial supply from both internal and external carotid branches. The major feeders, however, are from internal carotid artery branches (Fig. 3, *A* and *C*; 5, *A*, *B* and *E*; 6*A*; and 7, *A* and *C*).

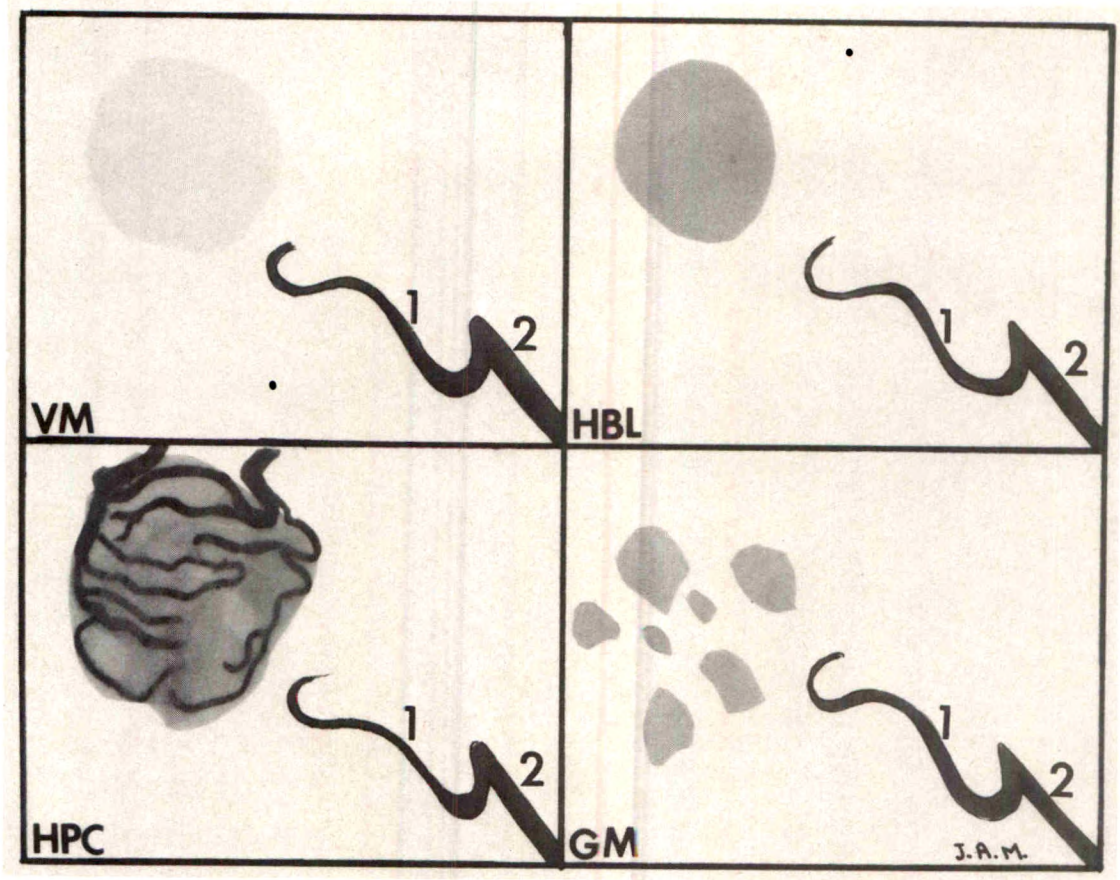

Fig. 2. A diagrammatic representation of the venous phase of the four hypothetical tumors as in Figure 1. Note the homogeneous stain of meningiomas (VM), the dense stain in hemangioblastoma (HBL), the dense stain in hemangiopericytoma (HPC) augmented by lingering of contrast material in venous channels in tumor region. In glioblastoma multiforme (GM) the stain is irregular and blotchy and there commonly is an early draining vein.
1. Internal cerebral vein.
2. Straight sinus.

(2) A main feeder or feeders from which a myriad of small corkscrew vessels arises rather than a multitude of large vessels (Fig. 3*A*; 4*A*; and 5*C*).
(3) A degree of vascular displacement somewhat less than expected from the size of the area showing tumor stain (Fig. 3*B*).
(4) A very dense tumor stain augmented by lingering of contrast material in venous channels within and adjacent to the tumor (Fig. 4*B*; 5*D*; 6*B*; and 7*B*).
(5) A slow circulation time.
(6) All are placed on the convexity, none at the base of the skull or the Sylvian region. One case is paratorcular (Case 1; Fig. 3, *A–C*).

DISCUSSION

Stout and Murray[13,14] coined the term "hemangiopericytoma" based upon their experience with a total of 34 extracranial neoplasms which led them to conclude that the typical tumor cells surrounding capillary endothelium were the pericytes of Zimmerman.[17] Reports appeared thereafter and certain characteristics of this tumor emerged. They can occur in soft tissues anywhere in the body[9,13] including the neck,

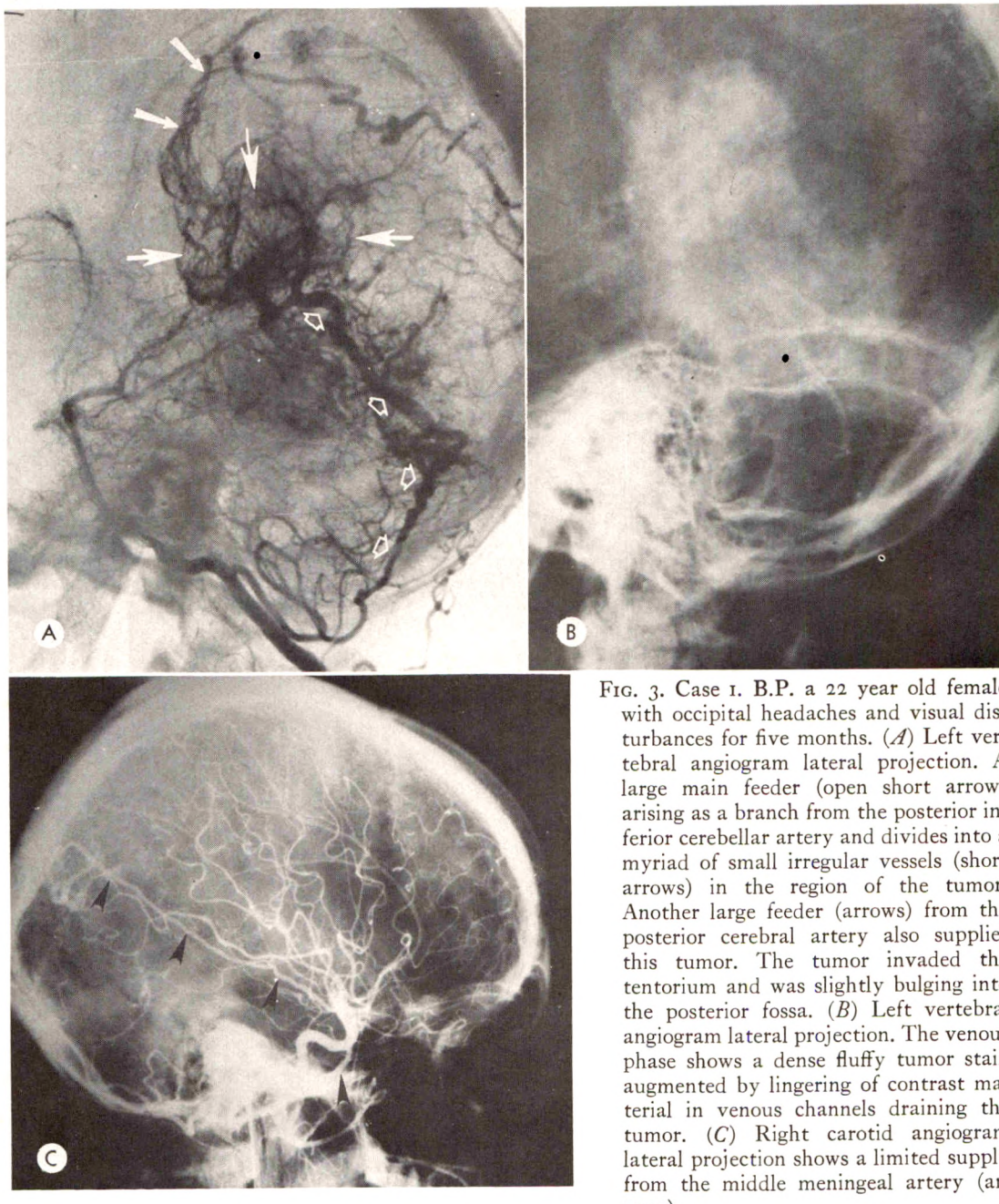

FIG. 3. Case 1. B.P. a 22 year old female with occipital headaches and visual disturbances for five months. (*A*) Left vertebral angiogram lateral projection. A large main feeder (open short arrow) arising as a branch from the posterior inferior cerebellar artery and divides into a myriad of small irregular vessels (short arrows) in the region of the tumor. Another large feeder (arrows) from the posterior cerebral artery also supplies this tumor. The tumor invaded the tentorium and was slightly bulging into the posterior fossa. (*B*) Left vertebral angiogram lateral projection. The venous phase shows a dense fluffy tumor stain augmented by lingering of contrast material in venous channels draining the tumor. (*C*) Right carotid angiogram lateral projection shows a limited supply from the middle meningeal artery (arrows).

extremities, buttock, orbit,[4] breast, uterus, scalp, or retroperitoneum, as well as intracranially arising from the meninges.[3,8] Their universal distribution is obviously related to their perivascular growth; in other words, they can occur wherever capillaries exist.

In spite of their characteristic histologic appearance, intracranial hemangiopericytomas have been called angioblastic meningioma,[1,10,15] vascular meningioma, meningeal sarcoma,[5] atypical sarcoma, or angiosarcoma. They may be mistaken for supratentorial hemangioblastoma and sometimes even misdiagnosed as glioblastoma multiforme, or a metastatic deposit. In par-

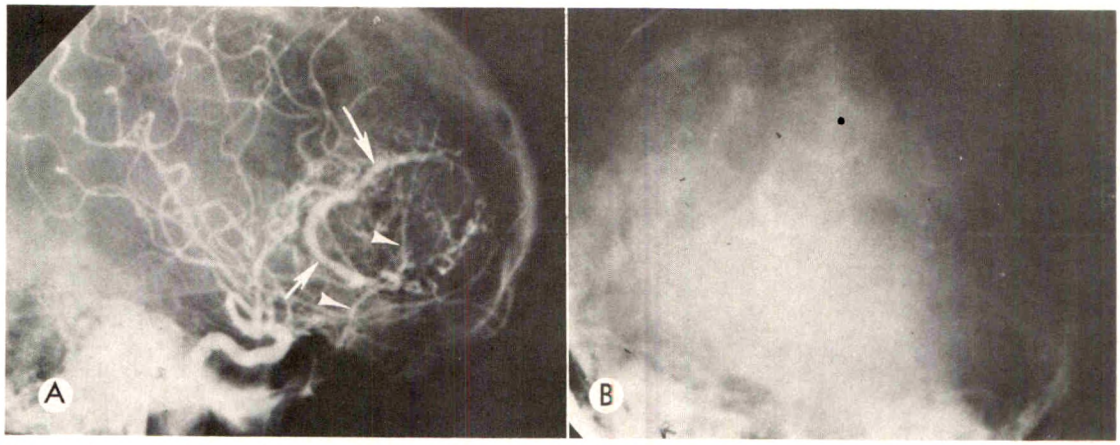

FIG. 4. Case II. O.R., a 36 year old female with six month history of gradual memory loss and personality change. Visual disturbance three months prior to admission. (*A*) Right carotid angiogram, lateral projection, shows a large main feeder (arrows) arising from the anterior cerebral artery and dividing into a myriad of small feeders to supply a large frontal tumor. A limited contribution from the middle meningeal artery (arrowheads) is seen. Similar angiographic findings were found on the opposite side. Note the absence of an early vein. (*B*) Right carotid angiogram, frontal projection, capillary phase. Note absence of early draining vein as well as the dense tumor stain in midline.

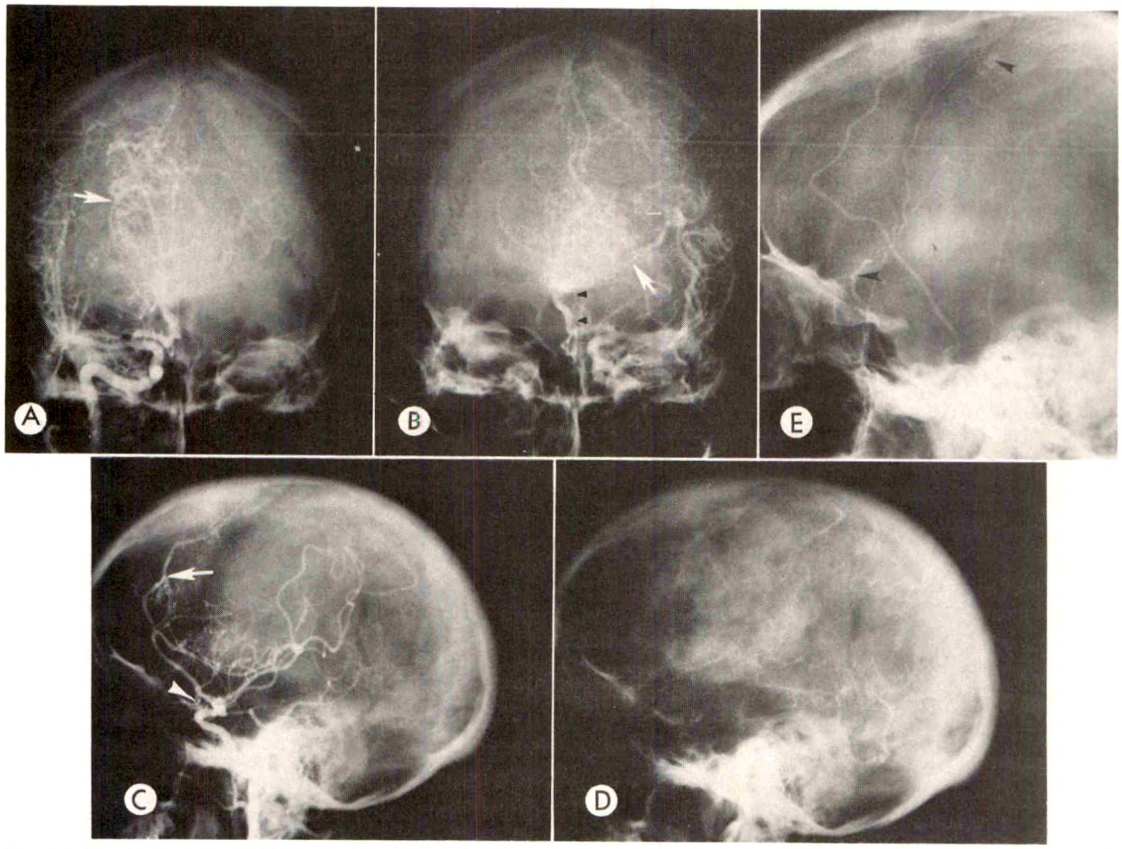

FIG. 5. Case III. C.J. a 28 year old male with history of frontal headaches and personality changes for six months. (*A* and *B*) Right and left carotid angiograms, frontal projection, showing the main feeder (arrows) from which a myriad of small vessels springs to supply a midline frontal tumor. A limited supply from the anterior falx artery is seen (arrowheads in *B*). (*C* and *D*) Left carotid angiograms, (*C*) arterial and (*D*) venous phases. The main feeders are from the anterior cerebral artery (arrow) with limited supply from the recurrent meningeal branch of the ophthalmic artery (arrowhead in *C*). Note the lingering of contrast material in venous channels augmenting the tumor stain. (*E*) Left external carotid angiogram lateral projection. Note the limited supply to the tumor by the middle meningeal artery (arrowheads).

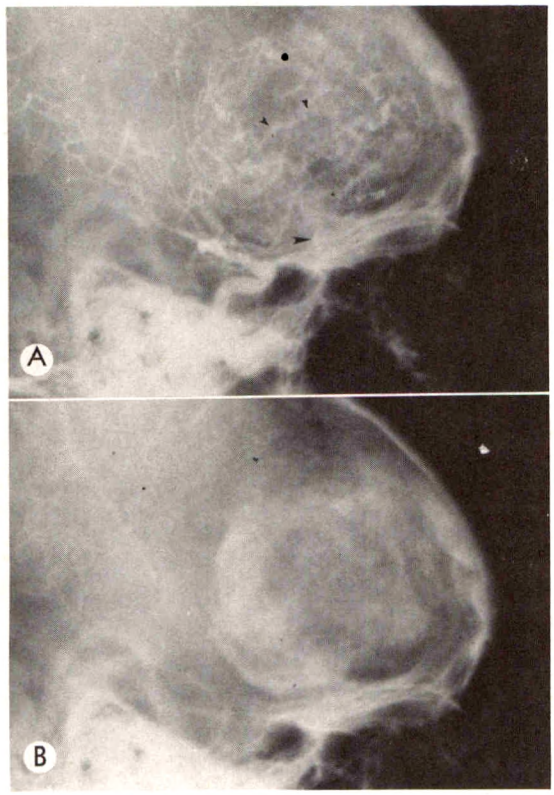

FIG. 6. Case IV. R.O. a 43 year old female with two year history of headaches and personality changes. Three month history of left sided weakness. (*A*) Right carotid angiogram lateral projection shows a major arterial supply from anterior cerebral artery branches (small arrowheads) with limited contribution from the ophthalmic artery (large arrowheads). No early vein is seen in the region of the tumor. (*B*) Right carotid angiogram lateral projection, early venous phase showing intense tumor stain in the frontal region augmented by lingering of contrast material in venous channels in the region of the tumor.

ticular, there are those who still classify this tumor as an angioblastic meningioma. This practice is based upon the classification of Cushing and Eisenhardt[6] who described this tumor as angioblastic meningioma (Type IV) variant 1. However, if the misleading notion that all tumors arising from the meninges are meningiomas is abandoned and the cytogenesis of the neoplasm in question is taken into consideration, then hemangiopericytoma as named by Stout and Murray[14] is clearly a distinct entity.

The histologic characteristics of the hemangiopericytoma are readily apparent by conventional light microscopy (Fig. 8, *A* and *B*). Numerous vessels may often be collapsed or slit shaped or at times appear as non-patent vascular buds surrounded by closely packed, mostly spindle-shaped cells with round to ovoid or elongated, moderately hyperchromatic nuclei. Mitotic figures are common (Fig. 8*A*). These cells form swirls or frond-like projections in the intervascular space which may be faintly reminiscent of meningocytic whorls (Fig. 8*A*). Each neoplastic pericyte or perivascular cluster of pericytes is enclosed by thin reticulin fibers (Fig. 8*B*). Under the electron microscope, these reticulin positive fibers are seen to be composed of moderately electron dense, granulofibrillary material arranged as a thin pericellular membrane or, frequently, as an amorphous mass in edematous intercellular space (Fig. 8*C*). The ultrastructure of this material is almost identical to that of the basement membrane of normally oriented pericytes which are also seen in the neoplasm (Fig. 8*D*). The tumor vessels usually have only one layer of normal endothelium lining the lumen (Fig. 8*D*).

Begg and Garret[3] indicated that the "malignancy" of hemangiopericytomas cannot be predicted by histological criteria despite the abundance of mitotic figures. Clinically these tumors present in a younger age group and have a shorter duration of symptoms than either meningiomas or metastatic lesions. This was also the preoperative roentgenologic impression in three of the above five cases.

Angiographically, a certain angioarchitectural pattern was found common to these tumors.

(1) A dual arterial supply from both the internal and external carotid arteries. However, the major feeding vessels are from the internal carotid artery branches or the posterior cerebral arteries rather than external carotid artery branches. This is in contrast to meningothelial meningiomas where the major blood supply is from the middle

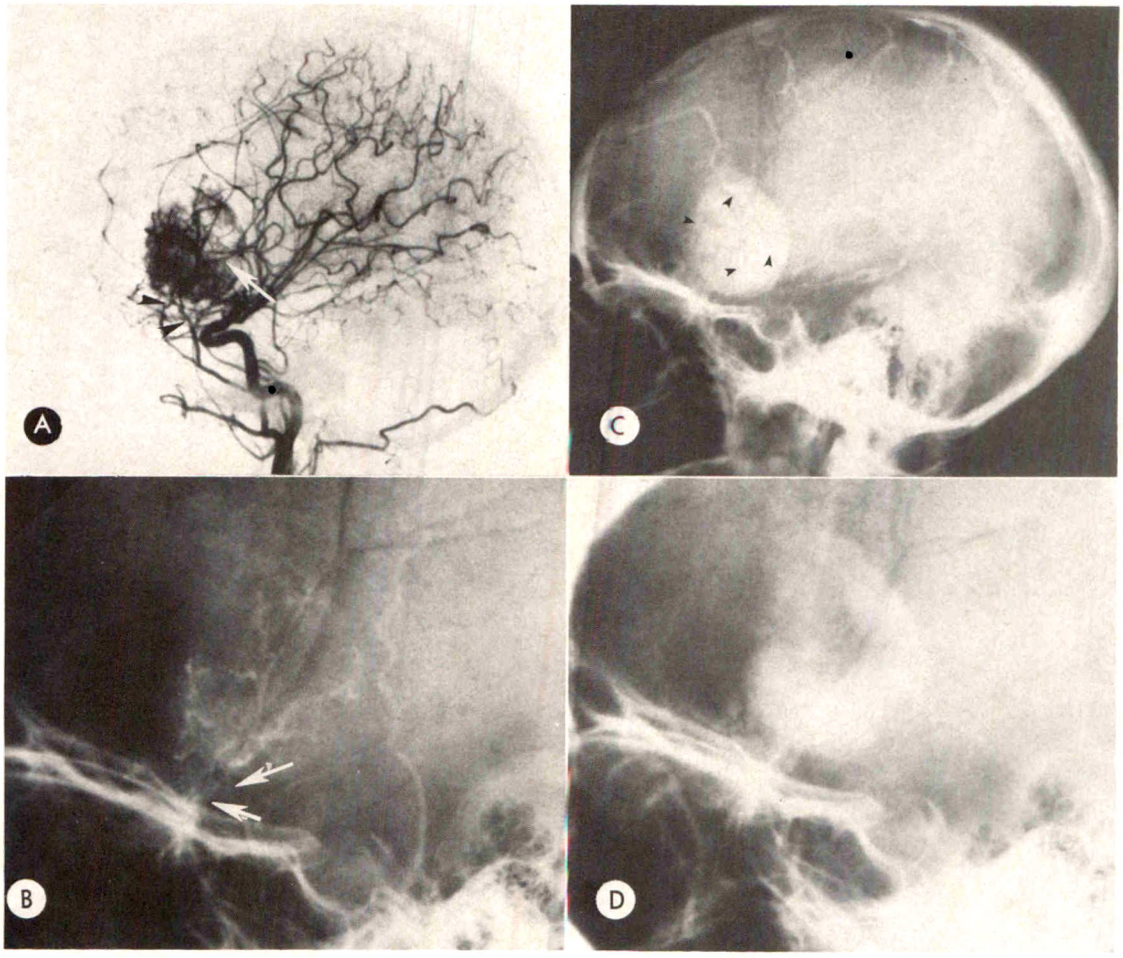

Fig. 7. Case v. F.M. a 54 year old female with history of personality changes and memory loss for one year. (*A*) Left carotid angiogram, lateral projection, arterial phase. Note the dual arterial supply from internal carotid artery branches (arrow) as well as the middle meningeal artery (arrowheads). (*B*) Venous phase showing the dense tumor stain in the frontal area. The stain is augmented by lingering of contrast material in venous channels in the tumor area (arrowheads). (*C*) Left external carotid angiogram, arterial phase showing the contribution of the middle meningeal artery to the frontal tumor (arrows). (*D*) Venous phase of the external carotid angiogram showing a rather unusual phenomenon, namely, the dense stain at the periphery of the tumor in contrast to what is seen in meningiomas where the middle meningeal artery supplies the core of the tumor as in Figure 9.

meningeal artery that supplies the center of the tumor by branching in a spoked wheel fashion[11] (Fig. 9). It is thus worth mentioning at this point that not all tumors with a meningeal blood supply are meningiomas,[12] for example, hemangiopericytomas, and glioblastoma multiforme that invades the meninges. It is interesting that in one of our patients (Case v. Fig. 7, *C* and *D*) the meningeal supply to the tumor was mainly to its periphery rather than the center as in meningiomas.

(2) A main large feeder or feeders from which a myriad of small corkscrew vessels arises within the tumor, rather than a multitude of small and large feeding vessels as is usually the case in glioblastoma multiforme[16] (Fig. 10, *A* and *B*). The major feeders and the numerous small off-springs have irregular lumens.

(3) A very dense vascular stain appearing

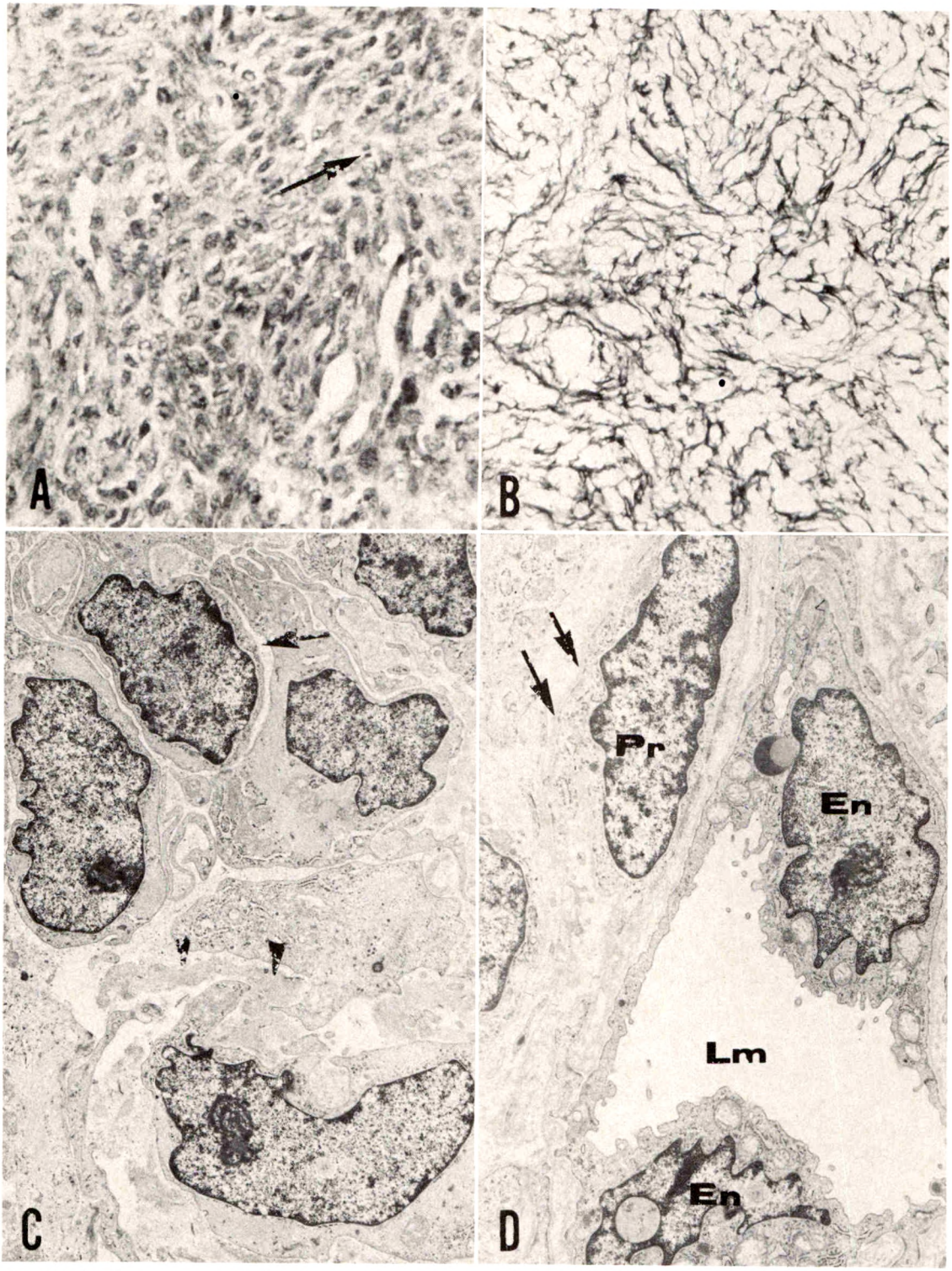

FIG. 8. Histologic characteristics of hemangiopericytoma. (*A*) Note cellular tumor with many vascular channels. A mitotic figure is present (arrow). A cluster of pericytes mimicking a meningeal whorl is seen at the bottom. H–E ×300 (*B*) Rich network of reticulin fibers surround tumor cells. Wilder ×300 (*C* and *D*) Electron micrographs (×6700) show a thin basement membrane partially surrounding a tumor cell (*C:* arrow), and amorphous aggregate of granulo-fibrillar material in edematous intercellar spaces (*C:* arrowheads) which is similar to the basement membrane of normal pericytes (*D:* arrows, Pr). Note normal endothelial cells (*D:* En) lining the lumen (*D:* Lm) of a tumor vessel.

in the mid arterial phase resembling the stain of hemangioblastomas. The intensity of stain is augmented in some cases by lingering of contrast material in draining venous channels in the region of the tumor.

(4) Slow circulation time. In none of our material was there evidence of an early draining vein in the region of the tumor. On the contrary, the veins draining the tumor stayed opacified for a prolonged period, and as they are in the immediate vicinity of the tumor they tend to augment their vascular stain as mentioned above. This is particularly significant in view of the previously mentioned reports of early filling veins draining so-called angioblastic meningiomas[7,15] or so-called malignant meningiomas.[16]

(5) The degree of arterial displacement is somewhat less than expected from the size of the area showing tumor stain, perhaps due to the infiltrative nature of the tumor.

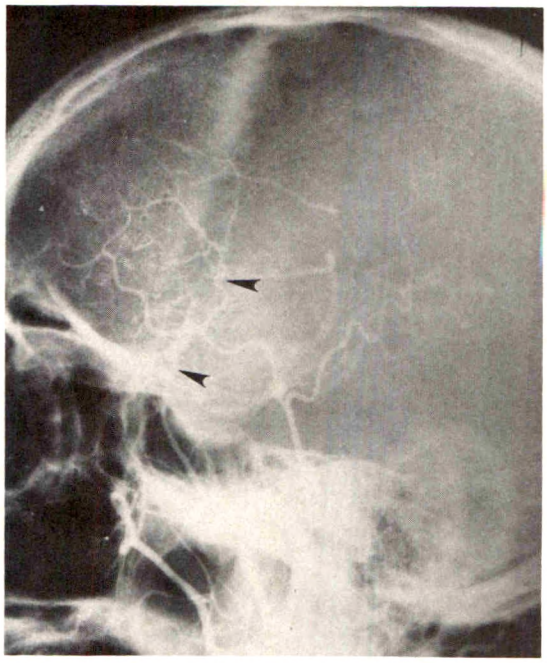

Fig. 9. Left external carotid injection in a patient with frontal meningioma showing the usual distribution of the middle meningeal supply to the core of the tumor dividing in a spoked wheel fashion (arrowheads).

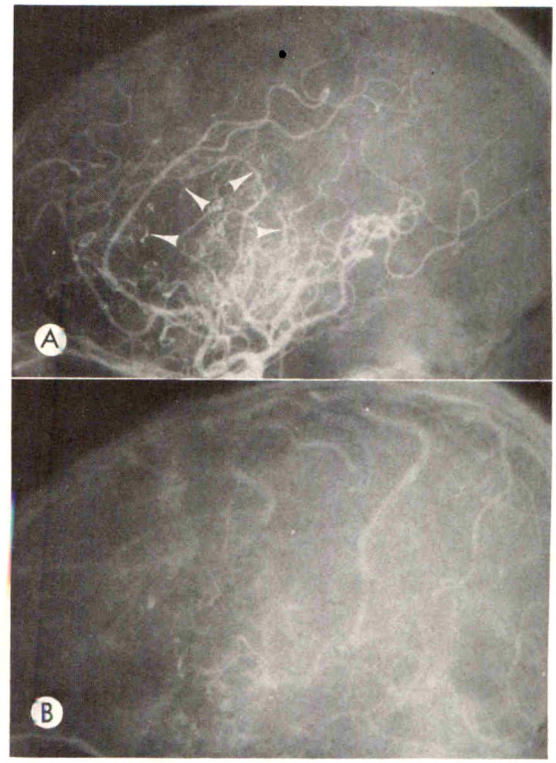

Fig. 10. (*A*) Left carotid angiogram in a patient with glioblastoma multiforme in the frontal area. Note the multiple large and small feeders (arrowheads) compared to the main feeder in Cases I–V. (*B*) Venous phase showing the irregular and blotchy stain most commonly seen in glioblastoma multiforme; compare it with the dense stain of hemangiopericytoma in Cases I–V.

(6) All cases were placed on the convexity, none at the base of the skull or the Sylvian region. One case was paratorcular. It is important to recall at this point the observation of Banna and Appleby[2] that "in tumors situated near the midline, angiography should not be limited to the side of the tumor." Although the paratorcular location has been said to be a site of predilection, only one of our cases was in that location. The rest of our cases were on the convexity, none at the base of the skull.

We should like to emphasize that in order to suspect the diagnosis of hemangiopericytoma the radiologist must diligently search for the various details of the angioarchitecture. Alerting the pathologist to the

possibility of a hemangiopericytoma will also help him to choose the proper staining methods and to look for the particular features of this tumor. Needless to say, due to the hypervascularity of this tumor, a more difficult operation will be anticipated, a fact of which the surgeon should be apprised.

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Case v was kindly contributed by Dr. William C. Spencer, Department of Radiology, Crawford W. Long Hospital, Atlanta, Georgia.

REFERENCES

1. BAILEY, P., CUSHING, H., and EISENHARDT, L. Angioblastic meningiomas. *Arch. Path.*, 1928, *6*, 953–990.
2. BANNA, M., and APPLEBY, A. Some observations on angiography of supratentorial meningiomas. *Clin. Radiol.*, 1969, *20*, 375–386.
3. BEGG, F. C., and GARRET, R. Hemangiopericytoma occurring in meninges. *Cancer*, 1954, *7*, 602–606.
4. BROWN, N. D., MACCARTHY, C. S., and SOULE, E. H. Orbital hemangiopericytoma. *J. Neurosurg.*, 1965, *22*, 354–361.
5. CASSADY, J. R., and WILNER, H. I. Angiographic appearance of intracranial sarcomas. *Radiology*, 1967, *88*, 258–263.
6. CUSHING, H., and EISENHARDT, L. Meningiomas: Their Classification, Regional Behavior, Life History and Surgical End Results. Charles C Thomas, Publisher, Springfield, Ill., 1938, pp. 19–55.
7. KEIFFER, S. A., LARSON, D. H., GOLD, L. H. A., PRENTICE, W. B., STADLAN, E. M., and SEYFERT, S. Rapid circulation in intracranial meningiomas. *Radiology*, 1973, *106*, 575–580.
8. KRUSE, F., JR. Hemangiopericytoma of meninges (angioblastic meningioma of Cushing and Eisenhardt). *Neurology*, 1961, *11*, 771–777.
9. O'BRIEN, P., and BRASFIELD, R. D. Hemangiopericytoma. *Cancer*, 1965, *18*, 249–252.
10. PITKETHLY, D. T., HARDMAN, J. M., KEMP, L. G., and EARLE, K. M. Angioblastic meningiomas: clinicopathologic study of 81 cases. *J. Neurosurg.*, 1970, *32*, 539–544.
11. SCATLIFF, J. H., GUINTO, F. C., JR., and RADCLIFF, W. B. Vascular patterns in cerebral neoplasms and their differential diagnosis. *Seminars in Roentgenol.*, 1971, *6*, 59–69.
12. STATTIN, S. Significance of some angiographic signs of intracranial meningiomas. *Acta radiol.* (Diag.), 1966, *5*, 530–535.
13. STOUT, A. P. Hemangiopericytoma: study of 25 new cases. *Cancer*, 1949, *2*, 1027–1054.
14. STOUT, A. P., and MURRAY, M. R. Hemangiopericytoma: vascular tumor featuring Zimmermann's pericytes. *Ann. Surg.*, 1942, *116*, 26–33.
15. TELENIUS, R. Angiographic appearance of angioblastic meningiomas. *Acta radiol.* (Diag.), 1966, *5*, 554–561.
16. WICKBOM, I. Angiographic determination of tumor pathology. *Acta radiol.*, 1953, *40*, 529–546.
17. ZIMMERMANN, K. W. Der feinere Bau der Blutkapillaren. *Ztschr. Anat.*, 1923, *68*, 29–109.

Vol. 125, No. 4

DISPLACEMENT OF PONTINE ARTERIES BY FUNGATING PONTINE ASTROCYTOMAS*

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ABSTRACT:

Pontine astrocytomas may fungate, encircling the basilar artery and pontomesencephalic vein and intruding between these structures and the clivus. The tumor thus may prevent anterior displacement of these vascular structures against the clivus or produce paradoxical posterior displacement. However, the pontine arteries are usually swept forward by the tumor nodules to lie anterior to the basilar artery. These arteries are also stretched as they encircle the enlarged pons, losing their normal undulations. An increased posterior extent of the vessels may be seen, presumed due to hypertrophy or displacement or both. These findings seem highly specific for the diagnosis of fungating pontine astrocytoma.

AN intra-axial pontine mass characteristically displaces the basilar artery and pontomesencephalic vein anteriorly against the clivus. However, pontine astrocytomas may fungate with tumor nodules interposed between the vascular structures along the anterior border of the pons and the clivus. The basilar artery and pontomesencephalic vein may then appear in a normal position or displaced posteriorly.

Huang and Wolf[4,5] and Takahashi[7,8] stated that the pontine arteries may lie anterior to the basilar artery in patients with a fungating pontine astrocytoma. This finding was termed the paradoxical phenomenon and the authors presented four examples.

At the University of Tennessee we have studied four children with fungating pontine astrocytomas in whom the course and extent of the pontine arteries seemed abnormal. A previous report[2] describing the normal course of the pontine arteries was written prior to magnification techniques. Stimulated by these patients, we reviewed the pontine arteries in 50 normal vertebral angiograms in the lateral projection utilizing 2:1 magnification.

The purpose of this report is to compare the normal course and appearance of the pontine arteries to that seen in fungating pontine astrocytomas.

NORMAL APPEARANCE OF THE PONTINE ARTERIES

Gabrielsen and Amundsen[2] described the arteriographic appearance of the pontine arteries about the upper pons. The median branches are minute twigs which enter the pons immediately from the posterior portion of the basilar artery and cannot be identified angiographically. The transverse circumferential branches were seen in 87 percent of selective vertebral angiograms in the lateral view. These arteries were described as often making a distinct turn immediately adjacent to the anterior border of the pons. As they coursed about the lateral border of the pons the arteries were faintly seen, eventually vanishing as they entered the pontine parenchyma.

We reviewed the lateral arterial phase in each of 50 normal unselected vertebral angiograms performed transfemorally.

Table I

AGE DISTRIBUTION OF NORMAL PATIENTS

0– 9 years	13
10–19 years	12
20–29 years	10
30–39 years	7
Over 40 years	8
Total	50

* From the Department of Diagnostic Radiology, University of Tennessee College of Medicine, Memphis, Tennessee.

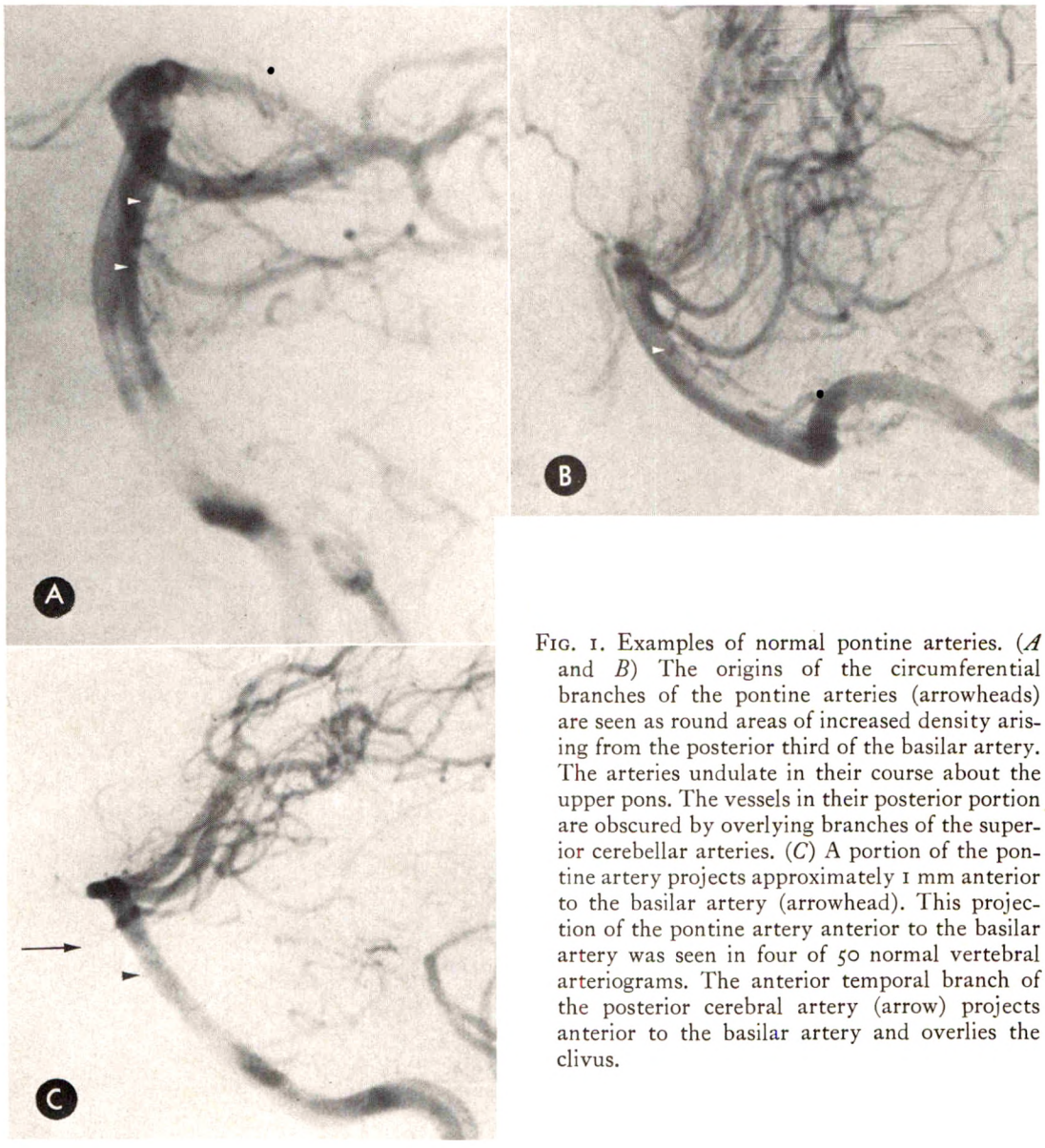

FIG. 1. Examples of normal pontine arteries. (*A* and *B*) The origins of the circumferential branches of the pontine arteries (arrowheads) are seen as round areas of increased density arising from the posterior third of the basilar artery. The arteries undulate in their course about the upper pons. The vessels in their posterior portion are obscured by overlying branches of the superior cerebellar arteries. (*C*) A portion of the pontine artery projects approximately 1 mm anterior to the basilar artery (arrowhead). This projection of the pontine artery anterior to the basilar artery was seen in four of 50 normal vertebral arteriograms. The anterior temporal branch of the posterior cerebral artery (arrow) projects anterior to the basilar artery and overlies the clivus.

Two-to-one magnification was used in all patients. The ages of the patients are presented in Table 1. The youngest was two weeks and the oldest 56 years. A small number of retrograde brachial studies with magnification was also reviewed but the pontine arteries could be identified in none. The number of magnification studies in the anteroposterior projection available for review was insufficient for evaluation. The measurements presented are not corrected for magnification.

The pontine arteries about the upper pons were identified in 43 of the 50 patients (Fig. 1, *A* and *B*). The origins of the vessels could be seen as tiny areas of increased density along the mid and posterior portions of the basilar artery. The number of arteries varied from two to five. From their origins, the vessels coursed posteriorly around the lateral border of the pons with a generally undulating contour. The vessels could rarely be traced more than 5 mm posterior to the basilar artery and in no case beyond 10 mm. The inability to see the vessels in their more posterior portions

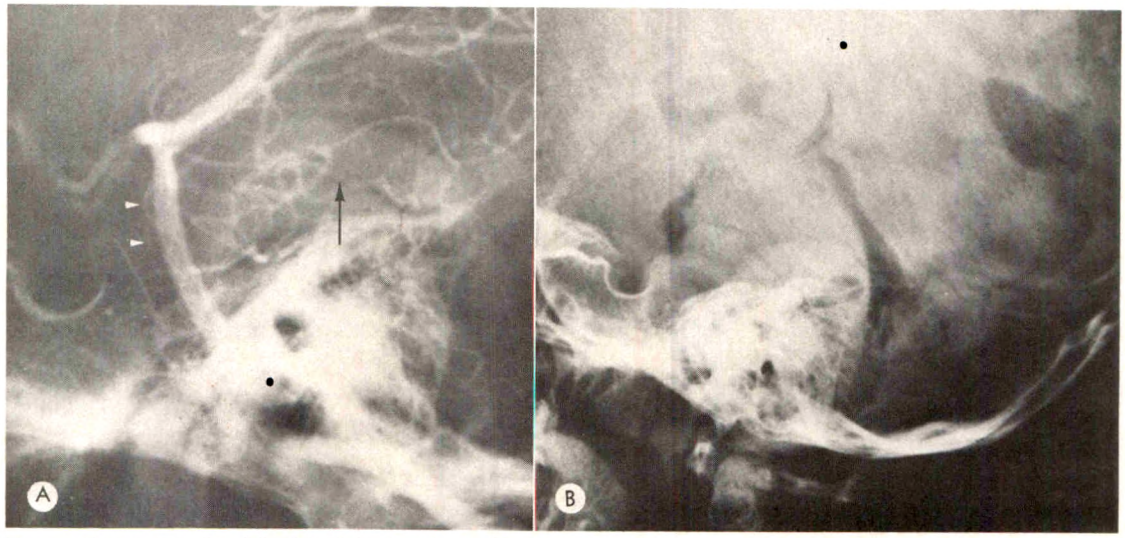

Fig. 2. Four year old girl with right hemiparesis, difficulty swallowing, slurred speech, left facial twitching, ataxia, and incoordination. (*A*) Pontine arteries (arrowheads) project anteriorly to a normally positioned basilar artery. The pontine arteries can be traced approximately 18 mm posterior to the basilar artery (arrow). (*B*) The fourth ventricle and aqueduct are displaced posteriorly. The prepontine cistern is compressed. A ventriculoperitoneal shunt was performed and radiation therapy given.

may be due to their entering the pontine parenchyma, but visualization is difficult also because of superimposed superior cerebellar branches.

The pontine arteries were seen to extend 1 to 2 mm anterior to the anterior border of the basilar artery in four patients before looping posteriorly (Fig. 1*C*). Rotation did not seem to be a factor in these patients. Atherosclerotic changes seem unlikely as three of the four patients were in the third decade. We assume the vessels were not lying along the anterior border of the pons but rather were looping in the prepontine cistern and therefore did not give an accurate idea of the anterior border of the pons. The anterior temporal branch of the posterior cerebral artery, which normally extends anterior to the pons, should not be confused with the pontine arteries.[6]

MATERIAL*

Four children have been seen with a final clinical and roentgenographic diagnosis of fungating pontine astrocytoma. Each presented with varying cranial nerve deficits, hemiparesis, ataxia and hyper-reflexia. Transfemoral cerebral angiography was performed in each as well as pneumoencephalography or ventriculography. In three of the four, the posterior fossa was not surgically explored. These patients were treated with ventriculoperitoneal shunts and irradiation. Two are alive six and twelve months later. The third patient expired three months after shunting. In the fourth patient, a fungating pontine astrocytoma was confirmed at surgery. This patient expired one month later.

Angiographically, the basilar artery and pontomesencephalic vein were in normal position in two of the four patients (Fig. 2–5). Both vascular structures were displaced posteriorly in the other two. In each of the four patients the pontine arterial loops lay 3 to 4 mm anterior to the basilar artery. In two children, the arteries were distinctly stretched with loss of their normal undulations as they encircled the pons. The arteries could be traced 18–20 mm posterior to the basilar artery in three instances and approximately 8 mm in the fourth. Encephalography showed posterior

* Since submission of this paper for publication, we have performed cerebral angiography on a fifth patient with a pontine astrocytoma. The basilar artery and pontomesencephalic vein were in normal position. Pontine arteries were displaced anterior to the basilar artery. Pneumoencephalography confirmed the diagnosis.

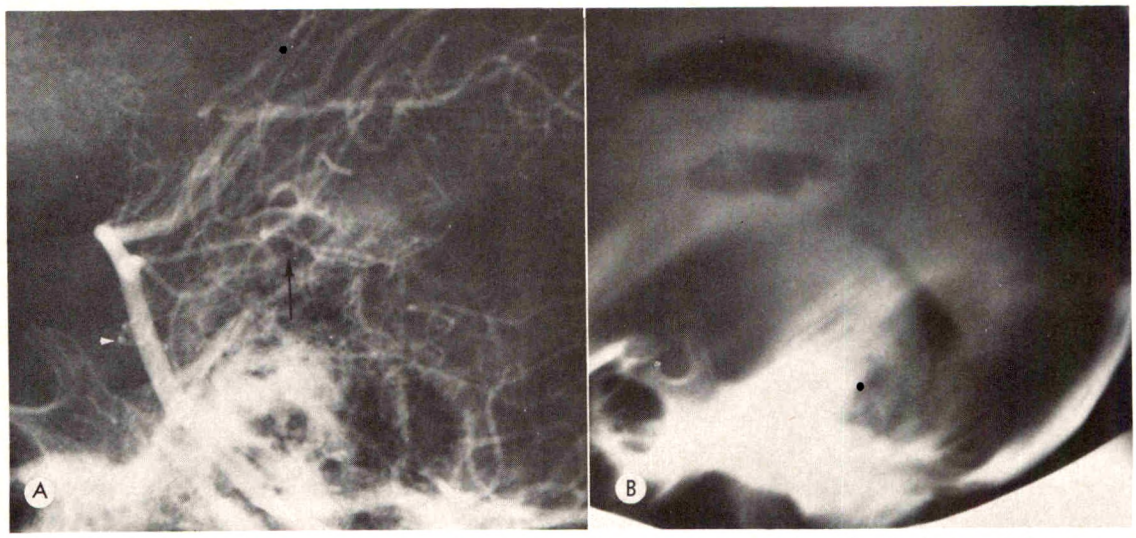

FIG. 3. Fourteen year old boy with staggering, left facial weakness, truncal ataxia, deviation of the tongue to the left, and left hemiparesis. (*A*) Pontine arteries (arrowhead) project anteriorly to a normally positioned basilar artery and are stretched as they encircle the pons. The arteries can be seen approximately 20 mm posterior to the basilar artery (arrow). (*B*) The aqueduct and fourth ventricle are displaced posteriorly. At craniotomy, a fungating pontine astrocytoma was found.

displacement and widening of the fourth ventricle and aqueduct in each of the patients, confirming the diagnosis of an intra-axial pontine mass.

The angiograms of ten patients with fourth ventricular or cerebellar masses were reviewed. The basilar artery and pontomesencephalic vein were displaced anteriorly against the clivus in each patient. The pontine arteries did not project anterior to the basilar artery in any case and their course around the pons showed normal undulations.

DISCUSSION

Bradac and Martins[1] show two anatomic specimens with the basilar artery encircled by a pontine astrocytoma. Other authors[3,6] have described posterior or lateral displacement, or both, of the basilar artery in pontine tumors extending eccentrically into the cerebellopontine angles and into the prepontine cistern. Displacement of the pontine arteries was not mentioned in these reports.

It has been stated that an intra-axial pontine mass may displace the pontomesencephalic vein posteriorly and the basilar artery anteriorly.[9] Gabrielsen and Amundsen[2] felt the pontomesencephalic vein was a better index of the anterior border of the pons than the pontine arteries. In our cases, the basilar artery and pontomesencephalic vein were in normal position or displaced posteriorly and lay the same distance from the clivus. The pontine arteries, therefore, seem to give a more accurate picture of the location of the anterior pontine border.

In our four normal patients in whom the pontine arteries extended anterior to the basilar artery, the maximal anterior extent was 2 mm. The anterior extension in the four patients with pontine astrocytomas, in contrast, varied from 3 to 5 mm. The posterior extent of the pontine arteries in the normal patients seemed greater than 5 mm in only four patients and did not exceed 10 mm. In three of the four patients with an astrocytoma, the posterior extent was greater than 10 mm. In two of these patients the pontine branches were distinctly stretched with loss of their normal undulations on both anteroposterior and

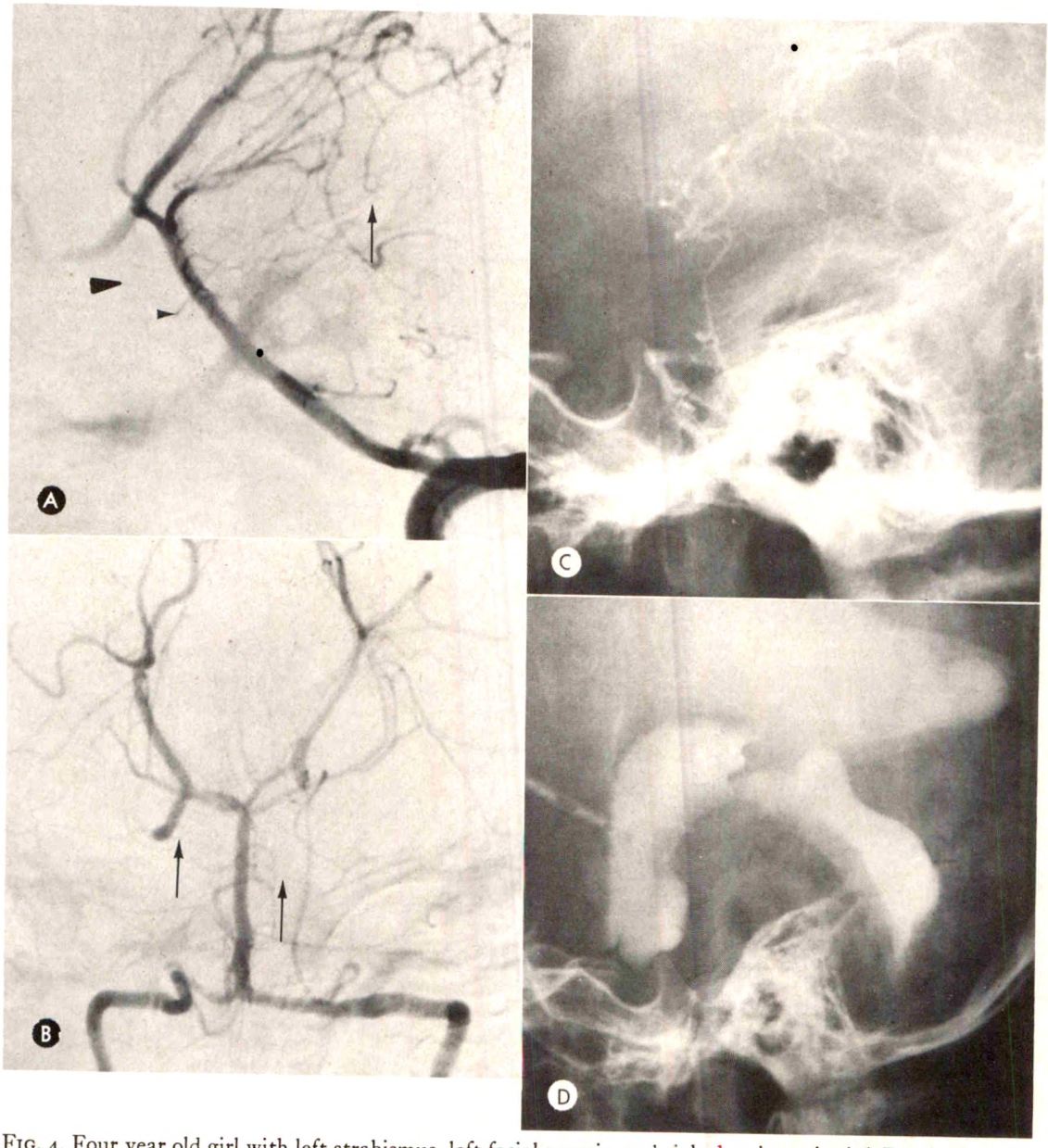

Fig. 4. Four year old girl with left strabismus, left facial paresis, and right hemiparesis. (*A*) Pontine arteries (arrowhead) project anterior to the basilar artery which appears somewhat posterior to the clivus (large arrowhead). The pontine arteries are stretched as they course about the pons and extend approximately 20 mm posterior to the basilar artery (arrows). (*B*) Stretching of the pontine arteries (arrows) is again seen. (*C*) The pontomesencephalic vein is displaced posteriorly. (*D*) Conray ventriculography shows a posteriorly displaced fourth ventricle with the mass producing an eccentric defect in the floor. A ventriculoperitoneal shunt was performed and radiation therapy begun.

lateral views.

The most reasonable explanation for the anterior displacement and stretching of the pontine arteries is that proposed by Huang and Wolf,[4,5] *i.e.*, displacement of the less rigid pontine arteries by tumor nodules. The increased posterior extent may be due to hypertrophy of vessels supplying the tumor as well as displacement. The role played by rotation of the brain stem in

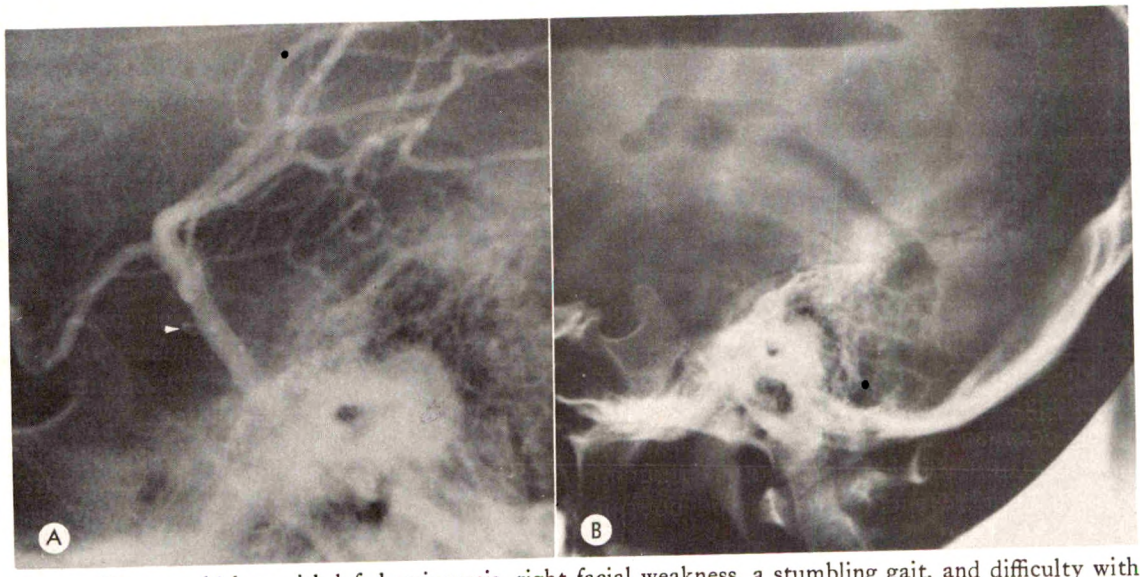

FIG. 5. Six year old boy with left hemiparesis, right facial weakness, a stumbling gait, and difficulty with speech. (*A*) The basilar artery is displaced away from the clivus. Pontine arteries (arrowhead) project anterior to the basilar artery. The pontomesencephalic vein (not illustrated) also was displaced posteriorly. (*B*) Pneumoencephalography shows the fourth ventricle and aqueduct to be displaced posteriorly. A ventriculoperitoneal shunt was performed and radiation therapy begun.

these findings is not definite. However, the position of the superior cerebellar and posterior cerebral arteries did not suggest brain stem rotation in our patients.

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REFERENCES

1. BRADAC, G. B., and MARTINS, L. F. Remarks about angiographic findings in cases of pontine tumors. *Neuroradiology*, 1974, *7*, 105–108.
2. GABRIELSEN, T. O., and AMUNDSEN, P. Pontine arteries in vertebral angiography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *106*, 296–302.
3. GLICKSMAN, M. G., and SHOLKOFF, S. D. Posterior displacement of basilar artery by intrinsic pontine tumors. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *112*, 276–280.
4. HUANG, Y. P., and WOLF, B. S. Angiographic features of brain stem tumors and differential diagnosis from fourth ventricle tumors. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *110*, 1–30.
5. HUANG, Y. P., and WOLF, B. S. Differential diagnosis of fourth ventricle tumors from brain stem tumors in angiography. *Neuroradiology*, 1970, *1*, 4–19.
6. MARGOLIS, M. T., NEWTON, T. H., and HOYT, W. F. Cortical branches of posterior cerebral artery: anatomic-radiologic correlation. *Neuroradiology*, 1971, *2*, 127–135.
7. TAKAHASHI, M. Atlas of Vertebral Angiography. University Park Press, Baltimore, 1974, pp. 137–145.
8. TAKAHASHI, M. Basilar artery. In: Radiology of the Skull and Brain: Angiography. Edited by T. H. Newton and D. G. Potts. C. V. Mosby Company, St. Louis, 1974, pp. 1971–1974.

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UNUSUAL FOREIGN BODIES ASSOCIATED WITH CRANIAL TRAUMA*

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ABSTRACT:

Two unusual cases of cranial trauma are presented because of the bizarre roentgenographic appearance of the traumatizing foreign bodies.

RADIOLOGISTS are accustomed to visualizing a multiplicity of opaque foreign bodies accidentally causing, or deliberately used to inflict, cranial trauma. Two cases are presented which attest to the incredibly fertile ability of "homicidal man" to utilize everyday objects in the commission of mayhem, and the unusual roentgenographic appearance of these intracranial opaque foreign bodies.

REPORT OF CASES

CASE 1. A 26 year old, white male was admitted to the Emergency Room after transfer from the California State Prison facility at San Quentin. He allegedly had been stabbed, had multiple torso and flank wounds and had a rubber eraser protruding from the medial aspect of the left orbit. Physical examination revealed blindness in the left eye, a temporal field defect on the right, some confusion, but no dysphasia nor gross hemisensory or hemiparetic signs. Roentgenograms revealed a pencil-shaped foreign body (Fig. 1, *A* and *B*). Following lumbar puncture demonstrating extremely bloody cerebrospinal fluid, immediate left frontal craniotomy was performed (Dr. Sam Hanzel). The pencil was removed from the left orbit. There was a large hole posterior to the cribriform plate on the left side, and a fracture line extended transversely and obliquely across the lesser wing of the sphenoid to the superior oblique orbital fissure. The pencil had passed from the left orbit through the floor of the anterior cranial fossa, through the left frontal

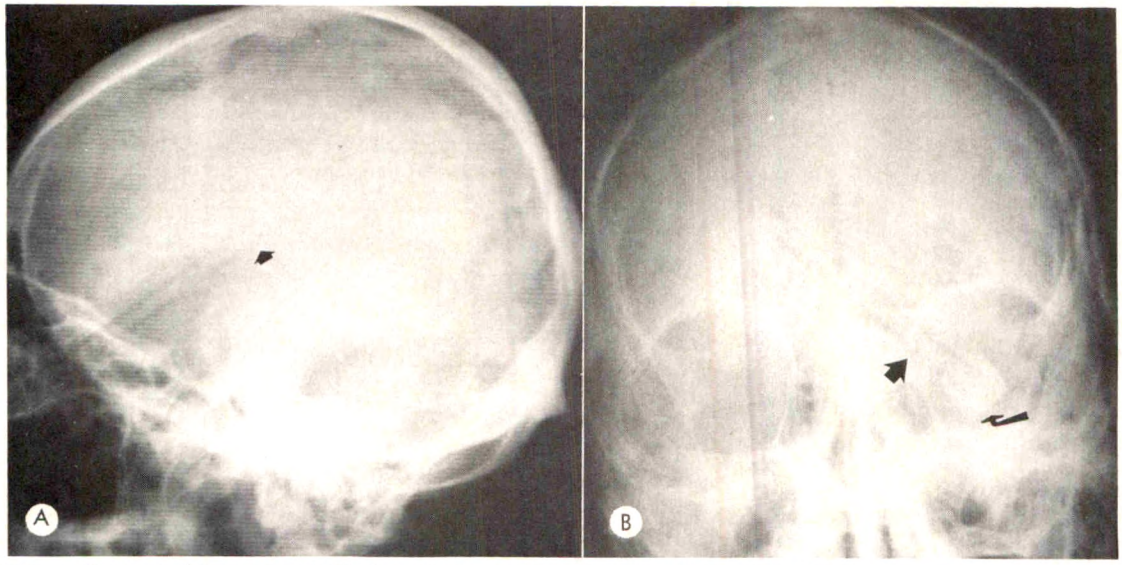

FIG. 1. Case 1. (*A*) Lateral and (*B*) frontal roentgenograms show shaft of pencil (short arrow) extending from left frontal area obliquely and posteriorly into the right midparietal area. Curved arrow indicates eraser.

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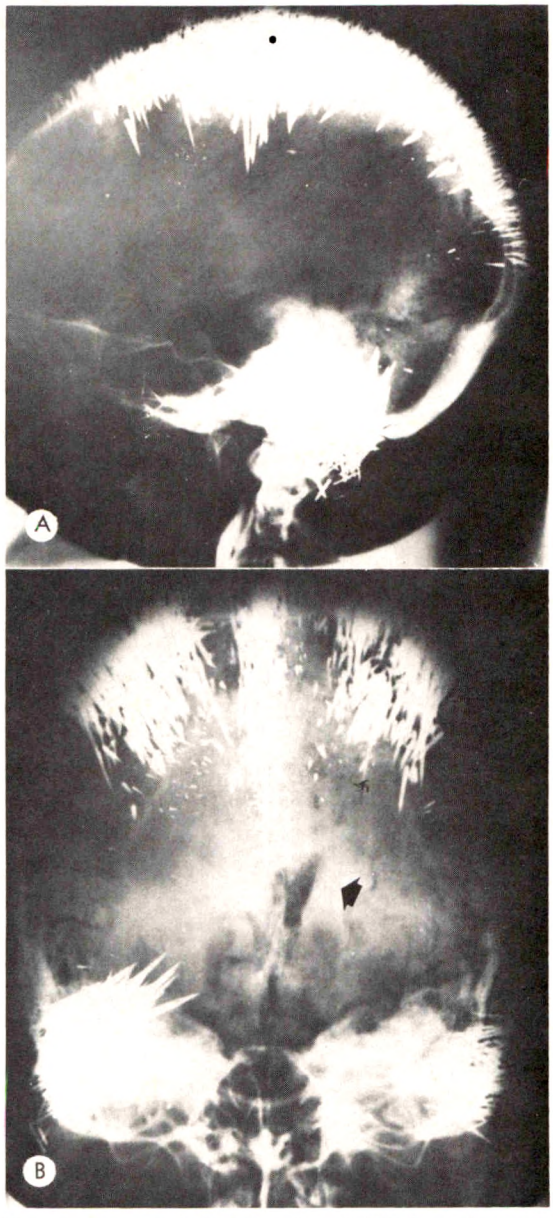

FIG. 2. Case II. (*A*) Lateral and (*B*) frontal roentgenograms demonstrate multiple opaque nails extending through the bony calvarium. Arrow indicates air in the ventricular system.

lobe and into the right lateral ventricle. The left optic nerve was severely injured. Bone fragments near the superior oblique orbital fissure were lifted out and patches of pericranium were used to seal off the dural openings with use of a biologic glue.

The patient was started on huge antibiotic coverage. He was initially aphasic, had a severe memory defect, and exhibited unusual behavior with inappropriate laughter. This was felt to represent organic brain damage.

On the twelfth hospital day, skull roentgenograms demonstrated increased pneumocephalus and it was postulated that air was coming in from a leak in the ethmoid sinus (entering the ventricular system of the frontal lobe by that route). He had evidence of a cerebrospinal fluid fistula. One month postinjury he was readmitted for isotope and indigo carmine dye studies to identify a possible cerebrospinal fluid fistula, but none could be demonstrated. He was finally discharged, off antibiotics, with continued blindness, organic brain damage, and an abnormal electroencephalogram compatible with brain injury in the left anterior frontal area.

CASE II. In 1967, a young male of unknown age was transferred from local detention facilities to the Emergency Room of the Medical College of Virginia Hospital in a moribund state. Skull roentgenograms were obtained and demonstrated multiple, metallic objects throughout the bony calvarium as well as pneumocephalus (Fig. 2, *A* and *B*). The patient expired before any treatment could be instituted. It was alleged that the metallic objects, which were carpet nails, were self-introduced by the patient using the heel of his shoe. (The complete records of this case are not now available and the history is as could be obtained at that time.)

DISCUSSION

The above cases are of interest because the unique roentgenographic appearance accurately reflects the bizarre history of each case. Also of interest is the difficulty, for experienced radiologists, in perceiving and conceptualizing the nature of the abnormality when presented with these roentgenograms without a history. This was particularly true in Case II, where, over a period of many years, experienced radiologists who were shown the roentgenograms have attempted to dismiss the opaque nails as artifactitious or relate them to an unusual disease state or unusual mode of medical therapy. Conversely, individuals

with less medical sophistication, such as lay people and medical students, have had less difficulty in perceiving the true nature of the foreign bodies as being nails. This is indicative of the difficulty in correctly perceiving something which one is conceptually unprepared to accept. Even in our violent society, it is difficult to conceive of intracranial nails or pencils.

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CHEST ROENTGENOGRAPHY AS A WINDOW TO THE DIAGNOSIS OF TAKAYASU'S ARTERITIS*

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ABSTRACT:

The chest roentgenographic findings in Takayasu's arteritis include widening of the ascending aorta, contour irregularities of the descending aorta, aortic calcifications, pulmonary arterial changes, rib notching, and hilar lymphadenopathy. The single most important diagnostic sign is a segmental calcification outlining a localized or diffuse narrowing of the aorta. The other signs may be suspicious or suggestive, but the diagnostic accuracy increases when several findings are present simultaneously.

TAKAYASU, a Japanese ophthalmologist, first described peculiar eyeground changes in a young female in 1908,[11] but it was not until 1951 that Shimizu and Sano[10] established the clinical entity of brachiocephalic arteritis and named the disease "pulseless syndrome." Recently, with the accumulation of autopsy and arteriographic findings, the whole extent of the disease became apparent. Takayasu's disease, as it is known today, is a form of arteritis involving the aorta and its branches, and the pulmonary arteries in a indiscriminate fashion.[4,5] The peripheral arteries are very rarely involved.[8]

In the early stages of the disease, granulation tissue accompanied by a cellular infiltrate invades and destroys the elastic and muscular framework of the tunica media. Gradually, the activity of the disease subsides and nonspecific scar tissue replaces functional elements of the aortic wall resulting in a marked thickening of the latter. The intima, although not directly involved in the inflammatory process, shows fibrous thickening as a secondary reactive phenomenon. This may assume enormous proportions and is primarily responsible for luminal narrowing and occlusion. Secondary thrombosis and accelerated atheromatosis are readily engrafted upon the diseased intima.

Fibrous adventitial thickening may be confined to the aortic wall or may deeply infiltrate the periaortic tissues. Consequently the external aortic contour irregularities occur. The outer circumference can be dilated or narrowed. Extreme luminal constriction can occasionally occur in the presence of only mild alteration of the outer diameter of the vessel.

In other instances with rapid destruction of the elastic and muscular elements of its wall, the aorta becomes dilated or aneurysms are formed. Dilatation of the aorta may be localized or may involve long, contiguous segments. Aneurysms are common and frequently alternate with aortic narrowing. Skip areas of aortic involvement are very characteristic for this type of arteritis.

The clinical manifestations are commonly subdivided into a systemic prepulseless and late pulseless phases. Nonspecific generalized signs and symptoms of the early phase are: malaise, arthralgias, myalgias, pleurisy, hemoptysis, chest and abdominal pain, skin rashes, etc.

The late pulseless phase is characterized by ischemic signs and symptoms secondary to arterial occlusions. Bruits over the affected vessels are audible and heart murmurs (usually transmitted) are common. Occlusion or stenosis of the brachio-

* Presented as a Scientific Exhibit at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.
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cephalic vessels may give rise to a variety of cerebrovascular and visual manifestations. Due to coarctation of the suprarenal aorta or renal artery stenosis, renovascular hypertension is common. Narrowing of the infrarenal aorta may cause claudication of the lower limbs.

According to recent reviews, the prepulseless phase of Takayasu's arteritis is rarely recognized.[1] The presenting signs and symptoms are usually of the late occlusive variety.

Takayasu's arteritis has no geographic bounderies. It is mainly a disease of young women (female to male ratio about 8:1). Although it is reported as early as three and as late as 75 years of age, the majority of the patients is between the age of eleven and 30 at the time of diagnosis. Its etiology is not clearly understood, but it is generally accepted to be an auto-immune disease.

The incidence of Takayasu's arteritis in the United States is apparently much higher than generally suspected. It has been estimated that there are several hundred thousand people in the United States with asymptomatic lesions of Takayasu's arteritis and in many symptomatic patients, the etiological nature of the disease remains unrecognized.[6] Recognition of the plain roentgenographic findings, therefore, is of primary diagnostic importance.

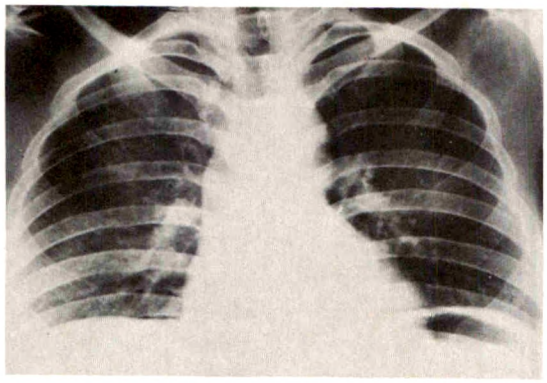

FIG. 1. A 28 year old female. Note prominence of the ascending aorta and contour irregularities of the descending aorta. Aortography showed typical changes of Takayasu's arteritis with involvement of the descending thoracic and entire abdominal aorta, and occlusion of both subclavian arteries.

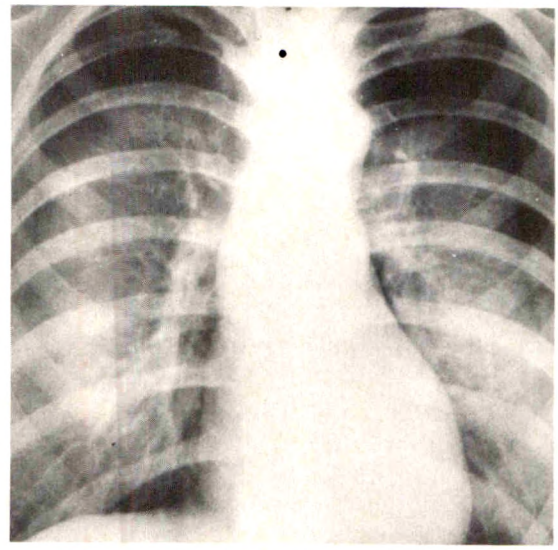

FIG. 2. An 18 year old female. Hypertensive with a two year history of headaches, blurred vision, dizziness, and occasional syncope. Note the prominence of the ascending aorta, undulation of the descending aorta with a constriction of subisthmic region, simulating congenital isthmic coarctation. Aortography showed widespread involvement of the aorta and its branches.

A radiologist, familiar with the natural history of the disease and the roentgenographic findings as observed at plain chest roentgenography, plays a crucial part in the diagnosis of this entity.

CHEST ROENTGENOGRAPHIC MANIFESTATIONS

Roentgenographic changes in the chest roentgenogram to be described are based on our experience with 25 cases of Takayasu's arteritis and a review of the literature.

AORTIC CHANGES

Aortic alterations may be subdivided into three categories: (1) widening of the aorta; (2) contour irregularities of the descending aorta; and (3) aortic calcifications.

Widening of the aorta: The ascending aorta is commonly involved in Takayasu's arteritis, but occlusive changes are not found in this portion.[2] Prominence is usually due to prestenotic dilatation. In about ten to 20 percent of the patients, aortic valvular insufficiency may occur and con-

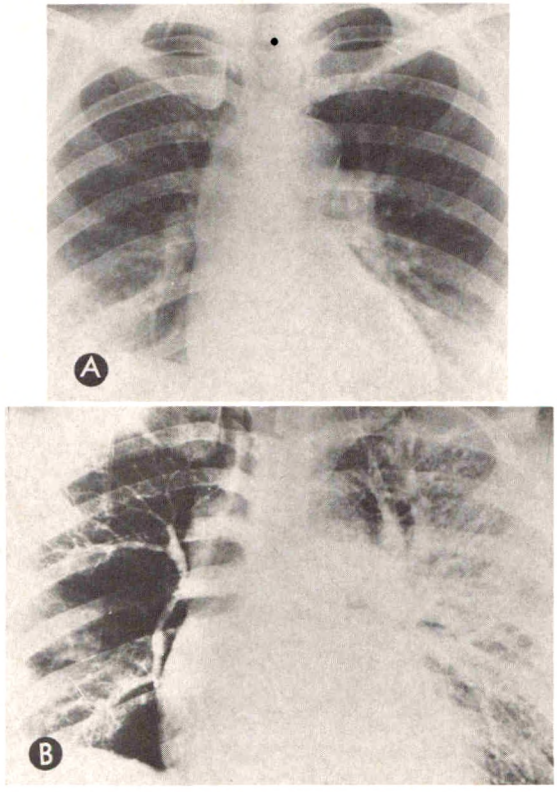

FIG. 3. A 24 year old female. Hypertensive and vascular murmurs since age of ten. (*A*) Chest roentgenogram shows prominent ascending aorta and aortic arch which has two segmental calcifications. There is a marked decrease in vascularity of the right lung and overcirculation of the left lung. (*B*) Pulmonary angiogram shows a slight prominence of the main pulmonary artery. The right main pulmonary artery is markedly narrowed. Two slender arterial twigs supply isolated segments of the right upper lobe and right lower lobe. There is hypercirculation of the left lung.

tribute to the enlargement of the ascending aorta.[2] This is usually a functional insufficiency secondary to a dilated aortic root. Direct involvement of the aortic valves is uncommon.[9]

Widening may only be seen in the ascending aorta, but quite often enlargement involves also the arch and descending aorta in contiguity (Fig. 1; 2; and 3).

Contour irregularities of the descending aorta: Contour irregularities are the result of alternating narrowings and dilatations of the aorta or periaortic fibrosis, or both, producing an undulant appearance of the descending aorta (Fig. 1; and 2). These changes can only be seen in a sufficiently penetrated film. If in doubt, oblique films and even tomography are recommended.

Linear aortic calcifications: These may be seen in short or long segments of the aortic arch, descending or abdominal aorta (Fig. 3*A*; and 4). They are usually dystrophic in nature and are, as a rule, fine and pencil-like in outline. In some instances characteristic narrowing of the aorta is delineated by a segmental calcification (Fig. 4).

PULMONARY ARTERIAL CHANGES

Since Takayasu's disease is a generalized arteritis, the large and intermediate pulmonary arteries are commonly involved in the process.[3,9] Occasionally the pulmonary arteries may be the major site of the disease.[7] Gross and histological changes in the pulmonary arteries are identical with those of the aorta and its branches. Arteritis of the pulmonary arteries can result in pulmonary hypertension,[4] where the main pulmonary artery becomes prominent. Marked narrowings and occlusions of the intrapulmonary branches may be reflected in the chest roentgenogram as segmental or lobar hypervascularity, although this finding may be difficult to detect (Fig. 3, *A* and *B*).

RIB NOTCHING

Subtle notching of the first three ribs may be an expression of subclavian artery occlusion. Notching of the lower ribs is usually associated with severe narrowing or occlusion of the upper abdominal aorta with intercostal arteries serving as prominent collateral channels. Rib notching is not a common finding since many years are required for its development (Fig. 1).

HILAR LYMPHADENOPATHY

This is an extremely rare occurrence in this condition and has not been previously reported. We believe it is a part of systemic manifestation of Takayasu's arte-

ritis, probably due to reactive hyperplasia (Fig. 5).

DISCUSSION

Although the diagnostic value of an isolated roentgenographic abnormality varies, the diagnostic accuracy is markedly increased when several findings are present simultaneously.

The single most important diagnostic sign is segmental calcification outlining a localized or diffuse narrowing of the aorta (Fig. 4).

An irregular and undulant outline of the aorta and linear calcifications are highly suggestive signs, especially when seen in a young woman. However, they become diagnostic when combined with other findings.

Fig. 4. A 52 year old female. Hypertensive for 20 years with headaches, palpitation, and dizziness for several years. On lateral chest roentgenogram gradual segmental narrowing of the distal thoracic aorta is outlined by linear calcifications.

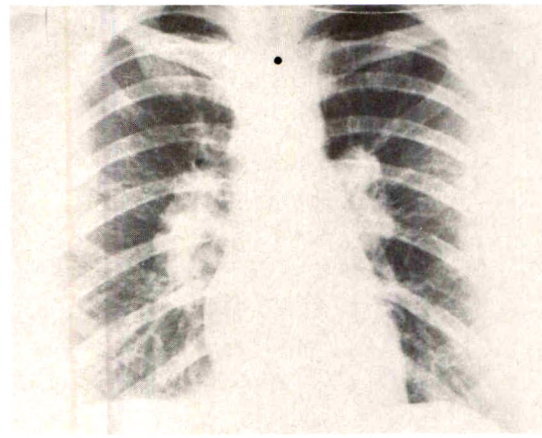

Fig. 5. A 20 year old female. History of febrile illness associated with polyarthritis, cough and skin rash six months prior to aortography. Paresthesia and numbness of the right arm occurred two months before admission. Chest roentgenogram shows bilateral hilar lymphadenopathy. Total aortography was normal. Right subclavian angiography showed smooth, tapered narrowing and occlusion of the axillary artery, typical of Takayasu's arteritis.

Notching of the lower ribs (usually sixth and below) is a highly suggestive sign.

Dilatation of the ascending aorta, arch and the descending aorta in contiguity is suspicious, although it becomes highly suggestive in the presence of calcifications and is diagnostic when seen together with contour irregularities of the descending aorta or skipped areas of narrowing and dilatation.

The large main pulmonary artery, areas of decreased pulmonary vascularity with segmental or lobar distribution, when seen alone, are not diagnostic, although Takayasu's arteritis enters into the differential diagnosis. Changes in the pulmonary vessels should be looked for, however, in any known or suspected case of Takayasu's arteritis, because of the common association of pulmonary arterial involvement in this disease.

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REFERENCES

1. GILLANDERS, L. A., and STRACHAN, R. W. Role of radiology in diagnosis of Takayasu's arteriopathy ("pulseless disease"). *Clin. Radiol.*, 1965, 16, 119–129.
2. HACHIYA, J. Current concepts of Takayasu's arteritis. *Seminars in Roentgenol.*, 1970, *5*, 245–259.
3. KZUKA, T., NOSAKI, T., SATO, K., and IHARA, K. Aortitis syndrome with special reference to pulmonary vascular changes. *Acta radiol.* (Diag.), 1968, 7, 25–32.
4. LANDE, A., and BARD, R. Takayasu's arteritis: unrecognized cause of pulmonary hypertension. Unpublished.
5. LANDE, A., and GROSS, A. Total aortography in diagnosis of Takayasu's arteritis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *116*, 165–178.
6. LANDE, A., and ROSSI, P. Value of total aortography in diagnosis of Takayasu's arteritis. To be published.
7. ORELL, S. R., KARNELL, J., and WAHLGREN, F. Malformation and multiple stenoses of pulmonary arteries with pulmonary hypertension. *Acta radiol.*, 1960, *54*, 449–459.
8. PALEHEIMO, J. A. Obstructive arteritis of Takayasu's type: clinical, roentgenological and laboratory studies on 36 patients. *Acta med. scandinav.*, 1967, Suppl. 467.
9. SCHRIRE, V. Arteritis of aorta and its major branches. *Australasian Ann. Med.*, 1967, *16*, 33–40.
10. SHIMIZU, K., and SANO, K. Pulseless disease. *J. Neuropath. & Clin. Neurol.*, 1951, *1*, 37–47.
11. TAKAYASU, M. Case with peculiar changes in retinal vessels. *Acta Soc. Opth. Jap.*, 1908, *12*, 554.

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ORIFICE-VIEW ROENTGENOGRAPHY FOR EVALUATION OF THE AORTIC VALVE*

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ABSTRACT:

Plain orifice-view roentgenograms permit measurement of the area circumscribed by calcium in patients with heavily calcified aortic valves. The measurements relate well to the actual size of the orifice. Therefore, this roentgenographic technique serves in a practical fashion as a non-invasive method for the assessment of the severity of the aortic stenosis in such patients. During the injection of contrast material, orifice-view roentgenograms serve as a useful adjunct to standard aortography. Such views permit assessment of the valvular leaflets, size of the aortic ring, and the degree of stenosis.

ORIFICE-VIEW roentgenography of the aortic valve is a method for evaluation of the aortic valve by means of a roentgenographic projection which permits visualization of the valve as if one were looking directly into the orifice. This view is achieved by angling the roentgen-ray beam obliquely across the heart, approximately from the right axilla to the left iliac crest.[2] Specifically, the beam of the roentgen ray is directed obliquely at a superior elevation of 45° with the patient in a supine position (Fig. 1). The method has been shown to be of value during the injection of contrast material as an adjunct to standard aortography.[2,3] It is also of value as a non-invasive, roentgenographic procedure for the evaluation of the severity of aortic stenosis in patients with heavily calcified aortic valves.[4,5]

BACKGROUND

The projection of value for orifice-view roentgenography was determined by studies of the position of the metallic annulus of prosthetic aortic valves[2] (Fig. 2). In 31 patients it was shown that the aortic valve has a rightward tilt of 39 ± 2 degrees (mean $\pm$ SE). If the beam of the roentgen ray were projected obliquely across the heart at an angle of 45°, the projected diameter in the frontal plane would be within 95 percent accuracy in 94 percent of patients[2] (Fig. 3). This is based upon the following trigonometric relation:

Projected diameter

$$= \text{Actual diameter} \times \cos \Delta$$

where Δ = angle by which projection at 45° differs from a true perpendicular. For angles of error of 19° or less the projected diameter would be 95 percent of the actual

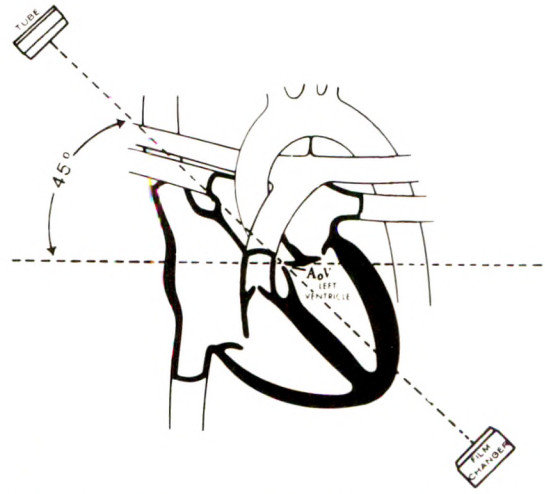

FIG. 1. Position of roentgen-ray tube and film changer utilized for visualization of orifice of aortic valve. Tube is elevated 45° superiorly. Patient is supine.

* From the Department, of Medicine, University of Oklahoma College of Medicine, and VA Hospital, Oklahoma City, Oklahoma.
Supported by the VA Research Service.

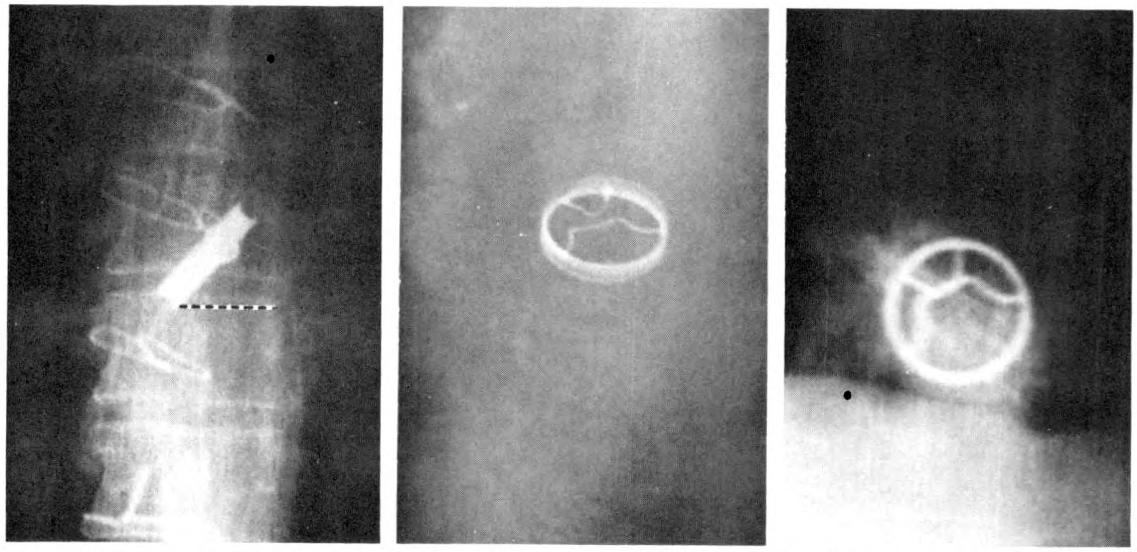

FIG. 2. Roentgenograms of patient with Björk Shiley prosthesis. Films obtained in anteroposterior projection (left), lateral projection (center), and 45° superior elevation (right). In the anteroposterior projection, the annulus of the valve tilts rightward at an angle of 45° from the horizontal (dotted line). Film taken obliquely across the heart at an angle of 45° shows valve as if looking directly into orifice (right).

diameter, since the cosine of such angles varies between .95 and 1.

The aortic valve was shown to tilt anteriorly in 81 percent of patients and posteriorly in 19 percent of patients,[2] the average tilt being anterior at 10 ± 3 (Fig. 4). With no elevation in the frontal plane, the projected diameter in this projection would be within 95 percent accuracy in 71 percent of patients and within 90 percent accuracy in 87 percent of patients.[2] A standard projection of 45° superior eleva-

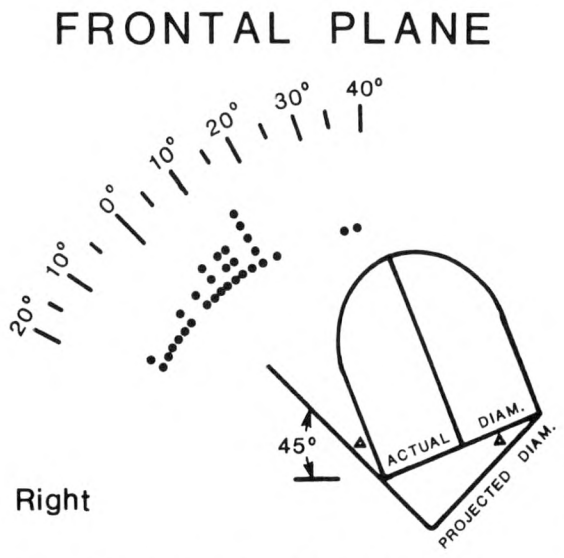

FIG. 3. Distribution of angle of error (Δ) in frontal plane. If all roentgenograms were taken at an oblique superior elevation of 45°, then the indicated angles would represent the deviation from a true perpendicular.

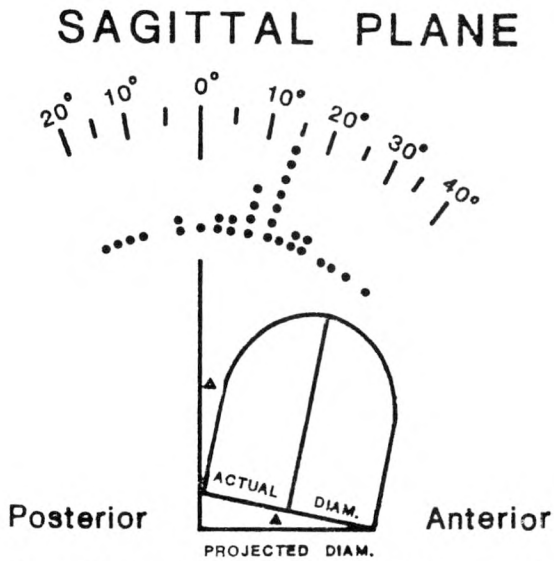

FIG. 4. Distribution of angle of error (Δ) in sagittal plane. If the beam of the roentgen ray were not tilted either anteriorly or posteriorly then the indicated angles would represent the deviation from a true perpendicular.

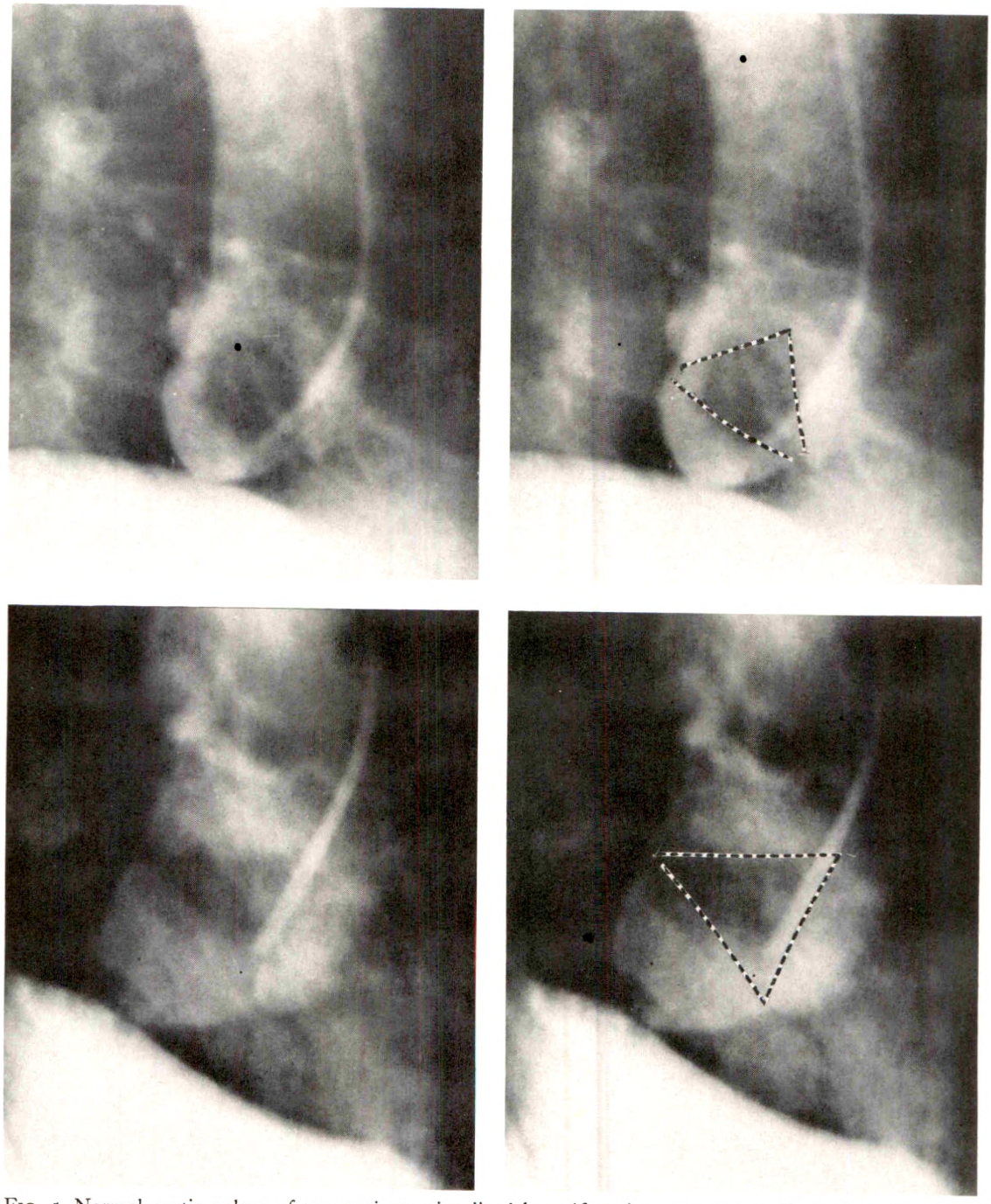

Fig. 5. Normal aortic valves of two patients visualized by orifice-view aortograms. Both show triangular configuration of orifice during ejection. Right, orifice of each outlined for clarity.

tion and no anterior elevation of the roentgen-ray beam would, therefore, permit one to measure the cross-sectional area of the orifice of the aortic valve with 90 percent accuracy in 81 percent of patients.[2]

Although the position of the valve in some patients differs from the normal, which would cause one to underestimate the size of the orifice by orifice-view roentgenography, one can avoid such an error by

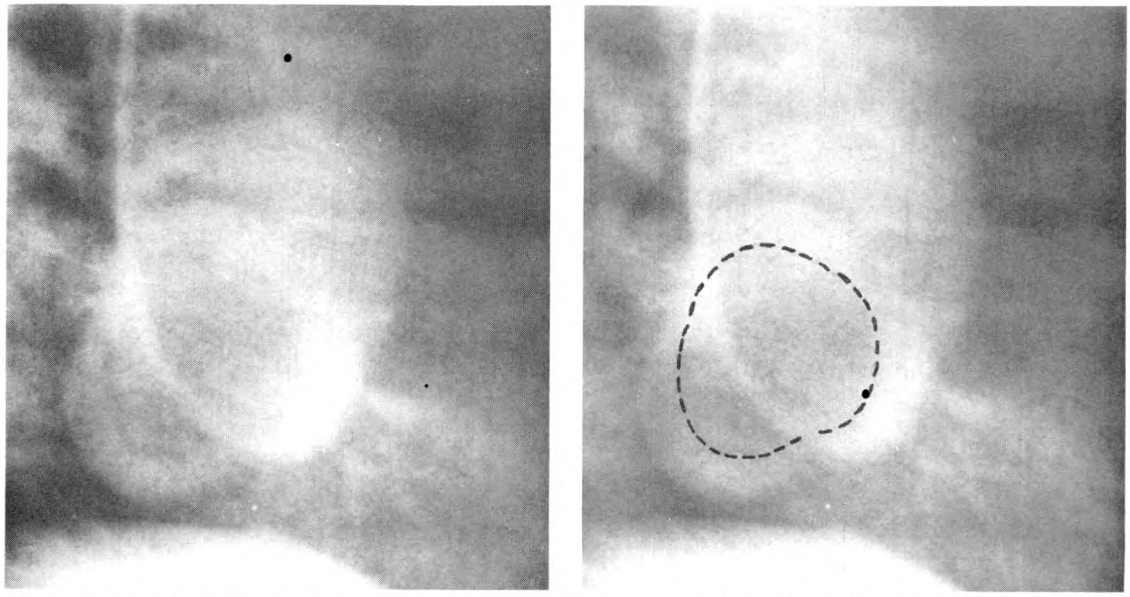

FIG. 6. Patient with severe aortic regurgitation. Orifice assumes nearly circular configuration to accommodate high stroke volume. Right, orifice outlined for clarity (dotted lines).

judging the orientation of the valve prior to making evaluations. During aortography by standard techniques (prior to orifice-view aortography) the orientation of the valve is readily apparent. In patients with heavily calcified aortic valves, in whom plain film orifice-view roentgenography is utilized, the orientation of the valve can be judged by the position of the calcium. In all instances, calculation of the actual size must be made after appropriate compensation for amplification.

ORIFICE-VIEW AORTOGRAPHY

Aortograms obtained in the oblique projection as described clearly delineate the

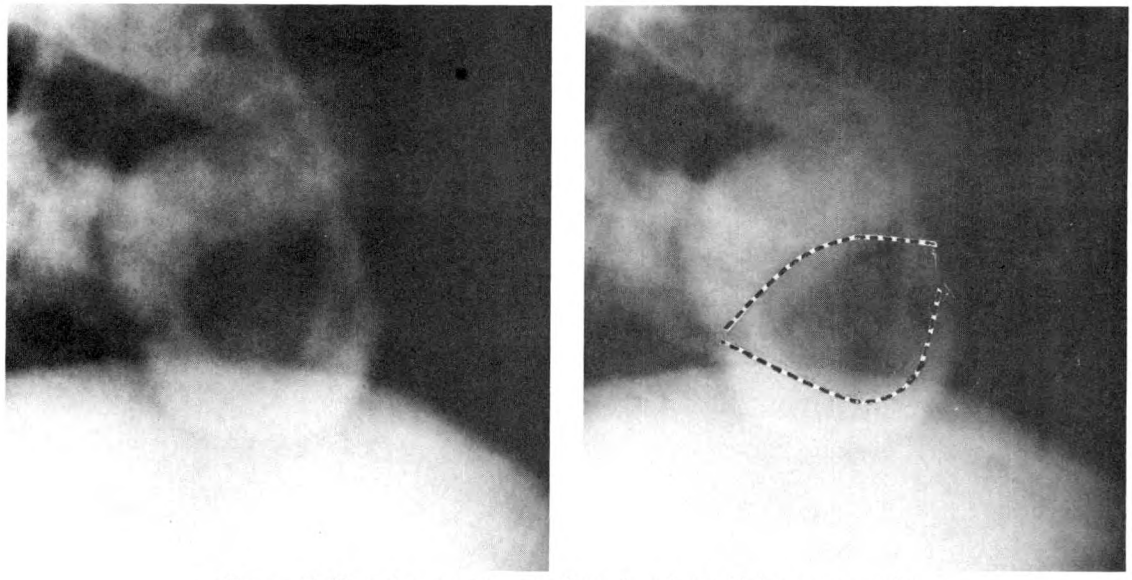

FIG. 7. Orifice-view aortogram of aortic valve which appears to be bicuspid. Patient has coarctation of the aorta.

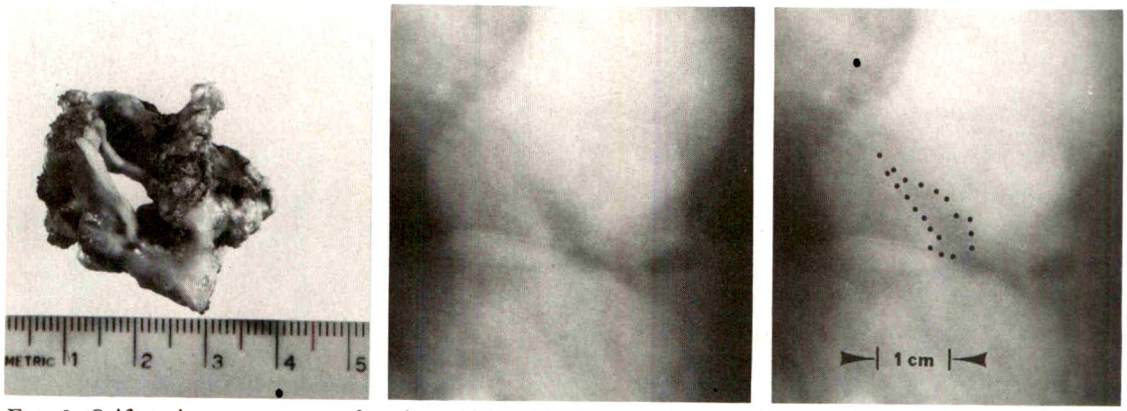

Fig. 8. Orifice-view aortogram of patient with critically severe aortic stenosis (right, orifice outlined for clarity). Left, photograph of surgically excised valve. Size and configuration of orifice as it appears on aortogram were comparable to actual orifice.

valvular leaflets and show the orifice during systole and diastole.[2] The triangular configuration of the leaflets during ejection as originally described by Leonardo da Vinci is readily recognized (Fig. 5). Previously unrecognized, however, is the fact that the orifice during ejection may assume a circular configuration[2] (Fig. 6). This has important physiological implications. It was shown that the size of the functional orifice is directly related to the stroke volume and rate of ejection of blood.[3] The aortic valve does not appear to open fully with the low cardiac output associated with the resting state. This suggests that the decreased size of the orifice due to a stenosing lesion would have an effect only during states of high cardiac output.

Aneurysmal enlargement of the valvular ring can readily be shown by orifice-view roentgenography.[1] Congenitally bicuspid valves can be identified (Fig. 7). Measurement of the extent of valvular stenosis is also possible by orifice-view aortography (Fig. 8).

HEAVILY CALCIFIED AORTIC VALVES

Plain orifice-view roentgenograms of patients with heavily calcified aortic valves can show the orifice with striking clarity[4] (Fig. 9). The dimensions of the valvular orifice circumscribed by calcium as shown on such roentgenograms and the dimensions as shown on roentgenograms of the surgically excised and reconstructed valves were measured. Comparison showed a good correlation ($r = .84$).[5] The orifice as circumscribed by calcium would be expected to be somewhat larger than the actual orifice due to a small inner rim of non-calcified material. In spite of this, the area outlined by calcium was small enough to demonstrate a tightly stenotic valve of one cm^2 or less in nine of twelve patients with heavily calcified valves which at surgery were proved to be critically stenotic (Table 1).[5] In three of twelve patients, the area circumscribed by calcium was 1.3 cm^2 to 1.7 cm^2 and this indicated an orifice of reduced size, although the actual size of the orifice was shown to be smaller. By implication, therefore, a fully circumferential ring of calcium, shown on orifice-view roentgenog-

Table I

Results of Plain Orifice-View Roentgenograms in 12 Patients with Critical Aortic Stenosis

(Surgically excised valve ≤ 0.7 cm^2)

Area Circumscribed by Calcium	No. of Patients	Percent
≤ 0.7 cm^2	6 of 12	50
0.8 to 1.0 cm^2	3 of 12	25
1.1 to 1.7 cm^2	3 of 12	25

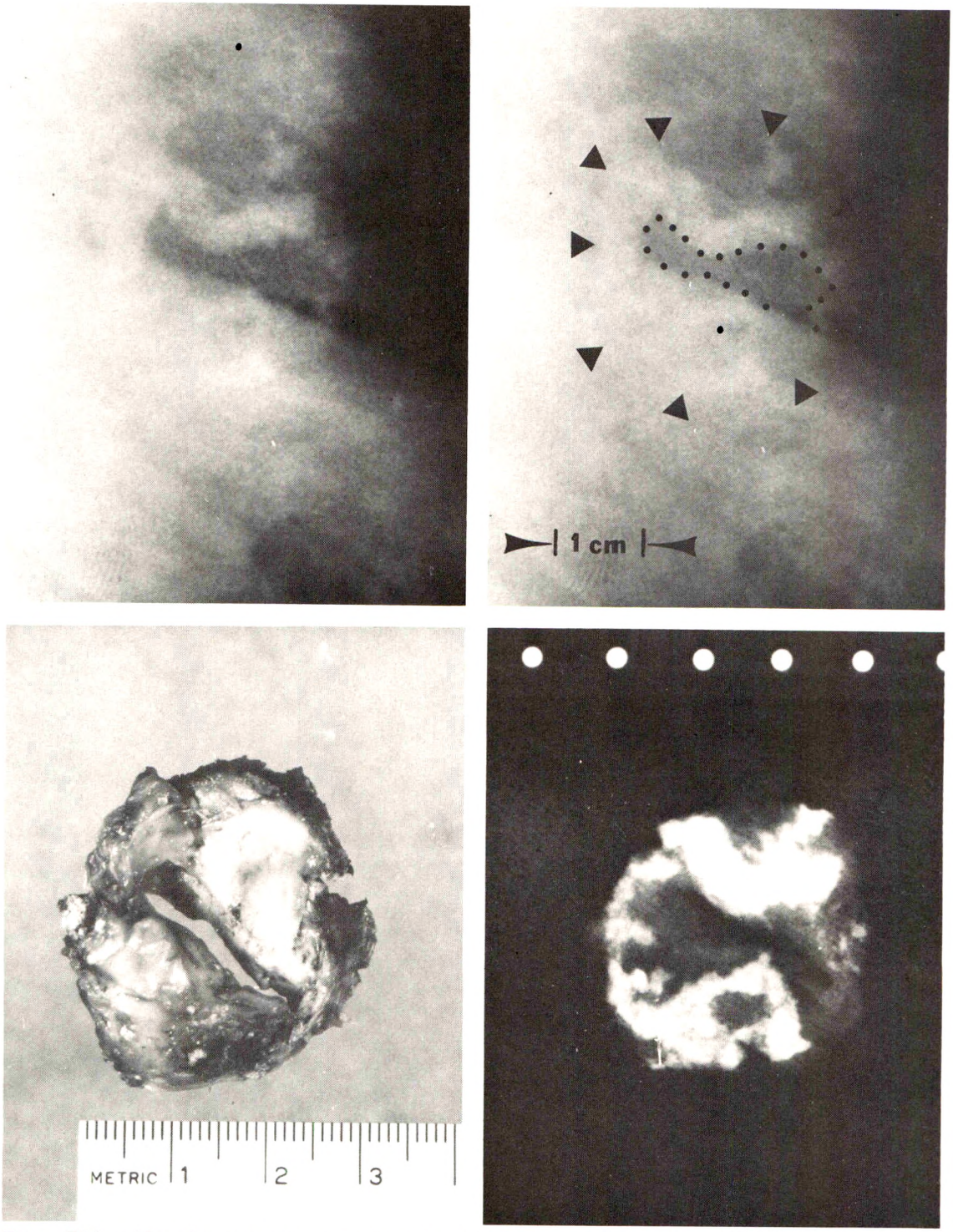

FIG. 9. Plain orifice-view roentgenogram of patient with heavily calcified aortic valve. Orifice, shown within the calcified valve, appears as an eccentric, elongated, radiolucent zone. For clarity, orifice is outlined by dots in duplicate illustration at right. The annulus is shown by arrows. Bottom left, excised aortic valve (same patient). Bottom right, roentgenogram of aortic valve (same valve). Radiopaque dots are placed at 1 cm intervals. The central radiolucent zone is the orifice. A nearly radiolucent (gray) rim of tissue is shown between the lumen and calcified region.

raphy, which defines an orifice of 1.7 cm^2 or less, indicates a critically stenosed aortic valve.[5]

The orifice-view technique requires attention to a variety of details in order to obtain satisfactory results. For the noninvasive evaluation of heavily calcified valves it has been found helpful to utilize fluoroscopy to distinguish the aortic valve from other radiodensities which may simulate a calcified valve. Pulsations indicative of the calcified valve are identified by fluoroscopy. A radio-opaque marker is then placed on the skin over the projection of the valve, and orifice-view roentgenograms are obtained. Having identified the valve on such films, the roentgenograms are then repeated without the identifying marker.

Compensation for magnification is readily accomplished after the position of the aortic valve is identified by anteroposterior fluoroscopy. Once the position of the valve is identified, the target-to-object and target-to-film distance can be measured and appropriate ratios can be calculated.* If cine is to be utilized, a scale is positioned at the level of the valve.

Since the amount of tissue which must be penetrated in these oblique views is greater than either in the posteroanterior or lateral projection, high voltage generators are desirable. Laminagrams may perhaps be of value, but they have not yet been utilized for this purpose.

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* Actual dimension = Apparent dimension $\times \frac{\text{target-to-object distance}}{\text{target-to-film distance}}$.

REFERENCES

1. Simon, A. P., and Stein, P. D. Aortic insufficiency in Ehlers-Danlos syndrome. *Angiology*, 1974, *25*, 290–296.
2. Stein, P. D. Roentgenographic method for measurement of cross-sectional area of aortic valve. *Am. Heart J.*, 1971, *81*, 622–634.
3. Stein, P. D. and Munter, W. A. New functional concept of valvular mechanics in normal and diseased aortic valves. *Circulation*, 1971, *64*, 101–108.
4. Stein, P. D. New roentgenographic assessment of anatomic deformity in calcific aortic stenosis. *Am. Heart J.*, 1972, *84*, 321–324.
5. Stein, P. D. Assessment of calcific aortic stenosis by measurement of area circumscribed by calcium on plain film orifice-view roentgenograms. *Chest*, 1974, *65*, 518–521.

ORIFICE-VIEW ROENTGENOGRAPHY OF THE MITRAL VALVE*

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ABSTRACT:

The mitral valve can be visualized as if looking directly into the valvular orifice by obtaining roentgenograms directed obliquely through the heart at a 25 degree superior elevation, with the patient rotated 60 degrees in the right anterior oblique direction. This view was based upon trigonometrical calculations of the spatial orientation of the annulus of prosthetic mitral valves in 25 patients. Calculations based upon measurement in these patients indicate that the area of the orifice of the mitral valve can be shown with less than ten percent error due to distortion of the projected image in 80 percent of patients. During the injection of contrast material into the left ventricle, orifice-view roentgenograms serve as a useful adjunct to standard ventriculograms. Such views permit assessment of the size of the mitral annulus and the degree of stenosis. Plain orifice-view roentgenograms of heavily calcified mitral valves permit measurement of the area circumscribed by calcium in such patients. The measurements indicate an upper limit of the possible size of the functional orifice. Therefore, this roentgenographic technique serves in a practical fashion as a non-invasive method for the assessment of the severity of mitral stenosis in such individuals.

THE purpose of this communication is to describe a roentgenographic technique for visualization of the mitral valve as if one were viewing it by looking directly into the orifice. This permits one to measure the mitral orifice area irrespective of the presence or severity of the stenosis. The equipment necessary for this technique is readily available in many cardiac catheterization laboratories. In patients with heavy circumferential deposits of calcium, the technique can be utilized non-invasively to assess the upper limits of the functional orifice area.

METHOD

PRELIMINARY STUDIES IN PATIENTS WITH PROSTHETIC MITRAL VALVES

Patients with prosthetic mitral valves served as standards for the determination of the spatial orientation of the annulus of the mitral valve. Since the ring of a prosthetic valve is metal, and therefore radiopaque, it was possible to determine the spatial orientation of the ring of the mitral valve from calculations based upon its position in plain frontal and lateral roentgenograms.

In the frontal plane (Fig. 1), the direction of flow through the orifice of the mitral valve is inferiorly and to the left. The orifice tilts at an angle, α, such that projections of the diameter of the orifice on the vertical axis (y) and on the horizontal axis (x) describe an angle, the tangent of which is y/x. That is, $\tan \alpha = y/x$.

In the sagittal plane, flow through the orifice is directed anteriorly and inferiorly. Projections of the diameter of the orifice upon the vertical axis (y) and upon the anteroposterior axis (z) describe an angle β, the tangent of which is y/z. Therefore, $\tan \beta = y/z$.

The position of the orifice of the mitral valve in the transverse plane (Fig. 1) can be calculated on the basis of the angles

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Supported by the V. A. Research Service.

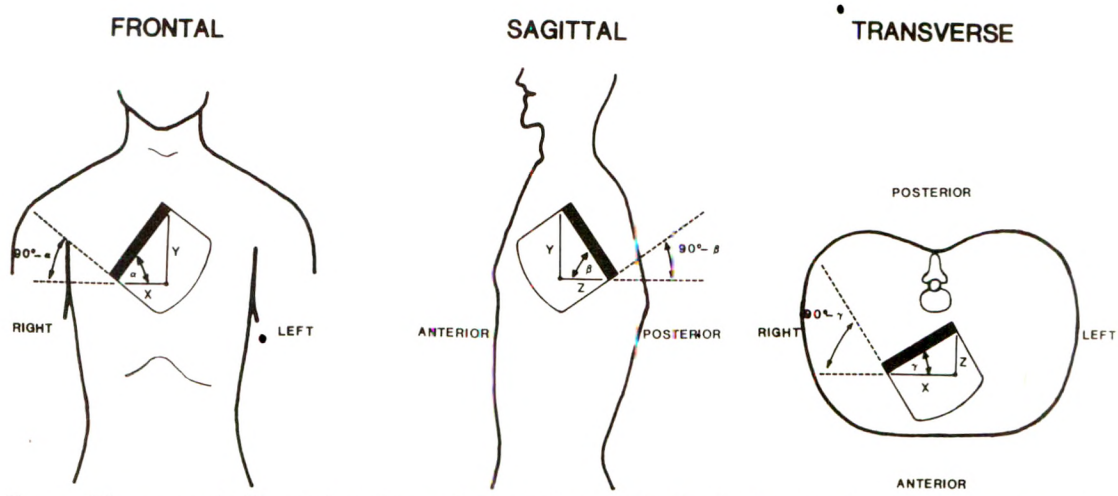

FIG. 1. Diagrammatic illustration of spatial orientation of mitral valve in frontal, sagittal, and transverse planes. In order to visualize the orifice, the roentgen-ray beam should be elevated superiorly at an angle of $(90\text{-}\alpha)$ degrees in the frontal plane. The patient should be rotated at an angle of $(90\text{-}\gamma)$ degrees in order to view the valve perpendicularly in the transverse plane.

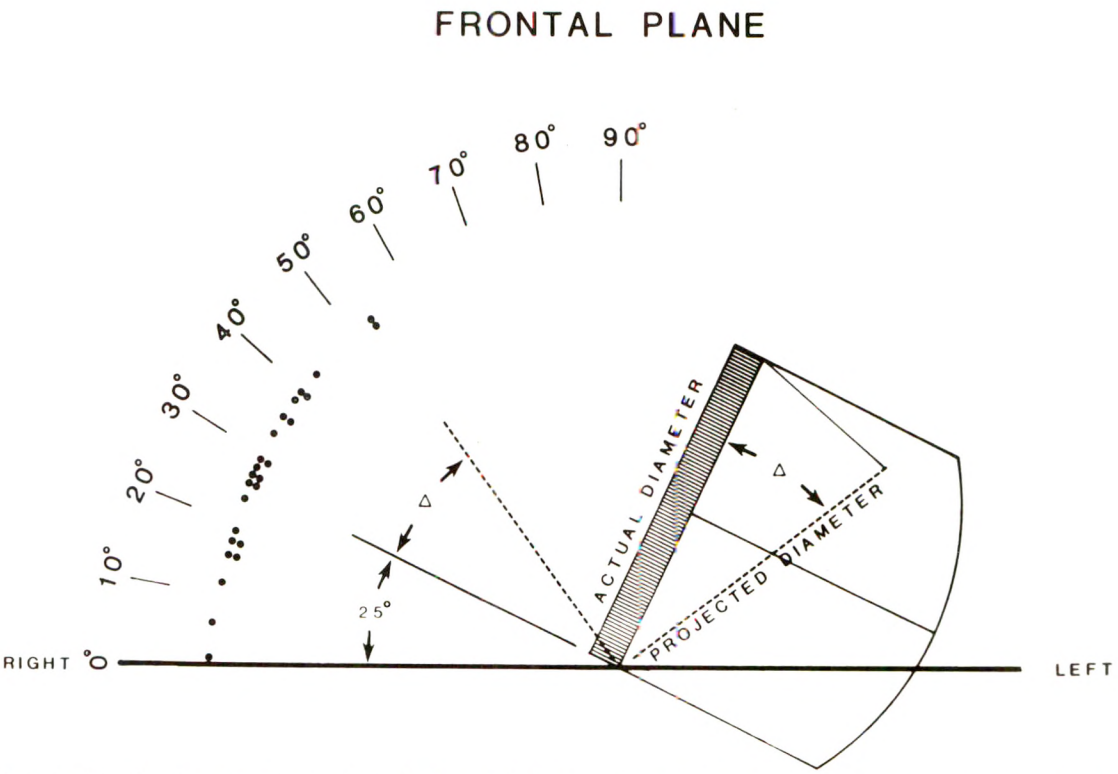

FIG. 2. Distribution of angles in frontal plane which were shown to be normal to the annulus of prosthetic mitral valves of 25 patients. If the roentgen-ray beam was routinely elevated 25 degrees, then a perpendicular to the orifice in each patient would differ from 25 degrees by an angle Δ. The projected diameter of the orifice would equal the actual diameter times $\cos \Delta$.

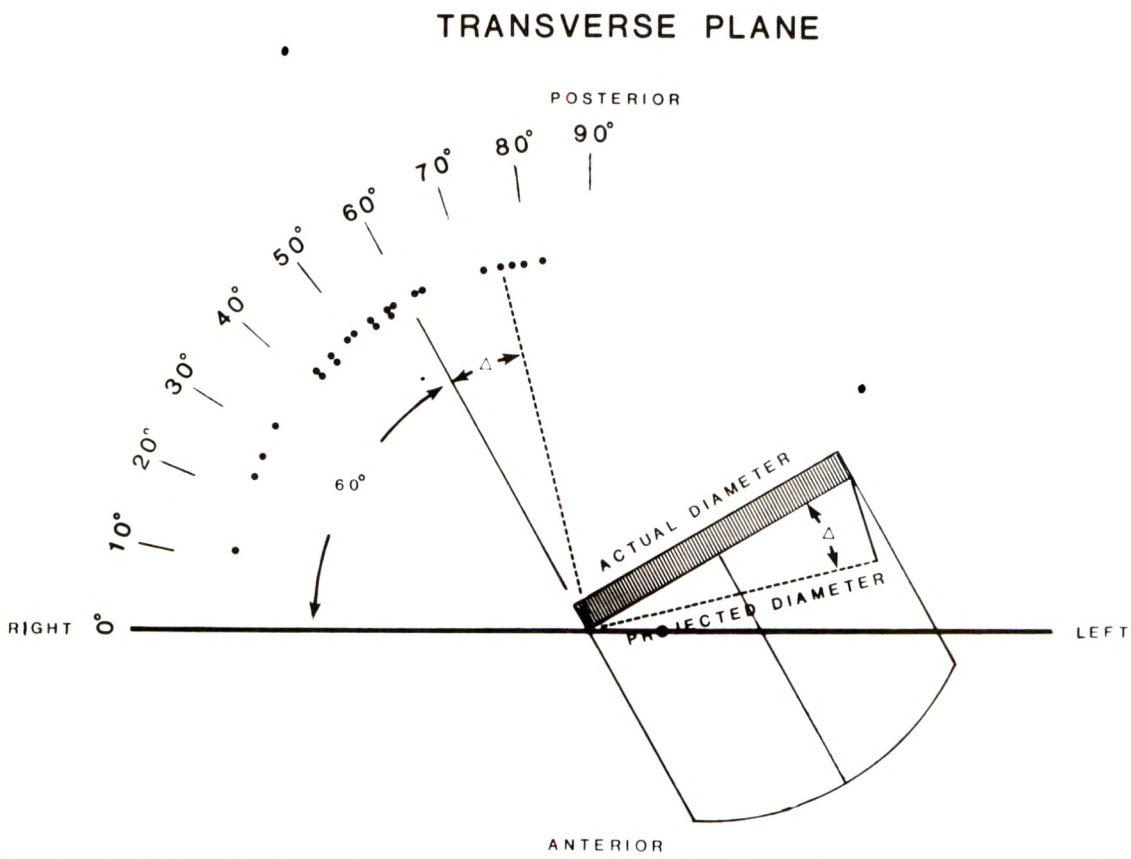

FIG. 3. Distribution of angles in transverse plane which were shown to be normal to the annulus of prosthetic mitral valves of 25 patients. If the patient was routinely rotated 60 degrees, then perpendiculars to the valve in this plane would differ from 60 degrees by an angle Δ. The projected diameter of the orifice would equal the actual diameter times cos Δ.

which described its tilt in the frontal and sagittal projections. In the transverse plane, flow through the mitral valve is directed anteriorly and to the left at an angle γ, the tangent of which is z/x. That is, tan $\gamma = z/x$. Since $y = x \tan \alpha$, and $y = z \tan \beta$, then $z/x = \tan \alpha / \tan \beta$. Therefore, the anterior and leftward tilt, γ, can be determined from the angles of the tilt in the frontal and sagittal planes.

For visualization of the mitral orifice without distortion, the beam of the roentgen-ray should be perpendicular to the plane of the orifice of the valve. To accomplish this, the tube should be elevated superiorly at an angle of $(90-\alpha)$ degrees, and the patient should be rotated counterclockwise (or the tube rotated clockwise) by an angle $(90-\gamma)$ degrees. Calculations in these patients with mitral prosthetic valves were made to determine the value of angles α and γ.

Frontal chest roentgenograms (usually posteroanterior) were available in 25 patients with prosthetic mitral valves. The angle of flow into the orifice (the angle perpendicular to the orifice) is shown in these patients (Fig. 2). It was 27 ± 3 degrees (mean $\pm$ SD). If the roentgen-ray beam routinely were directed obliquely across the chest at an angle of 25 degrees in the frontal plane, then a true perpendicular to the valve would differ from 25 degrees by an angle Δ. The projected diameter would equal the product of the actual diameter times cos Δ (Fig. 2). With an oblique frontal plane projection of 25 degrees, the projected diameter would differ from the actual di-

Fig. 4. Projection utilized for orifice-view of mitral valve. The roentgen-ray beam was elevated 25 degrees superiorly from the horizontal. The patient was rotated 60 degrees in the right anterior oblique direction.

ameter by ten percent or less in 24 of 25 patients (96 percent), and it would differ by twelve percent in one patient.

The angle of flow into the mitral valve (the angle shown as $90°-\gamma$ in Fig. 1) in the transverse plane was calculated from anteroposterior and lateral roentgenograms in 25 patients (Fig. 3). It was 55 ± 4 degrees. If the patients routinely were rotated counterclockwise 60 degrees, the projected

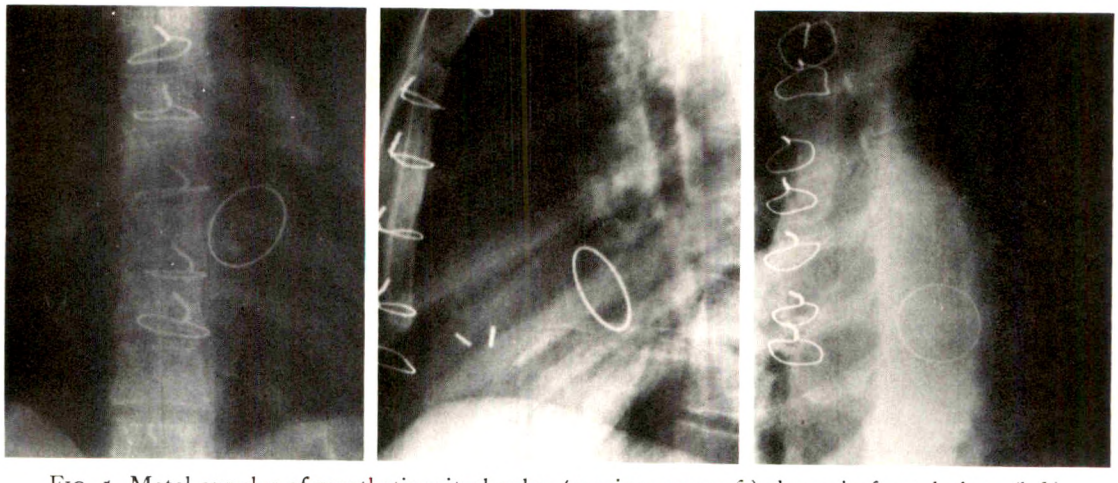

Fig. 5. Metal annulus of prosthetic mitral valve (porcine xenograft) shown in frontal plane (left), sagittal plane (center), and at orifice-view projection (right).

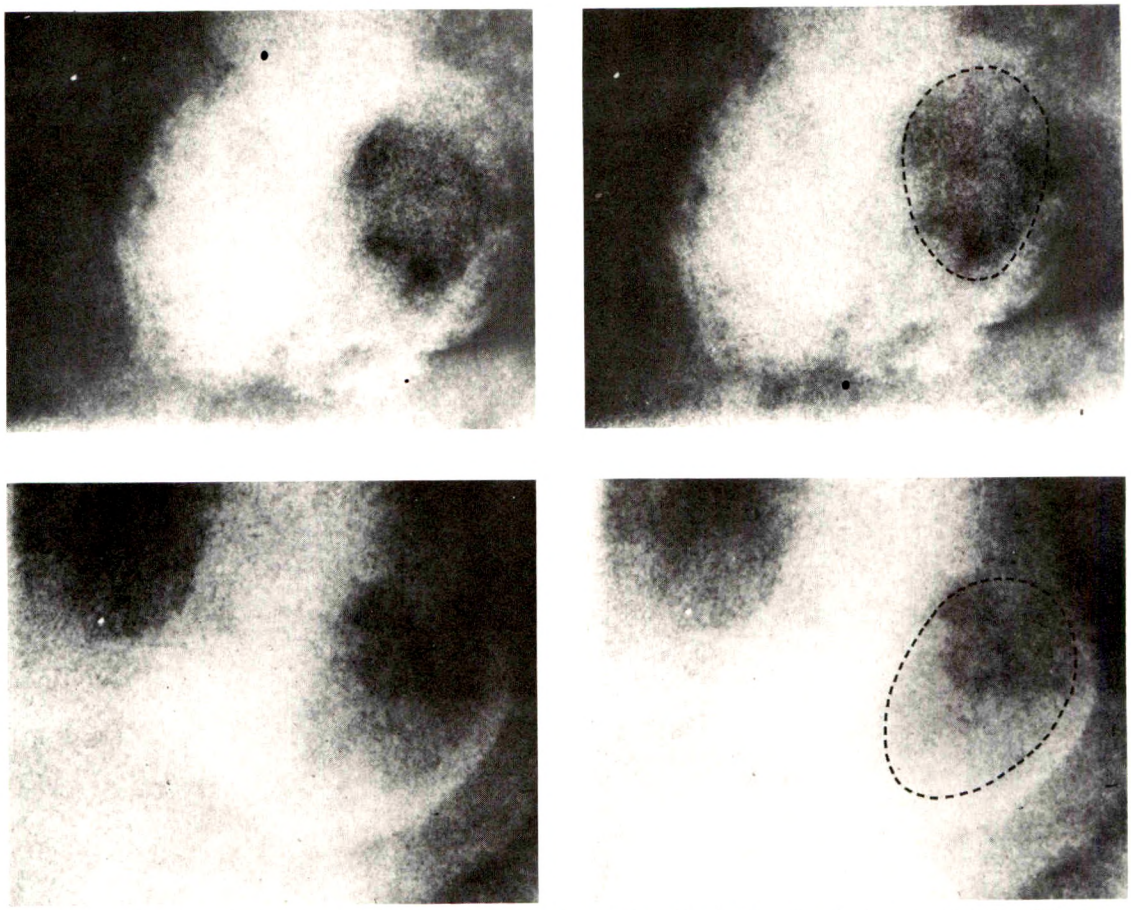

FIG. 6. Cineventriculogram of two patients showing orifice of mitral valve during ventricular filling. (Orifice of each outlined at right.) Top, orifice area 4.3 cm^2. Bottom, orifice area 4.6 cm^2.

diameter in the transverse plane would differ from the actual diameter by ten percent or less in 21 of 25 patients (84 percent). In three patients, the projected diameter in this view would differ by twelve to 20 percent; and in one patient it would differ by 34 percent. This rotation of 60 degrees showed the diameter of the mitral valve in the transverse plane with the smallest error due to projection in most patients, although the magnitude of the error in occasional patients is higher than it would be with a smaller rotation.

These calculations, based upon the spatial orientation of the sewing rings of prosthetic mitral valves, showed that the orifice of the mitral valve can be visualized in most patients with the least distortion if the roentgen-ray beam were angled obliquely across the heart 25 degrees superiorly in the frontal plane with the patient rotated counterclockwise 60 degrees (Fig. 4). Measurements of the cross-sectional area would be less accurate than measurements of a single diameter, because errors due to projection of both the major and minor dimensions would be multiplied. Even so, this orifice-view projection of the mitral valve would permit measurement of the cross-sectional area with at least 90 percent accuracy in 80 percent of patients (20 of 25 patients).

The ring of a prosthetic mitral valve is shown in the frontal and sagittal projections, and at the orifice-view projection described above (Fig. 5). Calculations of the actual size were made after appropriate compensation for amplification. On cut

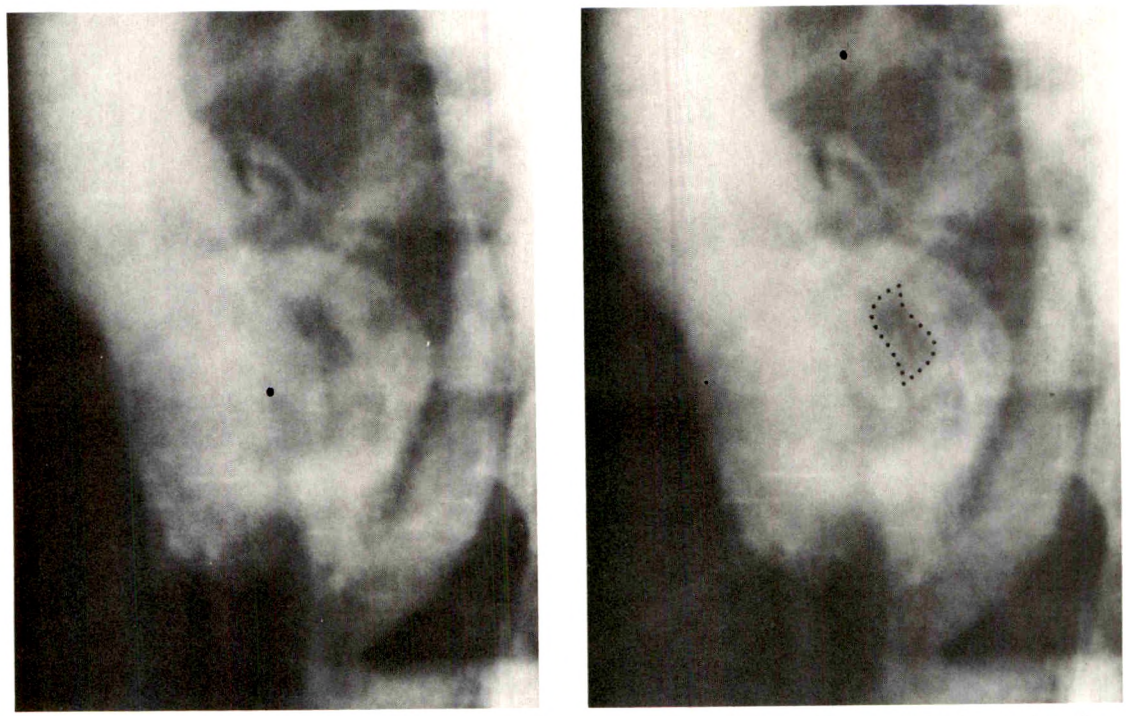

Fig. 7. Orifice-view ventriculogram of patient with mitral stenosis. Right, orifice outlined for clarity. Area of mitral orifice measured from orifice-view roentgenogram was c.6 cm². Estimated area of valve at surgery was 1.0 cm².

films, this was accomplished by multiplication of the apparent diameter by the ratio of the target-to-object distance divided by the target-to-film distance. On cineventriculograms, a scale was placed at the position of the mitral valve.

ORIFICE-VIEW VENTRICULOGRAPHY

Most ventriculograms were performed on 35 mm cine film at 30 frames per second during the injection of 40 ml of 75 percent sodium and meglumine diatrizoates at a rate of 10 ml/sec. Films were pulsed at 10 msec durations at 125 kV and 230 ma. A nine inch image intensifier (North American Philips, Long Island, N. Y.) was utilized with a 1,000 ma generator. Some of the ventriculograms of patients with mitral stenosis were recorded on 14 inch by 14 inch films taken at a rate of four per second on an Elema Schönander (Mount Prospect, Ill.) rapid film changer. Such films were obtained with an exposure time of 0.10 sec at 80 to 130 kV and 500 ma.

Orifice-view ventriculograms showed the outline of the mitral valve as the rapid inflow of blood displaced contrast material within the ventricle. The orifice of the mitral valve occupied much of the cross-sectional area of the ventricle of patients with normal valves (Fig. 6). The mitral orifice appeared ellipsoidal.

The outline of stenotic orifices can be shown with clarity by the jet of blood entering the opacified ventricle (Fig. 7; and 8). Rigid valves could be shown with openings both in systole and diastole (Fig. 8). The size of the stenosed orifices, as judged by cineventriculograms, was comparable to the orifice as measured directly on the excised valve (Fig. 8).

The posterior papillary muscle may simulate a stenotic orifice. Confusion can be avoided by being careful to measure the area of the mitral orifice during diastole. At this time the posterior papillary muscle is not apparent. During systole, the orifice of stenotic valves usually is inapparent,

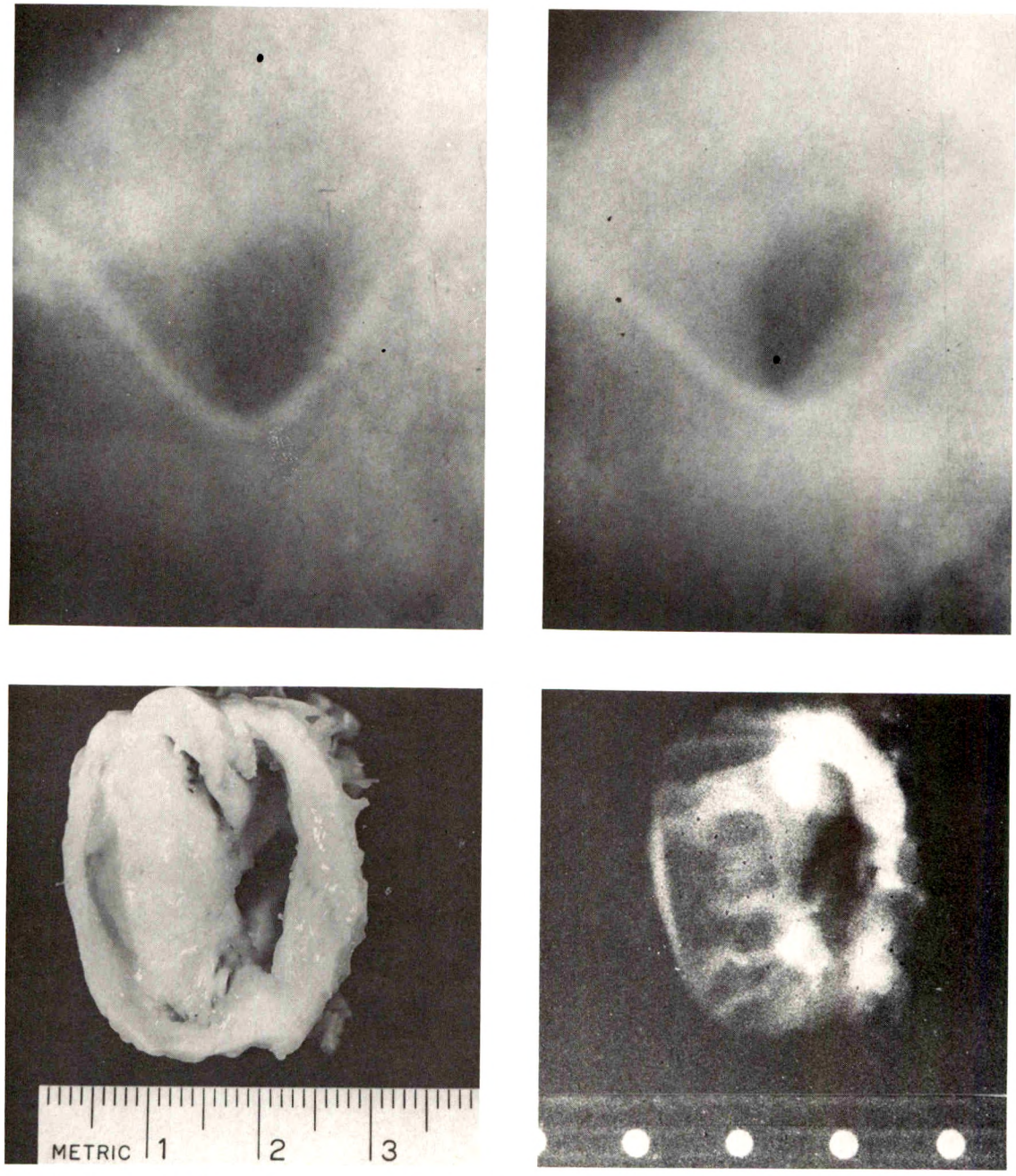

FIG. 8. Frame of orifice-view cineventriculogram showing stenotic orifice during diastole (top left), and systole (top right). Area of orifice during diastole measured on cineventriculogram was 1.5 cm^2. Area circumscribed by calcium on excised valve (bottom right) was 0.9 cm^2. Functional orifice area (bottom left), as measured from photograph of excised valve, was 0.7 cm^2.

and the posterior papillary muscle is visualized as a radiopaque circle.

In one patient with tight mitral stenosis, the mitral orifice appeared normal. In retrospect, the injection of contrast material into the left ventricle was in the outflow tract of the ventricle near the aortic valve. It would seem that streaming of contrast material simulated a normal orifice.

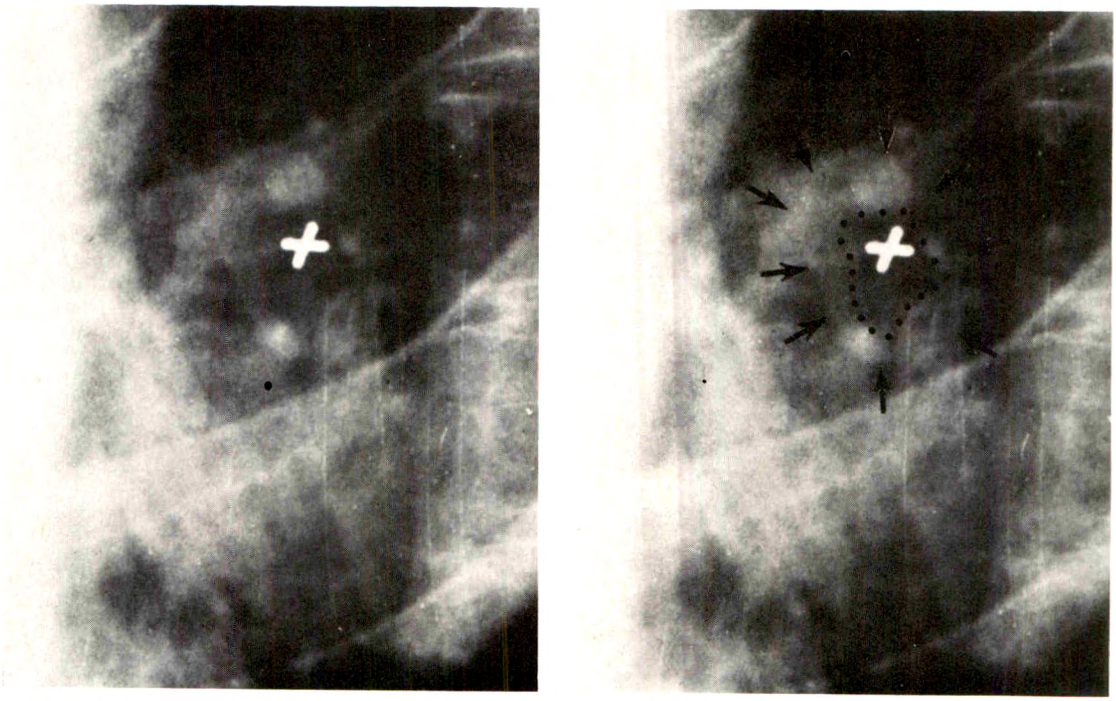

Fig. 9. Plane orifice-view roentgenogram of heavily calcified mitral valve. Opaque marker (x) was positioned on chest during fluoroscopy in order to identify orifice. Area circumscribed by calcium was 0.9 cm^2. Area of orifice calculated by hydraulic equation[2] was 1.0 cm^2. Right, orifice outlined by dots, and rim of calcium indicated by arrows.

Plain Orifice-View Roentgenograms of Heavily Calcified Mitral Valves

Plain orifice-view roentgenograms of patients with heavily calcified mitral valves were obtained at an approximate exposure time of .10 sec at 500 ma and 100 kV. A grid cassette with high-speed screens was utilized.

The area circumscribed by calcium represents the maximum possible size of the functional orifice. Since a rim of noncalcified fibrous tissue would be expected between the functional orifice and the calcified ring, the functional orifice of the valve would be smaller than the area circumscribed by calcium. The area circumscribed by calcium was measured in five patients with calcific mitral stenosis (Fig. 9; and 10). This area ranged between 0.8 cm^2 and 1.3 cm^2, which itself represents tight mitral stenosis. In one patient the valve was excised intact during surgery, and comparisons of the area circumscribed by calcium on orifice-view roentgenograms were made with the actual functional orifice area, and with the area circumscribed by calcium on the excised valve (Fig. 10).

Discussion

Visualization of the mitral valve at this orifice-view projection gives information related to the size of the mitral orifice that is not apparent from standard ventriculograms. Mitral stenosis can be recognized on lateral or oblique ventriculograms by the presence of a diaphragm and constriction of the stream flowing into the ventricle.[1] However, measurement of the orifice on such films is usually made in only one projection,[1] and therefore is only approximate. Mitral regurgitation is not well appreciated on orifice-view ventriculograms. Therefore, it would seem that orifice-view ventriculograms would be of supplementary value to previously established projections.

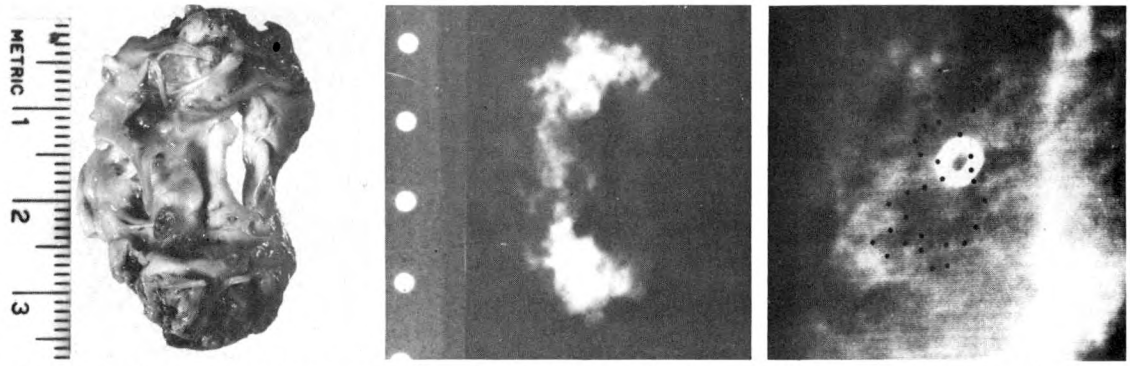

FIG. 10. Orifice-view roentgenogram of heavily calcified mitral valve (right). Orifice was identified by radiopaque marker (o) during fluoroscopy. Area circumscribed by calcium was 1.3 cm^2. Excised valve is shown at left. Functional orifice area was 0.5 cm^2. Roentgenogram of excised valve is shown at center. Some of the heavily calcified tissue was not included with this specimen. Area inscribed by calcium (extrapolating for missing segment) was 1.3 cm^2.

The size of the functional mitral orifice can be measured from orifice-view roentgenograms, irrespective of the severity of narrowing. Since hydraulic equations require the measurement of a gradient across the valve, which occurs only after the orifice has narrowed considerably,[2] this is a potential advantage. Patients with mild narrowing of the valve may be identified by this method. Regurgitant flow does not interfere with the orifice-view roentgenograms of mitral valves. It does interfere, however, with hydraulic calculations of the area of the orifice because the magnitude of regurgitant flow cannot readily be calculated.

Incomplete opening of the mitral valve, due to a low stroke volume, presumably can simulate a small orifice. The size of the functional orifice of aortic valves has been shown to be dependent upon flow,[4] and it is likely that this is true of mitral valves. Eddies in the periphery of the inflow jet through the mitral valve presumably can simulate a larger than actual orifice. Even so, in the majority of patients, this method of visualization of the mitral valve appears to give useful information regarding the size of the mitral orifice and the motion of the leaflets.

As would be expected, some patients are too large to permit penetration of the roentgen-ray beam on a diagonal line across the chest. Adequate amounts of contrast material within the ventricle are also required. The orifice can best be visualized if the injection of the contrast material is near the mitral valve or well within the cavity of the left ventricle.

For the non-invasive evaluation of heavily calcified valves, it has been found helpful to utilize fluoroscopy to distinguish the calcified mitral valve from other radiodensities which may simulate a calcified valve. Pulsations indicative of the calcified valve are identified by fluoroscopy, and a radiopaque marker is placed on the skin over the projection of the valve. Then orifice-view roentgenograms are obtained. Having identified the valve on such films, the roentgenograms are then repeated without the identifying marker.

The orifice-view projection described in this study enables one to measure the area of the mitral orifice in 80 percent of patients with less than ten percent error due to distortion of the projected image. However, occasional patients show significant distortion of the image at this projection. A more advantageous projection in such patients perhaps could be selected if the spatial orientation of the valve were estimated during standard cineventriculograms, which may sometimes be possible. During the plain roentgenographic evaluation of heavily calcified valves, the orientation of the calcified annulus can be determined from the posteroanterior and

lateral roentgenograms, and the beam of the roentgen-ray can be positioned perpendicular to the ring. Therefore, in the occasional patient with an unusual orientation of the calcified mitral valve, a more advantageous projection can be selected.

The mitral orifice can also be shown non-invasively by two-dimensional echocardiography.[3] Heavily calcified mitral valves, however, may not be visualized by two-dimensional echocardiography;[3] whereas, heavy circumferential deposits of calcium are necessary for the non-invasive roentgenographic evaluation of the valve. In this regard, the techniques may be complementary.

Orifice-view roentgenography has been utilized for evaluation of the aortic valve.[5,6] In patients with heavy circumferential deposits of calcium around the aortic valve, orifice-view roentgenograms have been shown to be useful for the non-invasive evaluation of the severity of calcific aortic stenosis.[7] The projection required for visualization of the aortic orifice differs from that required for visualization of the mitral orifice.

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We thank Dr. Ronald C. Elkins for excising the mitral valve intact from some of the patients illustrated in this study.

REFERENCES

1. FIGLEY, M. M. Angiocardiography in valvular heart disease: morphologic and volumetric considerations. *Radiol. Clin. North America*, 1964, *2*, 409–423.
2. GORLIN, R., and GORLIN, S. G. Hydraulic formula for calculations of area of stenotic mitral valve, other cardiac valves, and central circulatory shunts. *Am. Heart J.*, 1951, *41*, 1–29.
3. HENRY, W. L., GRIFFITH, J. M., MICHAELIS, L. L., MCINTOSH, C. L., MORROW, A. G., and EPSTEIN, S. E. Measurement of mitral orifice area in patients with mitral valve disease by real-time two-dimensional echocardiography. *Circulation*, 1975, *51*, 827–831.
4. STEIN, P. D., and MUNTER, W. A. New functional concept of valvular mechanics in normal and diseased aortic valves. *Circulation*, 1971, *64*, 101–108.
5. STEIN, P. D. Roentgenographic method for measurement of cross-sectional area of aortic valve. *Am. Heart J.*, 1971, *81*, 622–634.
6. STEIN, P. D., and SABBAH, H. N. Orifice-view roentgenography for evaluation of aortic valve. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1975, *125*, 847,-853.
7. STEIN, P. D. Assessment of calcific aortic stenosis by measurement of area circumscribed by calcium on plain film orifice-view roentgenograms. *Chest*, 1974, *65*, 518–521.

THE RECONSTRUCTED ESOPHAGUS*

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ABSTRACT:

Reconstruction of the esophagus has been used for more than half a century. Benign as well as malignant diseases are treated in this manner. Viscera used for reconstruction are: colon, jejunum, and stomach. The different modalities of surgical intervention have been briefly summarized. Complications have been enumerated with the major ones stressed in more detail.

The exact nature of surgical procedure should be known before attempting to evaluate roentgenologically a reconstructed esophagus. Preoperative roentgen evaluations, including barium study and angiography, are indicated for better postoperative results. Postoperative roentgen studies are needed to detect most of the postoperative complications.

RESTORATION of a poorly functioning or an obstructed esophagus has been the aim of surgeons for the past seven decades. Only since the advent of antibiotics, however, has visceral replacement of the esophagus become more successful. In contrast to gastrostomy, a palliative measure only, a reconstructed esophagus can offer to the patient a relatively normal existence by preserving the essential pleasures of eating and drinking.

The task of reconstructing the esophagus, by using other viscera, is not simple and many criteria have to be fulfilled before a neo-esophagus is declared properly functioning. There must be normal swallowing, "normal" vomiting, no significant reflux, no dumping, no anemia, no diarrhea, and no malabsorption. The reconstructed esophagus should not be digested by hydrochloric acid, or by duodenal or jejunal contents.

Six terms are currently used to designate these procedures: esophageal bypass, colon interposition, esophageal transplant, esophageal reconstruction, neo-esophagus, esophagoplasty. Although indicating a reconstruction of the esophagus, these terms do not specify the exact nature of the procedure.

The purpose of this communication is: (1) to summarize the different surgical modalities; and (2) to review the complications which may occur in conjunction with a reconstructed esophagus. This hopefully will make the roentgenological evaluation of such patients more meaningful.

INDICATIONS FOR RECONSTRUCTION

The indication for reconstruction of the esophagus is, in general, a poorly functioning or an obstructed esophagus.

In infants and children indications are: atresia of the esophagus with or without fistula; stricture following chemical burn; and esophageal varices with extrahepatic obstruction.[6,18,19] In adults, the esophagus may be reconstructed for the following benign conditions: achalasia; scleroderma; benign tumors; benign esophageal fistula; and strictures following instrumentation, nasogastric tube placement, chemical burns, or peptic esophagitis.[4,7,16]

Cancer of the esophagus, however, appears to be the most common lesion treated today by esophageal reconstruction.[4,24]

SURGICAL MODALITIES

A brief review of the historical develop-

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ment of the different modalities of reconstruction of the esophagus brings out the controversy surgeons have had in choosing the viscus or the location for reconstruction. Early the reconstructed esophagus was placed subcutaneously on the anterior chest wall. In an attempt to overcome technical problems, various viscera were used: a skin tube by Bircher in 1894; the jejunum by Roux and Herzen in 1907; the large bowel by Vulliet and Kelling in 1911; and the stomach by Kirschner in 1920.[21] In the past two decades, stomach, jejunum, and colon have been used to reconstruct the esophagus, but the reconstruction is usually performed in the anterior mediastinum.

There are now three established locations where the esophagus can be reconstructed: (1) subcutaneously on the anterior chest wall; (2) in the anterior mediastinum, in the retrosternal space; and (3) intrathoracically and intrapleurally in or about the esophageal bed. Viscera used today are segments of stomach, small bowel, or colon. The procedures utilized today for reconstruction are given in Table 1.

TABLE I

SURGICAL PROCEDURES FOR RECONSTRUCTION OF THE ESOPHAGUS

1. Use of Colon
   - Right colon in isoperistaltic position with terminal ileum[2,3]
   - Right colon in isoperistaltic position without terminal ileum and cecal pole[5,6,18]
   - Right colon in antiperistaltic position with the terminal ileum preserved or interposed[23]
   - Transverse colon in isoperistaltic position[10,13,22]
   - Left colon in isoperistaltic position[3,6,12]
   - Left colon in antiperistaltic position[4,18,20,24]
2. Use of the Jejunum
   - Esophagojejunogastrostomy[2,7]
   - Esophagojejunoduodenostomy[2,9]
   - Esophagojejunostomy[3]
3. Use of Stomach
   - Reversed gastric tube[7,8,10]
4. Location for Reconstruction
   - Subcutaneous (anterior chest wall)[3,8]
   - Retrosternal (extrapleural)[3,5,6,8]
   - Posterior (in the esophageal bed or intrapleural)[2,4,7–9]

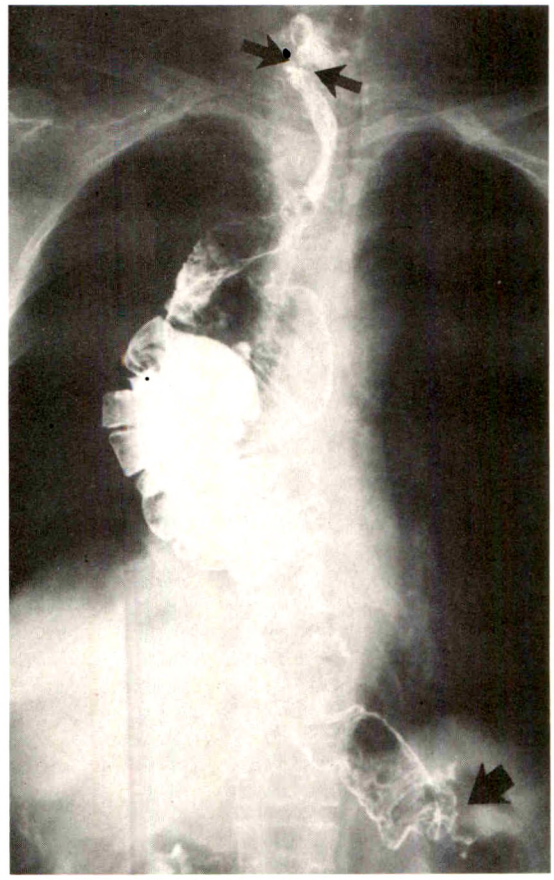

FIG. 1. Barium swallow examination of an 87 year old woman who had carcinoma of the esophagus resected with reconstruction using the right colon with the terminal ileum preserved. The proximal anastomosis (small arrows) is end-to-side, as is the distal anastomosis, high on the lesser curvature, close to the fundus (large arrow). The patient received postsurgical super voltage irradiation and died six months later from bronchopneumonia. (Courtesy Stefan Arnon, M.D., University of California at Davis.)

By far, the most popular organ used for esophageal reconstruction is the colon. The colon offers an adequate length, good marginal blood supply, and a resistance to digestion by hydrochloric acid. The right colon, with or without the terminal ileum, is used by most surgeons. The right colon is easily mobilized,[16] and its adequate blood supply is provided by the middle colic artery. The right colon with the preserved terminal ileum is used in an isoperistaltic manner in order to prevent reflux (Fig. 1;

FIG. 2. Barium swallow examination on a 70 year old man who had a squamous cell carcinoma of the esophagus just above the hiatus. The right colon was used in isoperistaltic fashion for reconstruction. The terminal ileum (arrows) was preserved and an end-to-end anastomosis with the remaining cervical esophagus was done. Note the ileocecal valve and the cecal pole.

2; and 3). When the terminal ileum is used to anastomose to the cervical esophagus, its small caliber has the advantage of being able easily to cross the thoracic inlet (Fig. 1; and 3). Having the same caliber as the remaining cervical esophagus, the ileum permits an easy end-to-end anastomosis.[16] The upper anastomosis can also be performed by using the right colon with the terminal ileum or cecal pole, or both, resected and discarded shortly before the anastomosis (Fig. 4, *A* and *B*).[5,6] The rationale for the latter procedure is that the cecum, the ileocecal valve and the terminal ileum are supplied by the ileocecal artery and its very small branches which have poor anastomoses with the major arcades. Frequently the cecal pole is too thick for a good cervical anastomosis.

When the right colon is used in the antiperistaltic fashion, the cecal pole and terminal ileum are preserved with the terminal ileum anastomosed to the stomach. A segment of ileum or jejunum can be interposed so that the cecum is anastomosed to the stomach, with the ileocecal valve functioning as a "cardia."[23]

The upper anastomosis is performed above the thoracic inlet in an end-to-end or end-to-side manner.[22] The colon used for reconstruction is placed on the anterior chest wall subcutaneously in patients with cancer, or in poor risk patients able to tolerate only a minimal surgical procedure.

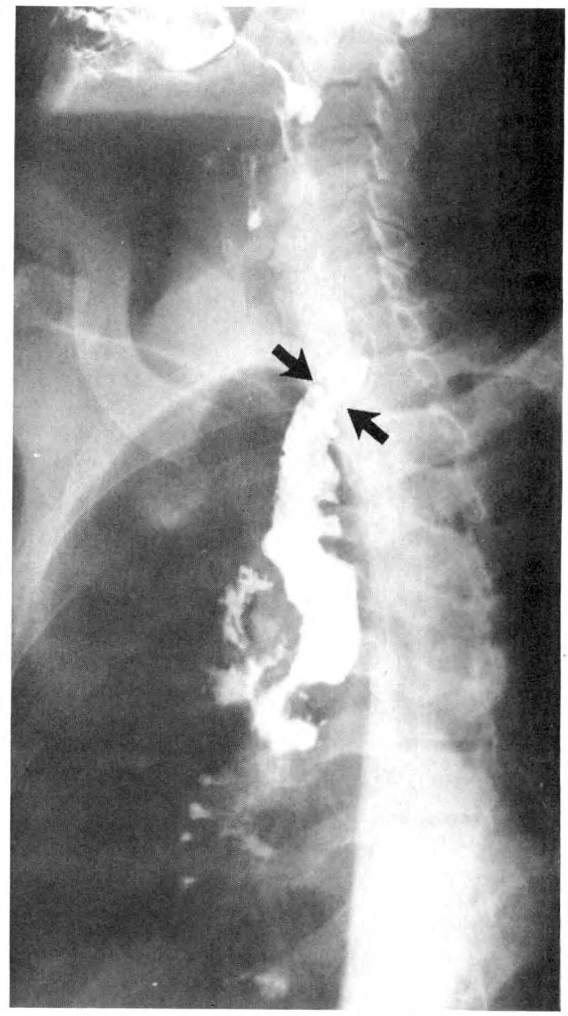

FIG. 3. Barium swallow examination of a 63 year old man who had a carcinoma of the mid esophagus resected and reconstructed by use of the right colon with cecal pole and terminal ileum preserved. Note the end-to-end upper anastomosis (arrows).

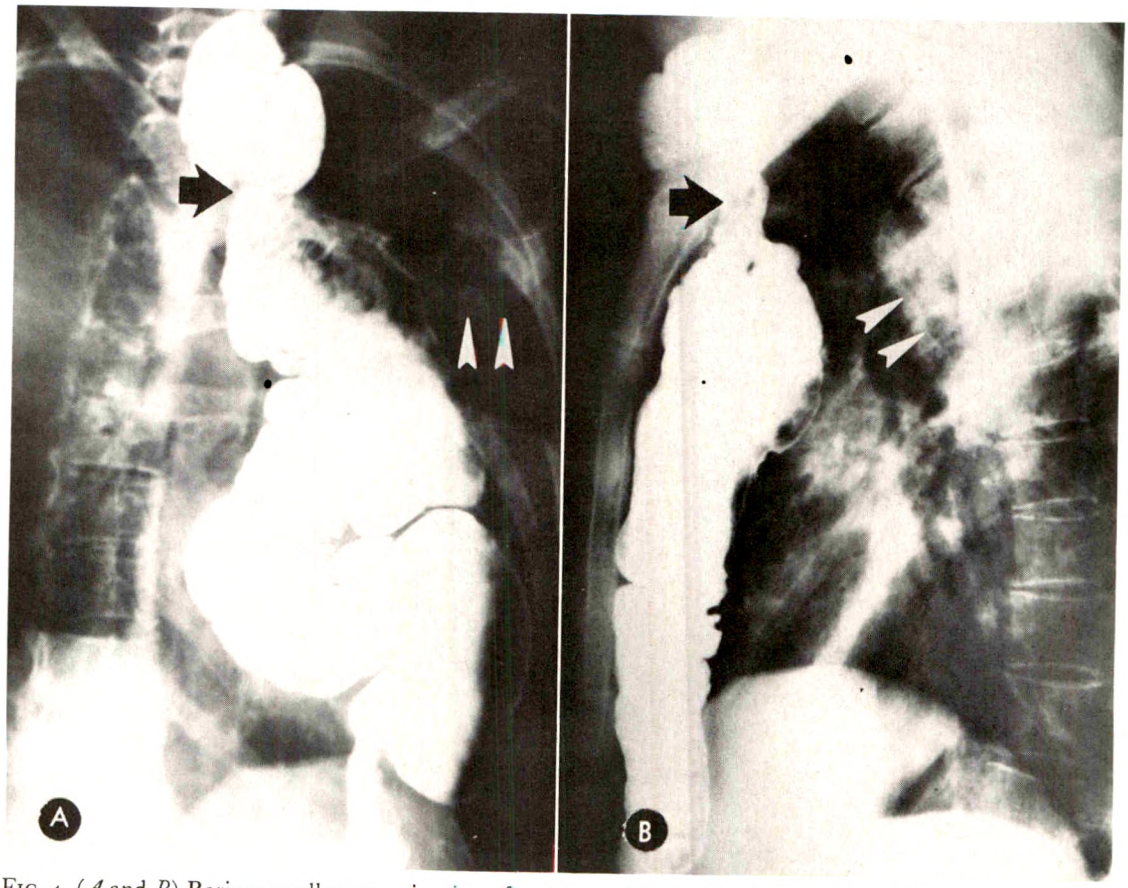

Fig. 4. (*A* and *B*) Barium swallow examination of a 49 year old woman who two years earlier had esophageal reconstruction for a stricture of the distal esophagus. The right colon was used in an isoperistaltic fashion; the terminal ileum was resected. An end-to-end cervical esophagus-cecal anastomosis was done. One month after surgery, she developed a stricture at the anastomosis (large arrows). The stricture was subject to repeated dilatations. These roentgenograms show barium retained due to the redundancy of the colon used for reconstruction. Note the left upper lobe tuberculous cavity (white arrowheads).

The next preferred route is the retrosternal. Finally, the colon can be used to reconstruct the lower esophagus in the esophageal bed. The colon should be as straight and as short as possible to avoid kinks and to prevent further obstruction.[6] There is no need to use a long and redundant colonic segment in children with the idea that it will be needed when the child grows. The colon used for reconstruction grows together with the patient.[4]

The lower anastomosis is usually placed high on the lesser curvature of the stomach (Fig. 1; and 5, *A* and *B*). A vagotomy and pyloroplasty are necessary to prevent gastric dilatation and retention of excessive amounts of secretion and food.

The use of the transverse colon (Fig. 6) is justified by the fact that the left colic artery is 2–4 mm wide from its origin to the marginal artery.[13] There is also a good venous network considered to be very important. The transverse colon can also be anastomosed proximally to a new esophagus and distally to the remaining segment of the lower esophagus thus preserving the cardia.[6]

Advocates of the use of the left colon,[6,24] claim it has sufficient length, good blood supply, and a smaller caliber, closer to the size of the esophagus (Fig. 7). The pedicle is usually based in the middle colic artery[12] (Fig. 10, *A* and *B*). When used in the antiperistaltic fashion, reflux may occur.

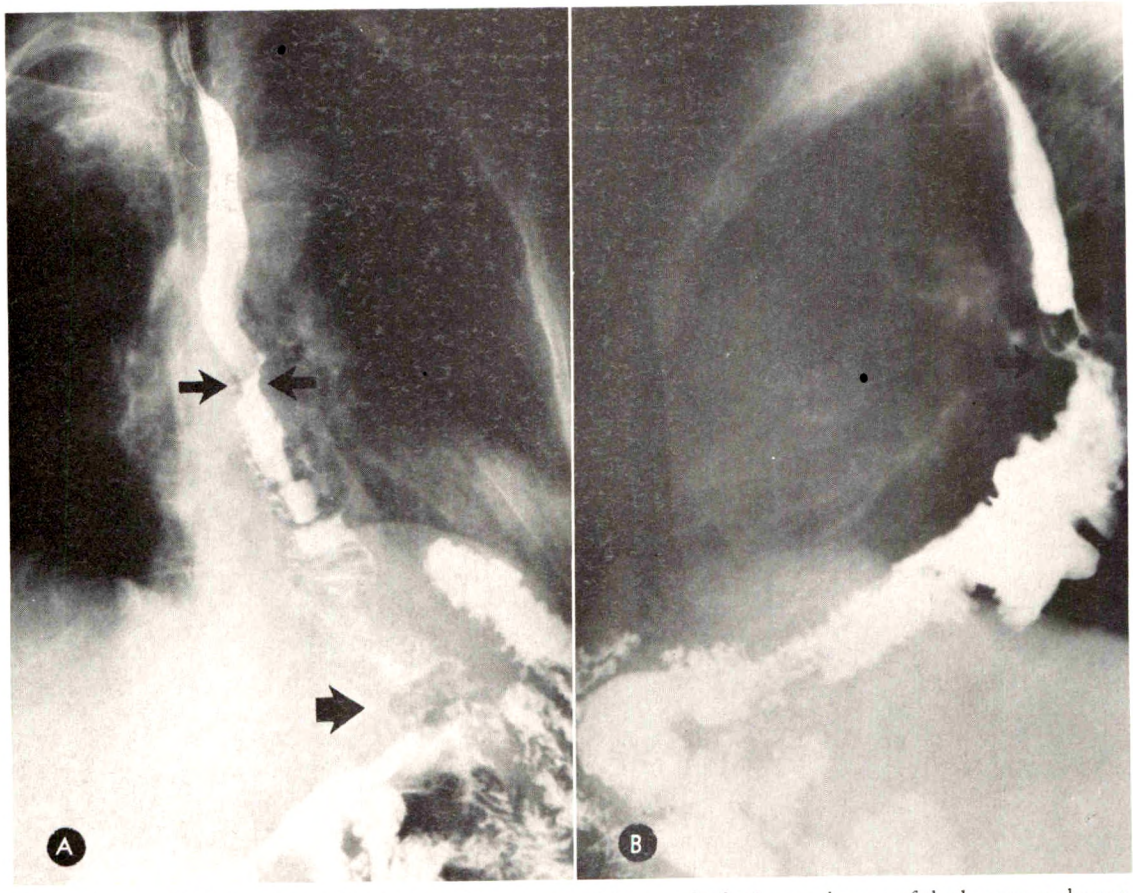

FIG. 5. (*A* and *B*) Reconstructed esophagus in a 74 year old man who had a carcinoma of the lower esophagus. The lower esophagus and the proximal stomach were resected and a segment of right colon was interposed between the remaining esophagus and the remaining distal stomach. The upper anastomosis is end-to-end (small arrows), and the lower anastomosis is end-to-side (large arrows). The terminal ileum and the cecal pole of the used right colon have been resected prior to the anastomosis. The patient died two years later from brain metastases.

Sigmoid colon has been used also as a free graft to reconstruct the pharynx.[15,16] The inferior mesenteric artery is anastomosed to the external carotid artery and the vein to the internal jugular vein.

Users of jejunum for reconstruction postulate that it is the only organ devoid of inherent disease.[2] Others, however, claim that the anatomical arrangement of the jejunal vessels fails to provide adequate vascularization for the length of the intestine needed in esophageal reconstruction.[10] Jejunum, first used 68 years ago for total esophageal reconstruction, is utilized today for replacement only of the distal esophagus.[2] A loop of jejunum may be interposed between the mid esophagus and the stomach after resection of the distal esophagus. After total gastrectomy and resection of the distal esophagus, the jejunum can be used as a link between the remaining mid esophagus and the duodenum or the jejunum (Fig. 8; and 9).[9] The stomach can also be bypassed with a direct anastomosis between the esophagus and the jejunum. The isolated jejunal loop is usually passed through the transverse mesocolon and it is anastomosed to the remaining esophagus. The continuity of the small bowel is maintained by an end-to-end or end-to-side jejunojejunostomy.

The stomach has been used for eso-

phageal reconstruction as long as 55 years ago. The stomach is readily available, has an excellent blood supply, and adequate mobilization permits it to be brought into the anterior mediastinum or even under the skin. The use, however, of a tube fashioned from the greater curvature of the stomach

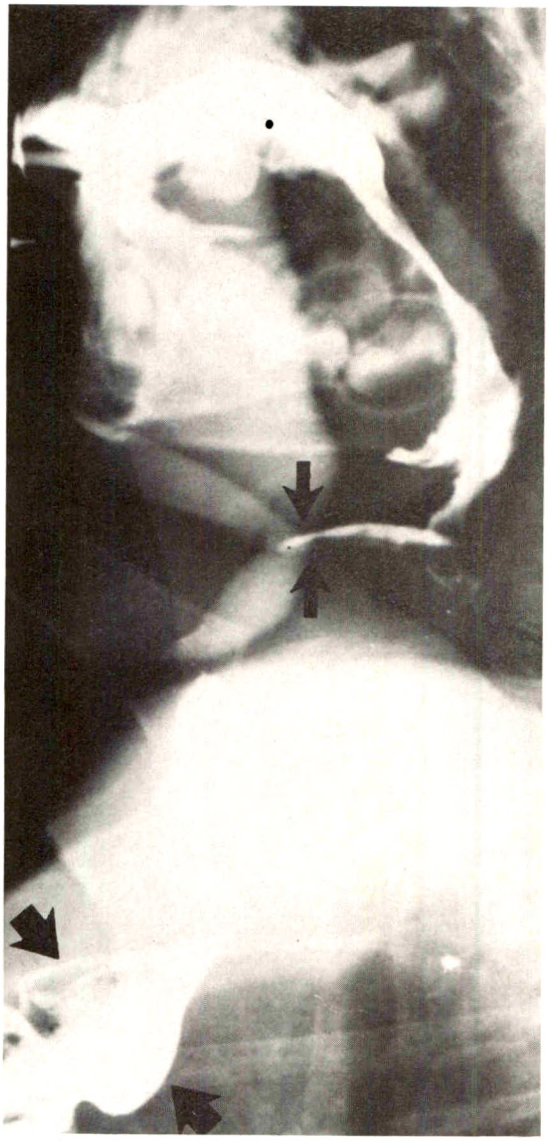

Fig. 6. Barium swallow examination of a 3 year old boy with esophageal atresia at birth. The mid transverse colon (large arrows) was used for reconstruction. Postoperatively there was a continuous fistula at the anastomosis (small arrows). The fistula closed spontaneously two months later. (Courtesy Stefan Arnon, M.D., University of California at Davis.)

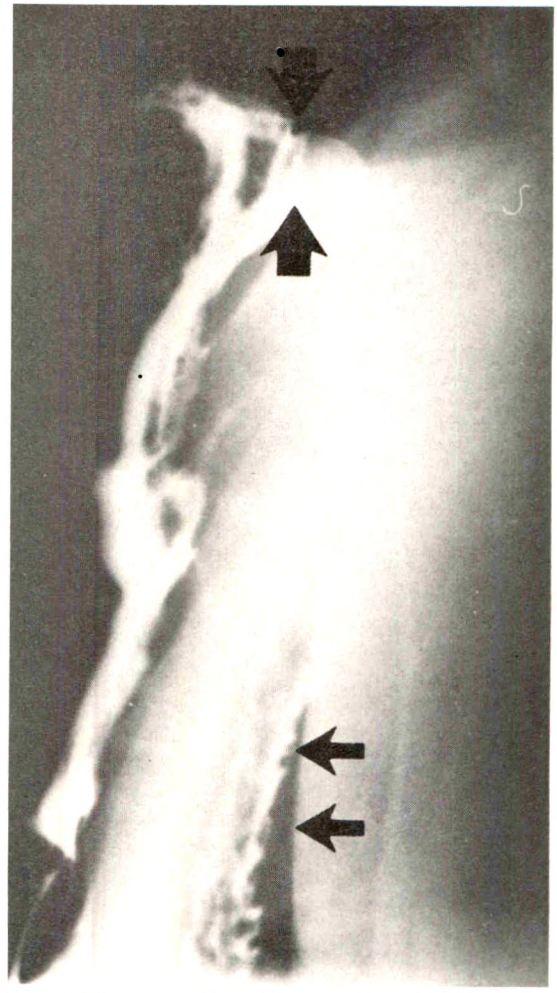

Fig. 7. Reconstruction of the esophagus in a 53 year old man with a carcinoma. The left colon was used for reconstruction in the isoperistaltic fashion. A continuous fistula developed at the anastomosis soon after surgery (large arrows). The anastomosis was revised, but three months later (present examination) the fistula was still present. Despite the fistula some barium will pass into the reconstructed esophagus (small arrows).

is more attractive. In 1966, Heimlich[8] devised the reverse gastric tube operation. A pedicle tube about 30 cm long and 2.5 cm in diameter is constructed from the greater curvature of the stomach by using a stapling instrument. The gastric tube is reversed and the antral end becomes the free end to be anastomosed to the cervical esophagus. The fundal end remains attached to the stomach. The gastric tube can be brought up subcutaneously, retro-

FIG. 8. Barium swallow examination performed on a 61 year old woman who two years earlier had a resection of the lower esophagus, total gastrectomy, and a reconstruction using a segment of jejunum interposed between the lower esophagus and the duodenum. Note the upper end-to-end anastomosis (small arrows) and the lower end-to-side anastomosis (large arrows).

sternally, or intrathoracically. Usually no esophagectomy is performed.

THE RESIDUAL ESOPHAGUS

Opinions vary on what to do with the residual esophagus. They range from leaving the diseased esophagus in order to avoid a thoracoplasty in sick patients, to combining esophagectomy and reconstruction, or radiation therapy, or both. Ong[15] removes the diseased esophagus *via* the abdomen through the hiatus. Griffiths and Shaw[5] give preoperative irradiation to lesions of the upper esophagus and larynx, followed by reconstructive surgery, or give a full course of postoperative irradiation therapy, or leave the esophagus in

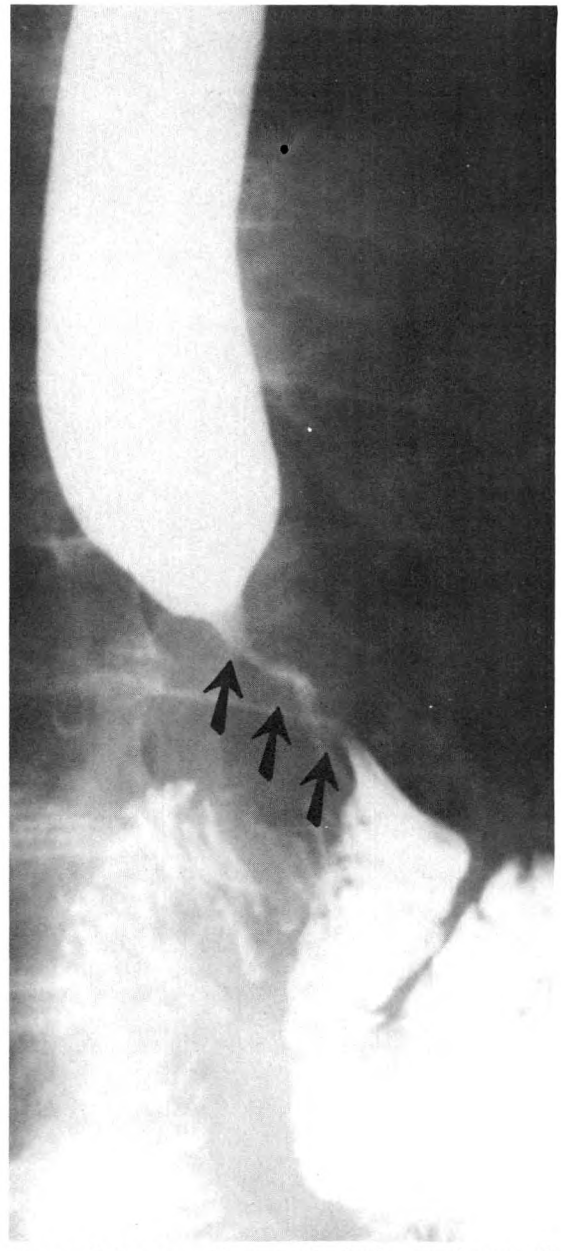

FIG. 9. A 65 year old man who had a carcinoma of the lower esophagus resected and reconstructed by an esophagojejunostomy. The barium swallow examination performed three months later shows a stricture of the lower esophagus (arrows).

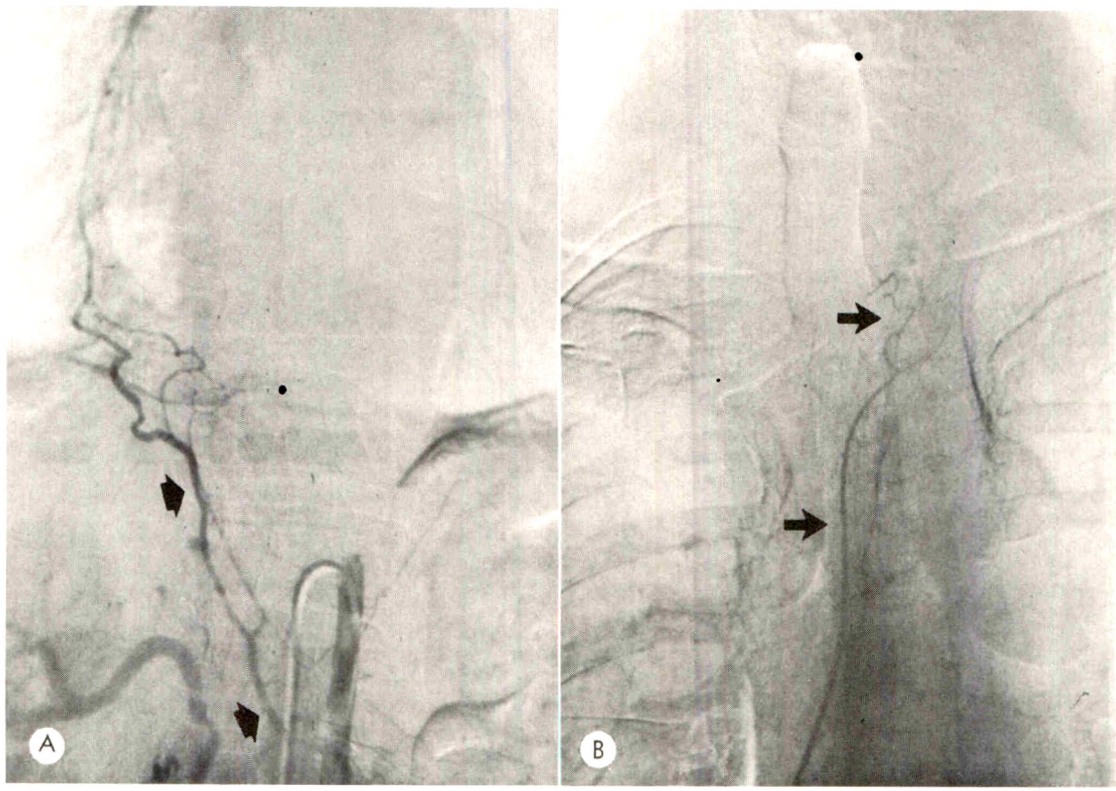

Fig. 10. (*A* and *B*) Subtraction roentgenograms from a selective superior mesenteric arteriogram in a 67 year old man who had esophageal reconstruction for a carcinosarcoma. The distal transverse and all of the descending colon were used with a pedicle based on the middle colic artery (large arrows in *A*). The marginal artery courses in the anterior mediastinum (small arrows in *B*). It terminates above the thoracic inlet, the site of the anastomosis. The blood supply in this case is normal. (Courtesy Harvey M. Goldstein, M.D. and Sidney Wallace, M.D., M. D. Anderson Hospital and Tumor Institute, Houston, Texas.)

place. Vincent and Webster[24] perform a descending colon esophageal reconstruction first, followed with radiation therapy, and then perform an esophagectomy. If the residual esophagus is left *in situ*, reflux esophagitis may develop and there is a greater chance that the patient may develop a carcinoma,[16] even if the reconstruction was performed for a nonmalignant condition.

COMPLICATIONS

In order to prevent unexpected complications, the viscus to be used for reconstruction must be examined with a barium study and preferably, with preoperative and postoperative angiography. It must be carefully inspected during surgery. Thorough cleansing of the viscera and prior antibiotic support also prevents some of the major complications.

Complications following esophageal reconstruction are listed in Table II. They occur mainly in the immediate postoperative period, with the leading complication being leakage at the cervical anastomosis. If the patient survives the procedure and does not succumb because of the underlying neoplastic process, late complications can develop. Reflux is the leading late complication.

The incidence of disruption of the cervical anastomosis with resulting subcutaneous fistula is between 35 and 41 percent.[3,10] The major factor causing high incidence of fistulae is the tenuous blood supply to the reconstructed esophagus (Fig. 10, *A* and *B*). A sudden drop in blood pressure in the

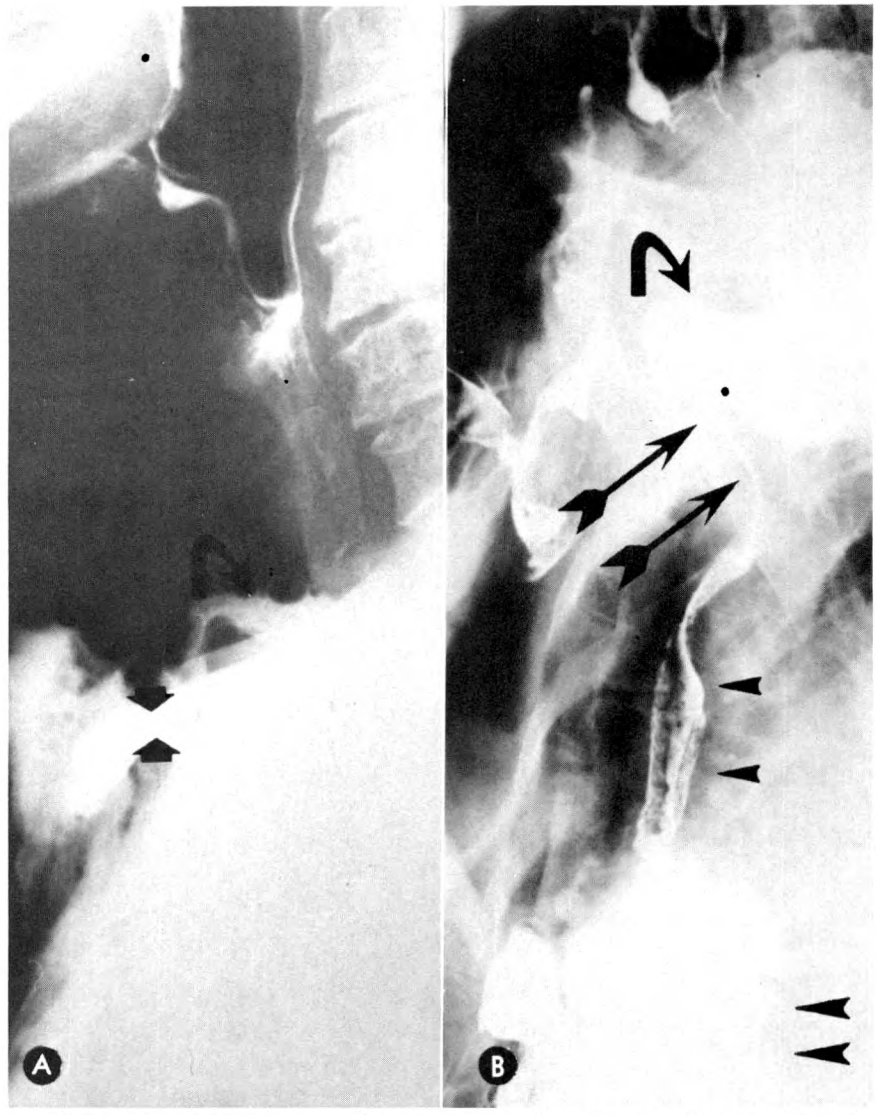

FIG. 11. One week postoperative barium swallow examination in a 65 year old man who had a resection of the esophagus for a squamous cell carcinoma of the mid segment. (*A*) A fistula (small arrows) is demonstrated at the anastomosis between the cervical esophagus and the terminal ileum which together with the right colon were used for reconstruction. (*B*) Two months later the fistula was still present but a stricture of the esophagoileal junction has developed (large arrows). Note the end-to-side anastomosis with the esophagus brought forward (curved arrows). In spite of the fistula and the stricture, barium will pass through the reconstructed esophagus: ileum (small arrowheads) and cecum (large arrowheads).

postoperative period for even a short period can cause temporary disruption of the blood supply around the upper anastomosis, causing thrombosis of vessels and a subsequent fistula.[13] Also, a narrow tunnel permits trauma to the vessels by the bony thorax which can lead to fistula formation. Early leaks do not close spontaneously, and usually a second operation is necessary.[15] The upper esophagus must be angulated anteriorly and around the trachea in order to meet the transplant, and this also accounts for fistula formation. Movement with every swallow interferes with healing and to minimize this, end-to-side anastomoses have been advocated.[16] It has

been postulated that fistulae develop because the esophagus lacks a protective serosal surface in the area of the anastomosis.[21]

If the fistula occurs after the immediate postoperative period, it becomes a terrible nuisance, but usually subsides with time.[20] If a fistula leaks into the mediastinum, death is unavoidable.

Stricture of the cervical anastomosis develops in about 32 percent of patients having a reconstructed esophagus.[3] It may follow healing of a fistula or may be unrelated (Fig. 11, *A* and *B*). It may occur early or late.

Another major complication is reflux of gastric contents into the interposed viscus (colon or jejunum) or into the remaining esophagus, particularly if no drainage procedure was done, and if the lower anastomosis is close to the pylorus (Fig. 12). The use of an isoperistaltic loop and draining procedures minimize reflux.[4] Some degree of regurgitation is common after esophageal reconstruction and patients must learn to swallow properly. When colon is used the

TABLE II

COMPLICATIONS FOLLOWING ESOPHAGEAL RECONSTRUCTION

I. Common
  (1) Early Postoperative
    Fistula at cervical anastomosis[2,3,10,15]
    Stricture at cervical anastomosis[2,9,13,15]
    Gangrene of transplant[2,13,15,16]
    Gastric stasis[14]
  (2) Later Complications
    Reflux[1,4,10]
    Stricture, (esophagojejunoduodenostomy)[2,3,9]
    Halitosis[16]
    Gastric stasis[14]
    Recurrent carcinoma (in the residual esophagus or stomach)[16]
II. Rare
  (1) Early Postoperative
    Fistula of lower anastomosis[10,13]
    Mediastinitis[3,16,20]
    Pneumothorax[10,16]
    Perforation (spontaneous)[12]
    Aspiration (recurrent nerve injury)[3,15]
  (2) Later Complications
    Radiation colitis[15]
    Peptic ulcer[3,12,14,21]
    Diverticulosis[3,17]
    Duodenal contents reflux[9]
    Migration of abdominal colonic segment into the chest[14]
    Duodenal obstruction[16]
    Dysphagia[17,24]
    Redundancy of colon with functional stenosis[18,24]
    Ulcer and bleeding of residual esophagus[4,16]

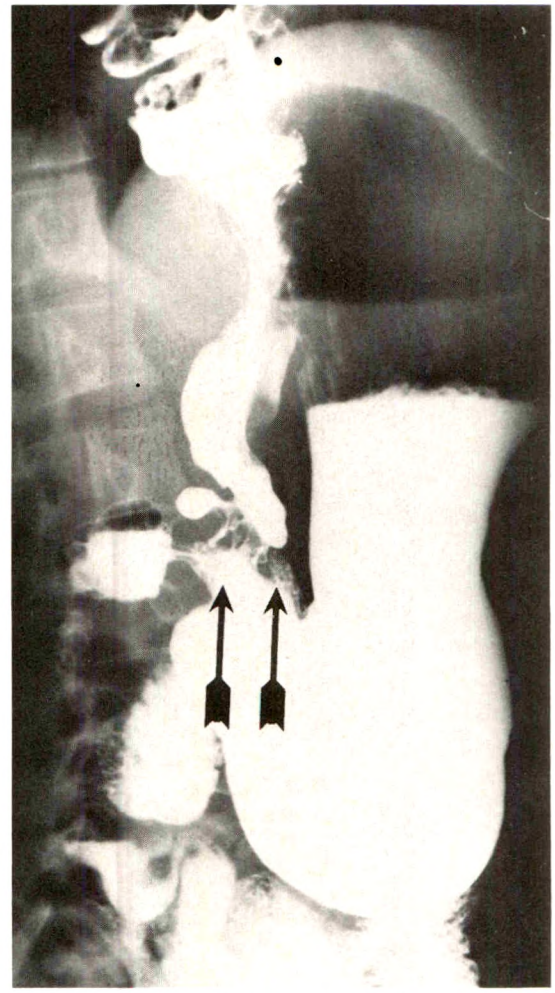

FIG. 12. Barium swallow examination in a 36 year old man who had a reconstruction of the esophagus for a lye stricture eight cm long, located at the level of the aortic arch. Surgery was performed four years earlier. The patient now complains of aphagia, reflux, and vomiting. Note the location of the distal anastomosis (arrows). This, together with the absence of pyloroplasty, has resulted in a marked distention of the stomach with retention of food and fluid. (Courtesy Stefan Arnon, University of California at Davis.)

FIG. 13. Barium swallow examination in a 63 year old woman who had the esophagus reconstructed for carcinoma four years earlier. The right colon and terminal ileum were used. At the time of examination, she complained of indigestion and occasional vomiting. The right colon has migrated from the abdomen into the chest and has become markedly redundant, and shows the retention of barium four hours after the barium swallow examination.

reconstructed esophagus contracts independently of both voluntary swallowing and cervical esophageal pressure.[1,23] Manometric and cinefluoroscopic studies of the reconstructed esophagus when the left colon is used show that one-third of patients have spontaneous peristalsis and all patients have peristalsis when the reconstructed segment is stimulated with 0.1 hydrochloric acid.[11]

Strictures secondary to reflux of intestinal content developed in patients with esophagojejunostomy.[2,9] Reflux of the duodenal content is the most likely cause for the esophagitis and secondary stricture

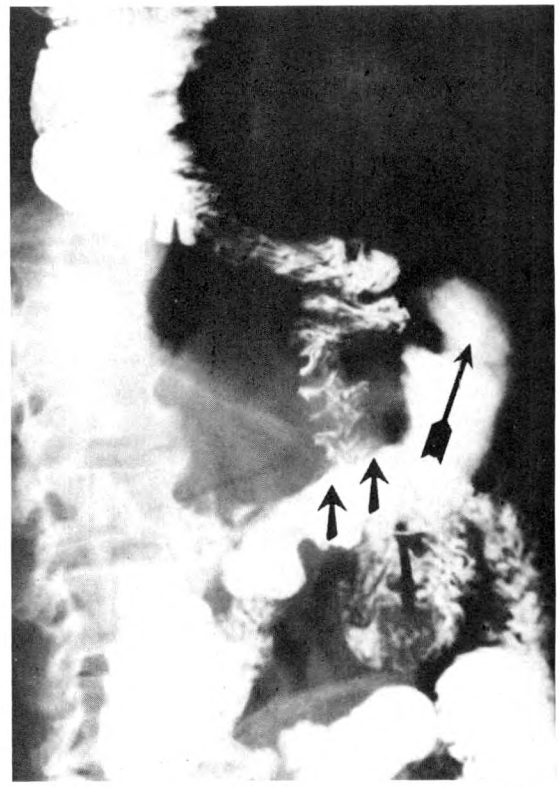

FIG. 14. Same patient as in Figure 2, two years later. The anastomosis between the stomach and the colonic segment is well seen (small arrows). The remaining two-thirds of the stomach (the fundus had been previously resected) show a diffuse infiltrative process, narrow lumen and an ulceration (large arrows). Exploratory laparotomy showed a poorly differentiated ulcerated squamous cell carcinoma invading the posterior wall of the stomach.

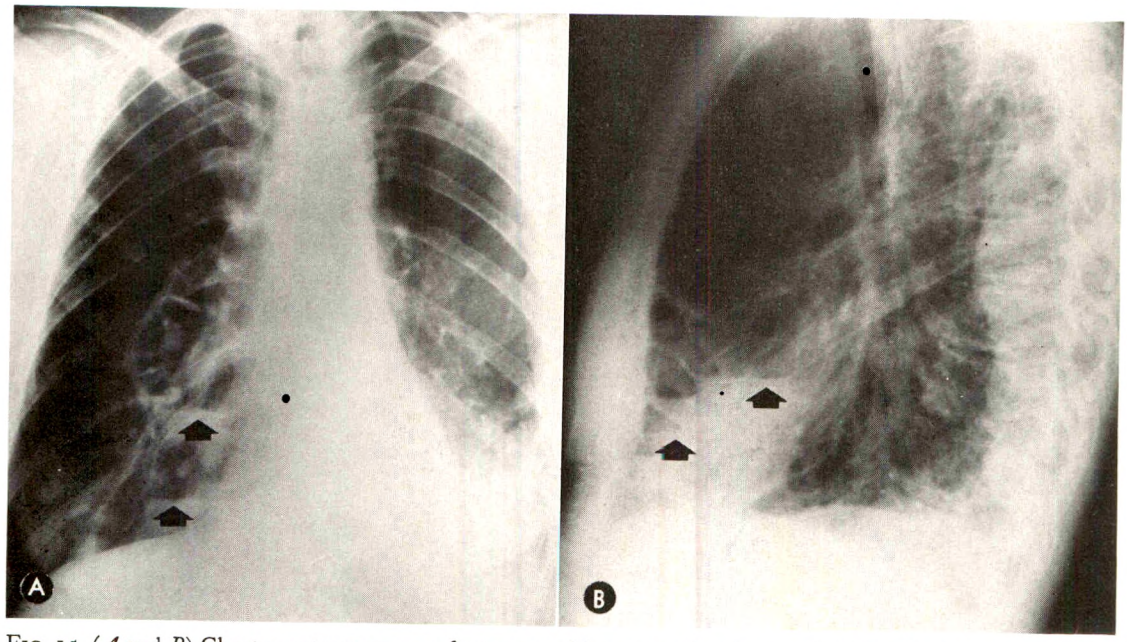

FIG. 15. (*A* and *B*) Chest roentgenograms of a 50 year old woman who six months before had a reconstruction of the esophagus using the right colon and terminal ileum. She had a squamous cell carcinoma of the mid esophagus which was previously resected. The air fluid levels (arrows) seen to the right of the heart and in anterior mediastinum are within the interposed colon used for reconstruction. The extensive left pleural changes are secondary to empyema resulting from metastatic involvement of the pleura. Note multiple pulmonary metastases.

(Fig. 9). Duodenal content can reflux into the esophagus if it is in direct anastomosis to the esophagus or if the interposed loop of jejunum is too short.

Development of peptic ulcer in the reconstructed esophagus is rare, but very fulminating.[12] Only eleven cases have been reported.[14]

Diverticulitis of the reconstructed esophagus is rare and is avoided by having the colon examined with barium prior to surgery for determining the presence of diverticula. Diverticular disease of the colon used for reconstruction can develop even if no diverticula were present prior to transplantation. This apparently results from the continuous ingestion of saliva and food which becomes a permanent stimulus for increase in intraluminal pressure and contraction of the colon.[17]

Migration of the abdominal part of the colon into the chest is an unpleasant complication.[14] The migrated redundant colon located in the chest can cause a physiologic obstruction (Fig. 4; and 13).

Recurrent carcinoma is usually a fatal complication. The carcinoma can recur in the remaining stomach (Fig. 14) or in the mediastinum (Fig. 15, *A* and *B*).

Duodenal obstruction occurs when the vascular pedicle to the right colon is brought anterior instead of posterior to the duodenum and stomach when mobilized and taken to the chest.[16]

RESULTS

In general, if no complication occurs following esophageal reconstruction, there is an improvement in the patients' condition. They can gain weight and can lead a normal life unless there is recurrence of the malignant process. In benign conditions, the reconstruction of the esophagus can be very gratifying and is consistent with a long life span. The operative mortality is in the range of 7.5 percent for benign conditions

and about 25 percent for malignant disease.[16] Strict selection of the cases can reduce operative mortality to 20 percent.[5]

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REFERENCES

1. BAERG, R. D., and BUTLER, M. L. Colon transplant for esophageal carcinoma: manometric study of case. *Am. J. Gastroenterol.*, 1972, *58*, 156–161.
2. DAVE, K. S., WOOLER, G. H., HOLDEN, M. P., BEKASSY, S. M., and IONESCU, M. I. Esophageal replacement with jejunum for nonmalignant lesions: 26 years experience. *Surgery*, 1972, *72*, 466–473.
3. EL-DOMEIRI, A., MARTINI, N., and BEATTIE, E. J., JR. Esophageal reconstruction by colon interposition. *A.M.A. Arch. Surg.*, 1970, *100*, 358–362.
4. GREGORIE, H. B., JR. Esophagocoloplasty. *Am. Surg.*, 1972, *175*, 740–749.
5. GRIFFITHS, J. D., and SHAW, H. J. Cancer of laryngopharynx and cervical esophagus: radical resection with repair by colon transplant. *A.M.A. Arch. Otolaryng.*, 1973, *97*, 340–346.
6. GROTTE, G., OLSEN, L., and REUTERSKIOLD, A. Reconstruction of esophagus with colonic transposition. *Acta paediat. scandinav.*, 1970, *60*, 59–65.
7. GROVES, L. K., and SILVER, G. M. Esophagoplasty with bowel segments and reversed gastric tubes. *Surgery*, 1973, *74*, 381–386.
8. HEIMLICH, H. J. Elective replacement of esophagus. *Brit. J. Surg.*, 1966, *53*, 913–916.
9. HELSINGER, N., JR. Esophagitis following total gastrectomy: clinical and experimental study. *Acta chir. scandinav.*, 1961, *273*, 1–19.
10. HUGUIER, M., GORDIN, F., MAILLARD, J. N., and LORTAT-JACOB, J. L. Results of 117 esophageal replacements. *Surg., Gynec. & Obst.*, 1970, *130*, 1054–1058.
11. JONES, E. L., SKINNER, D. B., DEMEESTER, T. R., ELKINS, R. C., and ZUIDEMA, G. D. Response of interposed human colonic segment to acid challenge. *Ann. Surg.*, 1973, *177*, 75–78.
12. KOVARIK, J. L., and GIPSON, B. F. Spontaneous perforation of intrathoracic colon following total esophagectomy. *Rocky Mountain M. J.*, 1973, *70*, 27–29.
13. LORTAT-JACOB, J. L., and GIULI, R. Esophageal replacement. *Progr. Surg.*, 1973, *12*, 77–95.
14. MALCOLM, J. A. Occurrence of peptic ulcer in colon used for esophageal replacement. *J. Thoracic & Cardiovasc. Surg.*, 1968, *55*, 763–772.
15. ONG, G. B. Resection and reconstruction of esophagus. *Current Problems in Surg.*, 1971, 3–56.
16. POSTLETHWAIT, R. W., SEALY, W. C., DILLON, M. L., and YOUNG, W. G. Colon interposition for esophageal substitution. *Ann. Thoracic Surg.*, 1971, *12*, 89–109.
17. RAIA, A., GAMA, A. H., PINOTTI, H. W., and RODRIGUES, J. J. G. Diverticular disease in transposed colon used for esophagoplasty: report of two cases. *Ann. Surg.*, 1973, *177*, 70–74.
18. SCHILLER, M., FRYE, T. R., and BOLES, E. T., JR. Evaluation of colonic replacement of esophagus in children. *J. Pediat. Surg.*, 1971, *6*, 753–760.
19. SINGH, A., and RICKHAM, P. P. Subtotal colonic replacement of esophagus in infancy. *Brit. J. Surg.*, 1971, *58*, 377–381.
20. STEPHENS, H. B. Colon bypass of esophagus. *Am. J. Surg.*, 1971, *122*, 217–222.
21. STOREY, C. F. Acquired Surgical Lesions of the Esophagus. Charles C Thomas, Publisher, Springfield, Ill., 1962, pp. 188–204.
22. TURNEY, S. Z. Lye stricture of esophagus in children: role of colon interposition. *Am. Surgeon*, 1974, *40*, 105–109.
23. TUSZEWSKI, F. K. Radiologic appearance of reconstructed esophagus. *Acta radiol.* (Diag.), 1972, *12*, 193–216.
24. VINCENT, R. G., and WEBSTER, J. H. Total esophagectomy and descending colon bypass for squamous cell carcinoma of esophagus. *Am. J. Surg.*, 1971, *121*, 527–530.

VOL. 125, NO. 4

THE ROENTGENOLOGIC INVESTIGATION OF CONGENITAL SUBGLOTTIC STENOSIS*

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ABSTRACT:

Congenital subglottic stenosis, which is life-threatening to the infant, can be diagnosed by the correlation of the clinical and the roentgenologic findings. The main clinical sign is the persistence of stridor from birth. The indicative roentgenologic finding is a symmetrical narrowing of the subglottic segment which is constant in form and length during the various phases of the respiratory cycle.

Two cases are presented, one confirmed by postmortem examination and the other by endoscopy. The etiology of this condition and the differential diagnosis are discussed.

UNTIL recently, congenital subglottic stenosis was considered a rare entity usually discovered only on necropsy by the pathologist.[5] With the diagnostic tools now available, this disease can be recognized during life and the affected infant can be helped.[1,2,4,6]

It is the purpose of this presentation to outline the roentgenologic approach and the roentgenologic signs which indicate congenital subglottic stenosis.

METHOD

The roentgenologic examination includes two phases: (1) A roentgenogram of the chest and neck on one film, using a high KV technique. Roentgenograms are obtained in two planes; (2) Fluoroscopy of the neck and mediastinum with spot films of the laryngeal and tracheal regions taken in the anteroposterior and lateral positions during inspiration and expiration.

RESULTS

On the chest roentgenograms and spot films, an air laryngogram is well visualized. This demonstrates a symmetrical narrowing of the subglottic region which is 2–3 mm in width and extends for a length of at least 5 mm. The passage from the constricted segment to the extrathoracic trachea is abrupt and sharply outlined. This air column is centrally placed and cone-shaped, with its apex extending to the vocal cord region, and does not change in appearance during respiration.

On fluoroscopy, during the inspiratory phase there is a marked distention of the hypopharynx by air with a widening of the mediastinal shadow. The reverse is observed during the expiratory phase of the respiratory cycle. As above, however, there is no change in the shape of the narrowed subglottic segment.

REPORT OF CASES

CASE 1. A three month old female infant from birth had presented noisy breathing which became worse during crying. She was hospitalized because of increasing dyspnea. On admission a marked inspiratory stridor with severe dyspnea and sternal retractions was noted. Physical examination showed the lungs to be equally aerated with an inspiratory and expiratory wheeze. The remainder of the examination was without pathological findings.

The roentgenologic investigation included chest-neck roentgenograms and fluoroscopy of the mediastinum with spot-films of the laryngeal region. During inspiration there was widening of the mediastinal shadow with distention of the hypopharynx. On the various spot-films obtained during the respiratory cycles and from different directions, an air laryngogram demonstrated a long, narrow segment in the subglottic region. In its proximal part, this air

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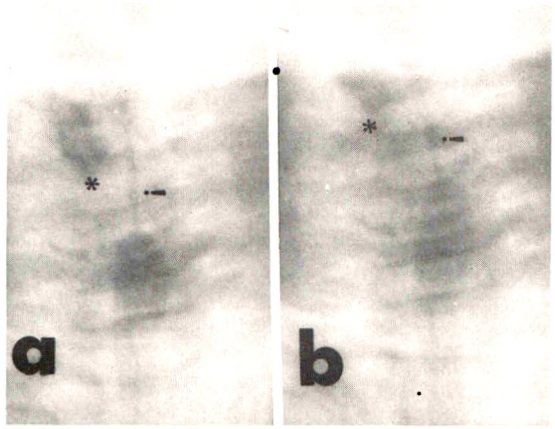

Fig. 1. Case 1. Spot films of the laryngeal region. (*A*) Inspiratory phase and (*B*) expiratory phase. Marked constant, symmetrical narrowing of the subglottic region (black arrow) with an abrupt transition from the narrow segment to the extrathoracic trachea and a distended hypopharynx (above black star).

column had a symmetrical cone-shaped appearance. Morgagni's ventricle was preserved (Fig. 1). On the basis of the roentgenologic findings as well as the history of stridor, a diagnosis of subglottic stenosis was made.

Tracheal intubation performed because of increasing dyspnea brought immediate relief but during the night the tracheal catheter came out and respiratory arrest occurred. The infant was resuscitated, but expired soon after.

Postmortem examination revealed a normal epiglottis, aryepiglottic folds and cricoid cartilage. Beneath the true vocal cords, in the conus elasticus region, a symmetrical thickening of the wall covered with mucosa was seen narrowing the airway for a length of 8 mm, leaving a lumen of 1–2 mm in width. This tube-like formation, which was of a grayish-red color, was similar to a ring obstructing the air passage. The transition from the narrow segment to the extrathoracic tracheal lumen was abrupt (Fig. 2). Microscopic examination of the thick wall segment in the conus elasticus region revealed an unusual mass of striated muscle separated by connective tissue beneath the mucosa encroaching on the lumen.

Comment. The subglottic stenosis of congenital origin was due to excessive muscle tissue.

Case II. A twelve month old male infant from birth was known to have a "barking" voice which was diagnosed as chronic laryngitis. He was hospitalized because of increasing dyspnea during an upper respiratory infection.

The physical examination revealed a cyanotic infant with marked inspiratory stridor. Fine rales were heard over both lung fields. The remainder of the examination was negative.

An extensive roentgenologic investigation disclosed a constant, symmetrically narrowed segment in the subglottic region (Fig. 3). On fluoroscopy, during inspiration the hypopharynx became distended, with a widening of the mediastinal shadow. Pneumonic infiltrates were spread throughout both lung fields. The roentgenologic findings were evaluated as being compatible with a subglottic narrowing complicated by a patchy pneumonia.

The pneumonia responded to antibiotic

Fig. 2. Case 1. Sagittal section through the larynx and the extrathoracic trachea. Morgagni's ventricle appears normal. Severe concentric stenosis of the subglottic air-way with a sharp border between the narrow lumen and the extrathoracic trachea.

treatment but the inspiratory stridor persisted. Repeat roentgenograms showed that the narrowing of the subglottic segment was still present. Endoscopy revealed that the entrance to the subglottic region was concentrically narrowed with smooth borders. The patient was subsequently hospitalized several times because of increasing stridor. During one of these hospitalizations elsewhere, a tracheostomy was performed which improved the dyspnea. The patient succumbed at home during an episode of pneumonia, and necropsy was not performed because of the family's objection.

Comment. Subglottic stenosis of congenital origin with a superimposed pneumonia.

DISCUSSION

Roentgenologic investigation of the stridulous infant is today a *sine qua non.* Whereas instrumental examination, such as indirect or direct laryngoscopy, is always potentially dangerous in a patient having stridor of unknown origin,[3] roentgenologic investigation is a harmless procedure which can provide a great deal of information and can be of considerable assistance in establishing the cause of stridor.

There are two major origins of congenital subglottic stenosis: anomalies of the cricoid cartilage; or soft tissue anomalies in the walls of the conus elasticus in the subglottic region.[5] Case I, in which the stenosis was due to an excess of striated muscle tissue beneath the mucosa, belongs to the latter category. The roentgenologic and endoscopic findings in Case II also favor this etiology.

This condition can be diagnosed very early in life on the basis of the roentgenologic findings and should be suspected in any infant presenting a persistent inspiratory stridor, as is demonstrated in the above two cases. It should be noted that the fluoroscopic finding of a widening of the mediastinal shadow during the respiratory phase is not pathognomonic of this entity, but indicates an incomplete respiratory obstruction above the tracheal bifurcation.[3]

Table I

THE DIFFERENTIAL DIAGNOSIS OF CONGENITAL SUBGLOTTIC STENOSIS DURING INFANCY

1. Hemangioma or lymphangioma
2. Laryngeal or subglottic web
3. Paralysis of the vocal cord
4. Acute laryngotracheobronchitis

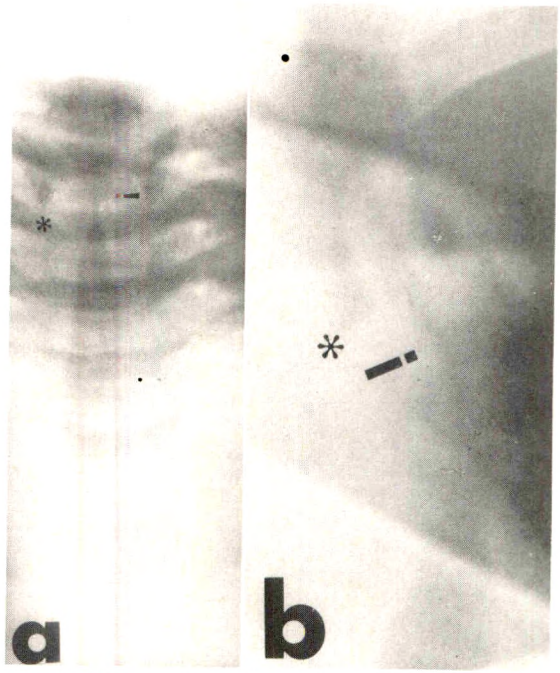

Fig. 3. Case II. Spot films of the laryngeal region. (*A*) Anteroposterior position and (*B*) lateral position. Narrow subglottic segment beneath the normal-appearing Morgagni's ventricle (black arrow in *A*); also well visualized in the lateral position (black arrow in *B*). The hypopharynx is distended with air (above black star).

From the roentgenologic point of view, the differential diagnosis includes several other possibilities but these are easily ruled out (Table I). In cases of subglottic hemangioma or lymphangioma, the stenosis within the conus elasticus region is not symmetrical. With a laryngeal or subglottic web there is no narrow segment seen. Vocal cord paralysis, especially if unilateral, gives a picture of asymmetry of the vocal cords and of Morgagni's ventricles. In acute laryngotracheobronchitis (croup) the stridor is present only during the acute

phase of the disease and the roentgenologic signs disappear when the stridor subsides.

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REFERENCES

1. CUNDY, R. L., and BERGETROM, L. B. Congenital subglottic stenosis. *J. Pediat.*, 1973, *82*, 282–284.
2. EVANS, W. A., JR. Congenital obstructions of respiratory tract: tracheal malformations. AM. J. ROENTGENOL. & RAD. THERAPY, 1949, *62*, 167–176.
3. GRÜNEBAUM, M. Respiratory stridor: challenge for pediatric radiologist. *Clin. Radiol.*, 1973, *24*, 485–490.
4. LALLEMAND, D., SAUVEGRAIN, J., and MARESCHAL, J. L. Laryngo-tracheal lesions in infants and children. *Ann. radiol. diag.*, 1973, *16*, 293–304.
5. PUVEENDRAM, A. Congenital subglottic atresia: case report. *J. Laryng. & Otol.*, 1972, *86*, 847–852.
6. TAYBI, H. Congenital malformations of larynx, trachea, bronchi and lungs. *Prog. Pediat. Radiol.*, 1967, *1*, 231–255.

VOL. 125, NO. 4

THE VARIOUS FORMS OF PULMONARY VARICES*

REPORT OF THREE NEW CASES AND REVIEW OF THE LITERATURE

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ABSTRACT:

True pulmonary varices are congenital local dilatations of a pulmonary vein or veins, with normal or collateral transpulmonary drainage into the left atrium.

Pulmonary varices do not cause pulmonary venous hypertension, but existing varices can further become dilated by pulmonary venous hypertension due to mitral valve disease.

Embryologically, pulmonary varices may represent residual primitive splanchnic venous components incorporated into the pulmonary venous system, or atresia of an individual pulmonary vein which occurred at a time that enabled adoption of unobstructed transpulmonary collateral drainage into the left atrium.

True pulmonary varices do not produce symptoms, do not change in diameter over the years, and, usually, do not require treatment.

PULMONARY varices are rare. Since the first report of a patient by Hedinger in 1907[18] only 42 new[2–4,8–10,12,15–17,19–21,22,23,26,29–35,38–40] and three doubtful[2,14] cases were published. Review of the literature revealed differences in the pattern of the pulmonary varices in several cases, possibly reflecting basic differences in the formation of varices in the embryo.

The purpose of this paper is to present three cases and discuss the differences between the two main categories of pulmonary varices.

REPORT OF CASES

CASE 1. H.W., a 50 year old female, was referred for a routine examination because of pulmonary tuberculosis in a member of her family. A chest roentgenogram (Fig. 1) revealed dilated vascular structures in the right lower lung field medially. Review of previous roentgenograms of the chest showed that these vascular shadows have existed, unchanged, for the past 24 years. The patient was asymptomatic and there was no evidence of congenital or acquired heart disease. Pulmonary angiography did not reveal evidence of pulmonary arterial or venous hypertension. The arterial phase of the pulmonary angiogram (Fig. 2*A*) revealed a normal atrial pattern. During the venous phase (Fig. 2*B*), dilated, tortuous pulmonary veins were seen in the right lower lobe; these veins drained into the right superior pulmonary vein, which entered the left atrium normally. There was no visualization of a right inferior pulmonary vein.

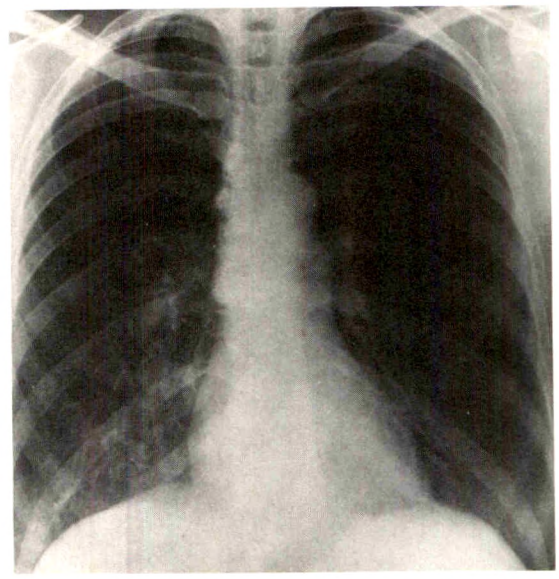

FIG. 1. Case 1. Chest roentgenogram. Prominent vascular markings in the right lower medial lung field and right hilum.

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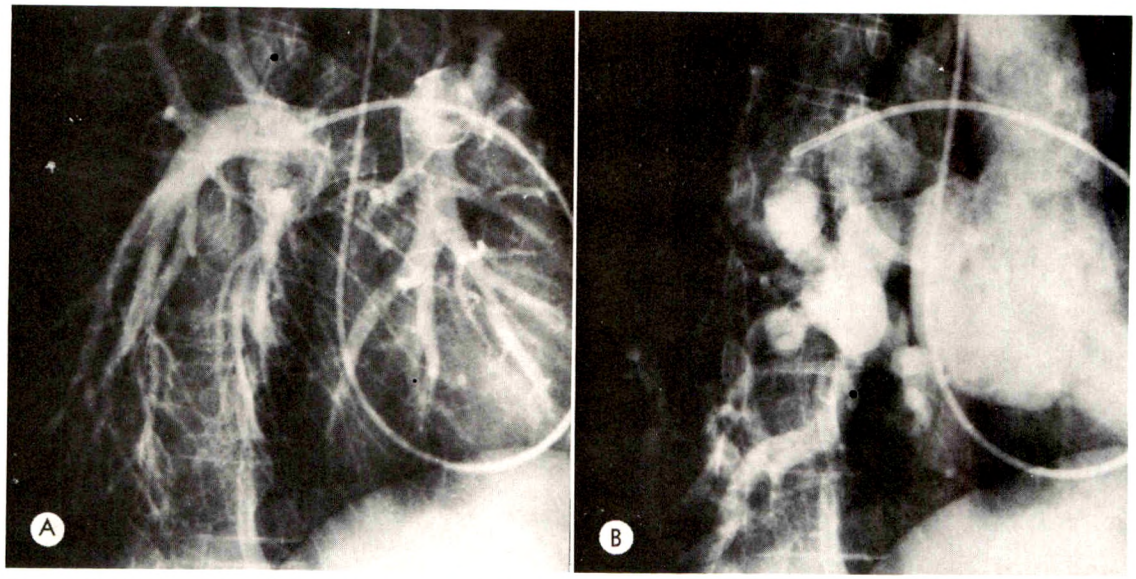

Fig. 2. Case 1. Selective right pulmonary angiogram, right anterior oblique position. (*A*) The arterial phase shows normal pulmonary arteries. (*B*) During the venous phase, large, lower pulmonary varices drain into the right superior pulmonary vein. There is no right inferior pulmonary vein.

Case II. R.W., a 53 year old female, was admitted for clinical suspicion of an intracranial space-occupying lesion. Cerebral angiography revealed a berry aneurysm of the left internal carotid artery. Roentgenograms of the chest (Fig. 3, *A* and *B*) revealed a rounded, slightly lobulated, soft tissue density in the right hilum. Pulmonary angiography showed no evidence of pulmonary arterial or venous hypertension. During the arterial phase (Fig. 4, *A* and *B*), the pulmonary arteries were found to be normal. During the venous phase (Fig. 5, *A* and *B*), all the veins from the right lung were found to be draining into a single, varicose, common pulmonary vein which drained normally into the left atrium. The left side was normal.

Case III. E.V., a 62 year old female, was admitted for evaluation of hyperparathyroidism. Angiography showed an adenoma of the left inferior parathyroid gland. Chest roentgenograms (Fig. 6, *A* and *B*) revealed a round,

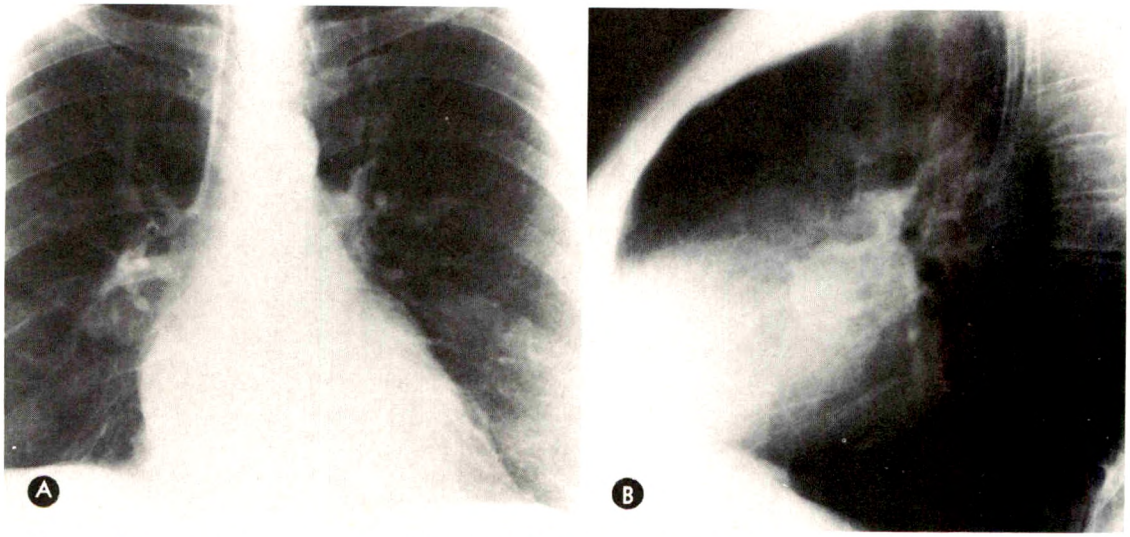

Fig. 3. Case II. Roentgenograms of the chest, in the (*A*) posteroanterior and (*B*) lateral projections. Note round, lobulated soft tissue mass in the right hilum.

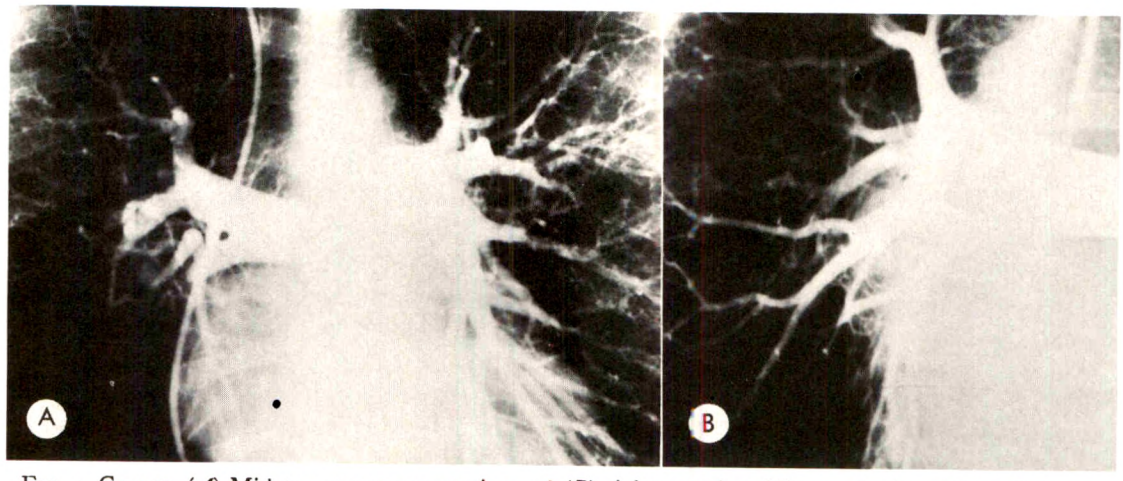

Fig. 4. Case II. (*A*) Midstream anteroposterior and (*B*) right anterior oblique selective right pulmonary angiogram, arterial phase. Note normal pulmonary arteries.

slightly lobulated, soft tissue density in the right hilum. Pulmonary angiography showed no evidence of arterial or venous pulmonary hypertension. During the arterial phase (Fig. 7, *A* and *B*) normal arterial architecture was found. During the venous phase (Fig. 8, *A* and *B*), the veins of the right lower and middle lobes were found to be draining into a varicose, right inferior pulmonary vein which drained normally into the left atrium. The right superior pulmonary vein and both left pulmonary veins were normal.

DISCUSSION

DEFINITION

The traditional definition of a pulmonary varix is a localized enlargement of a segment of a pulmonary vein which enters the left atrium normally,[2] or prominent, but normally draining pulmonary veins;[30] in the absence of pulmonary venous hypertension due to heart disease, the lesions are characterized by stability over many years of observation.[3,9,12,14,16,17,19,29,32]

According to this definition, at least seven of the 47 cases hitherto published,[2,4,20,23,26,31,39] as well as our own Cases I and II, are not true varices, as they all show anomalies in the involved pulmonary veins, even though the final drainage is into the left atrium.

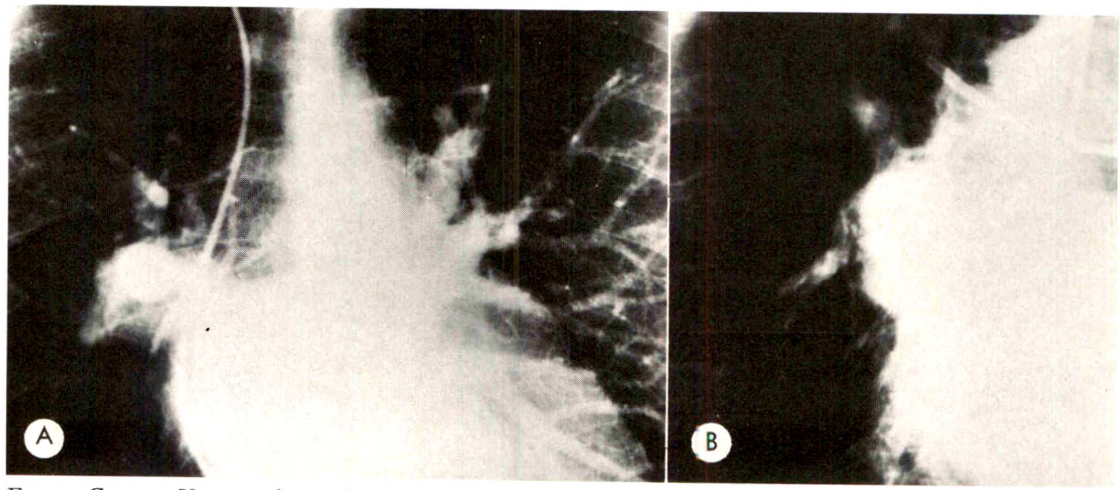

Fig. 5. Case II. Venous phase of pulmonary angiogram in the (*A*) anteroposterior and (*B*) right anterior oblique projections. Large hilar varices drain the entire right lung into the left atrium superiorly.

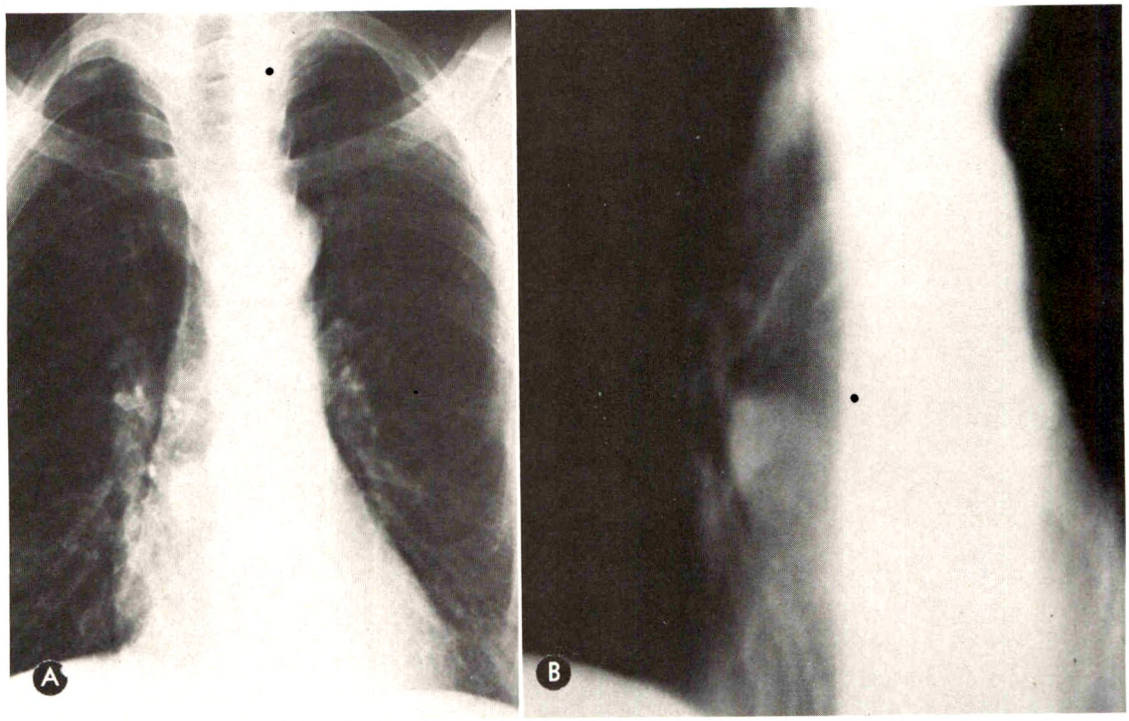

FIG. 6. Case III. (*A*) Roentgenogram and (*B*) tomogram of the chest. A round mass in the right hilum. The tomogram suggests the vascular nature of the lesion.

ETIOLOGY

It is generally agreed that pulmonary varices are local congenital anomalies without significant relation to other existing congenital cardiac, pulmonary, or other disorders, and without known etiology.[2,3,30,32,34] The development of the pulmonary venous system and its anomalies has been extensively studied.[1,6,24,25,28,42] Most probably the defect occurs at the time of transition of the lung from splanchnic venous drainage to pulmonary venous drainage proper, during horizons XIII–XVI[28] of the embryonic development. The existence of local varicose segments was attributed to weakness of the venous walls in these segments.[3] Incorporation of primitive splanchnic veins in the newly developing pulmonary venous system was suggested as a possible cause of such weakness;[2] there is, however, no evidence to suggest that the walls of the splanchnic veins are basically weaker than those of the pulmonary veins. It is hard to explain the dilatation on the basis of differences in wall structure. In most cases, in which the microscopic structure of the varicosities was studied, all layers, including the elastic fibers, were found to be normal.[9,18,22,27,29] Only two cases were reported as showing an abnormality: in one,[31] the wall was thinner than normal and the muscular layer was replaced by fibrous tissue; in the other,[32] the wall was very thick and fibrous.

DISTRIBUTION

Sex and Age. Among cases reported in the literature (28 females, 19 males, and three whose sex was not reported),[2,4,15] the varices were first discovered at age 32.2 years mean in the males and 37.5 years mean in the females.

In a small series such as this one, differences in age and sex distribution cannot be regarded as significant and are most probably coincidental, particularly as the varix is usually an incidental finding during investigations for unrelated conditions.

Localization. In most reported cases, varices were found in the right lower and

middle lobe (20 patients) or the left upper lobe and lingula (14 patients). Ten additional patients were reported as having hilar varices, without specification as to the vein involved, or varicosities over an entire lung. The reason for the relative sparing of the right upper lobe and left lower lobe is not understood and will not be speculated upon in this paper. In none of the reported cases did the patient have bilateral pulmonary varices.

THE VARIOUS PATTERNS OF PULMONARY VARICES AND DILATED PULMONARY VEINS

A. True Pulmonary Varices. These are the majority of the reported cases (about 30 patients), and our Case III belongs in this group. The only anomaly is a locally dilated pulmonary vein or veins in part of a lung or its hilum. Two pulmonary veins exist on each side and all enter the left atrium normally. We shall assume that this anomaly may be the result of incorporation of primitive splanchnic venous segments in the newly developing pulmonary venous system, as proposed by Bartram and Strickland.[2]

B. Dilated Transpulmonary Venous Collaterals. Seven patients[2,4,23,31,39] and our Cases I and II are recorded as having a single right or left upper or lower pulmonary vein, with dilated, tortuous (varicose) veins draining the rest of the lung into them. The single pulmonary vein enters the left atrium normally. This type of anomaly can theoretically be created by secondary atresia of the pulmonary vein after the separation of the splanchnic venous drainage, but before lobar separation of the pulmonary veins was completed, thereby allowing transpulmonary collaterals to develop. Had the atresia occurred earlier, pulmonary-splanchnic drainage would have been permanently established; a later occurrence would have resulted in individual pulmonary vein stenosis with all its symptomatology and sequelae.[5,24,25,36,37]

C. Dilated Pulmonaty Veins with Abnormal Insertion into the Left Atrium. Two cases are reported, one with a left superior pulmonary vein[26] and one with a left common pulmonary vein,[20] inserting too far to the right into the left atrium. Although the veins were dilated they cannot be regarded as true varices.

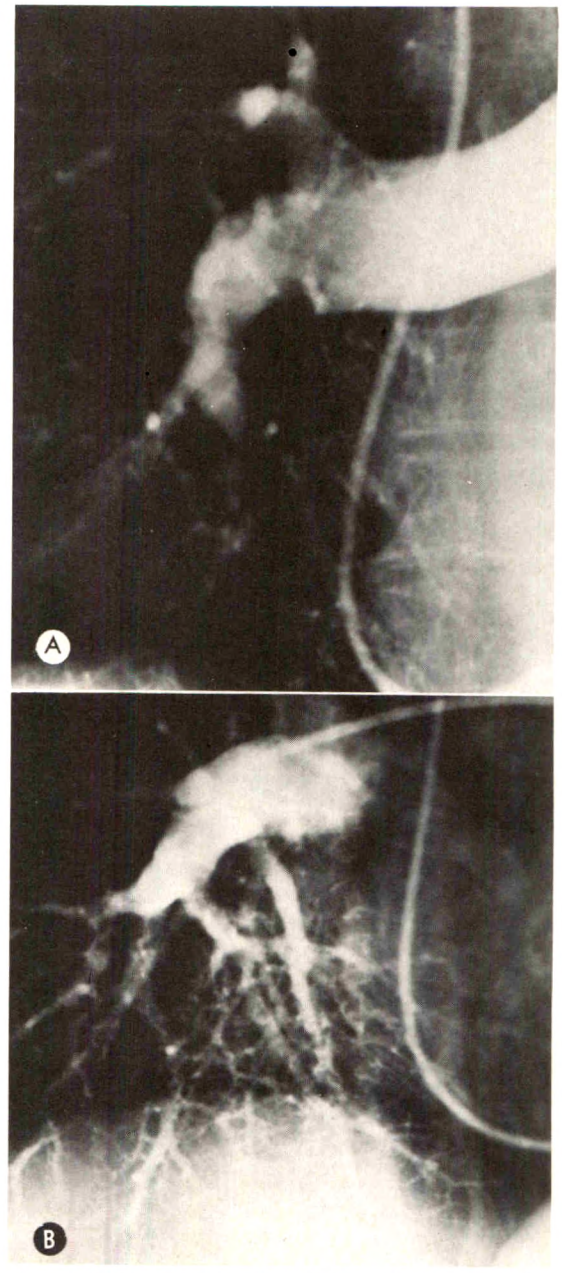

Fig. 7. Case III. Selective right pulmonary angiogram, arterial phase. (*A*) Anteroposterior and (*B*) right anterior oblique projections. Normal pulmonary arteries.

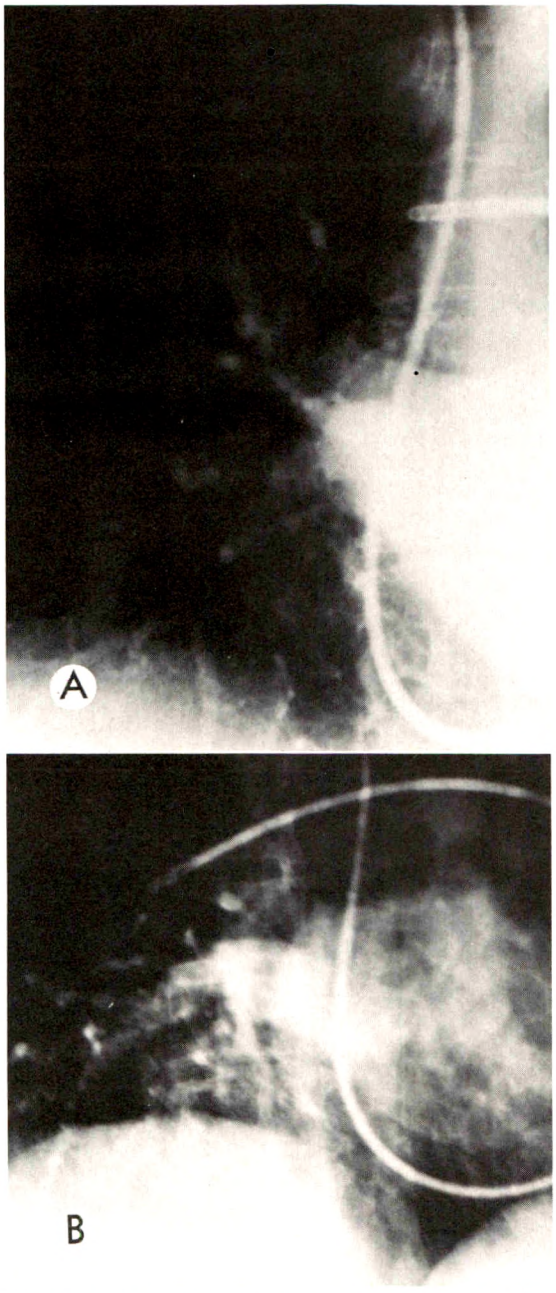

FIG. 8. Case III. Venous phase of pulmonary angiogram, in the (*A*) anteroposterior and (*B*) right anterior oblique projections. The right inferior pulmonary vein is varicose, but drains normally into the left atrium. The right superior pulmonary vein is normal.

D. "Pulmonary Varices" in Mitral Stenosis and Insufficiency. Viamonte and LePage[41] described a patient (their Case II) with mitral stenosis and bilateral pulmonary varices. Whether the dilated veins were true varices or not is difficult to say on the basis of the published material; and in the presence of mitral stenosis, their bilaterality and hilar location suggest that they were probably chronically distended main pulmonary veins rather than true varices. Bryk[7] describes seven patients with mitral insufficiency, and with markedly dilated right pulmonary veins. The localization of the dilatation in the right veins in Bryk's cases can be explained by the direction of the regurgitation flow, which would be preferentially into the right pulmonary veins. These, also, are not true pulmonary varices.

SYMPTOMS

When pulmonary varices are the patient's only abnormality, they do not produce symptoms.[2,3,11,13] In most reported cases the discovery of the varix was on routine chest roentgenograms or during investigation of cardiac or other diseases.

Hemoptysis was reported in five patients.[2,18,27,31] In one[31] it originated from bronchiectasis; in another[27] bleeding may have been due to pulmonary tuberculosis.

Three patients[8,18,29] presented with cough, which cannot be shown as being directly caused by the presence of pulmonary varices.

One patient[23] presented with a middle lobe syndrome, directly related to pressure on the middle lobe bronchus by the varices.

Two patients presented with dysphagia,[20,26] due to pressure on the esophagus by a large left pulmonary vein which inserted abnormally to the right of the left atrium. Both these patients are not regarded as having true varices.

Most symptomatic patients presented with congestive heart failure of various causes, but unrelated to the varices.

THE RELATIONSHIP BETWEEN ACCOMPANYING ANOMALIES OR DISEASES AND PULMONARY VARICES

Most commonly, accompanying anomalies were congenital or acquired heart disease: five patients had mitral insufficiency;[8,19,20,21,33] three had mitral steno-

sis;[8,9,16] and one each had coarctation of the aorta,[35] aortic stenosis and pseudocoarctation,[2] common origin of both great vessels from the right ventricle,[38] ventricular septal defect and patent ductus arteriosus,[40] malrotation of the cardiac chambers,[2] and absence of the right hemipericardium.[30]

Other associations included pulmonary tuberculosis,[27] systemic hypertension,[22] left upper lobe pulmonary emphysema,[2] intracranial aneurysm (our Case II), and hyperparathyroidism (our Case III).

Three patients had pulmonary arterial anomalies, including absence of the right main pulmonary artery,[12] hypoplasia of the descending branch of the right pulmonary artery,[31] and a trifurcation, rather than bifurcation, of the right pulmonary artery.[3] Two patients[2] had partial anomalous venous drainage contralateral to the side of the pulmonary varices.

Varices are not created by heart disease, and their existence in cardiac patients is purely coincidental.[2,3] In patients with pulmonary venous hypertension due to mitral stenosis or insufficiency, pre-existing varices may become wider.[11] In such cases, progression of the congestive heart failure may make the presence of an enlarging varix rather threatening,[23] by creating conditions for rupture.

In one patient with mitral insufficiency[19] the varices became progressively distended over a seven year period and disappeared following surgery. In another,[20] dysphagia due to a distended left pulmonary vein with an aberrant insertion into the left atrium and pressure on the esophagus disappeared after treatment of the patient's congestive heart failure.

COMPLICATIONS AND DEATH

Two patients[29,32] are reported to have suffered a cerebral embolus, the source of which was possibly in the varices.

Hedinger[18] felt that his patient's duodenal ulcer was caused by an embolus from a pulmonary varix.

Three deaths are reported as having resulted from a ruptured pulmonary varix.[22,27,32] In one,[22] the varix grew rapidly with aggravation of existing right heart failure. In another,[27] rupture may have been caused by erosion of the varix by pulmonary tuberculosis in the adjacent lung tissue. The complete clinical picture of the third patient[32] is not available.

A fourth patient[29] is reported to have died of a cerebral embolus, but the description of the clinical picture in this patient is strongly suggestive of congestive heart failure.

ROENTGENOGRAPHIC DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

The smooth, rounded or lobulated soft tissue density of the varix on the plain chest roentgenogram and tomogram should be differentiated from other pulmonary or hilar mass lesions, including those that are neoplastic, granulomatous, lymphatic, or vascular in origin.[2,3,8,10,13–16,19,20,21,38] At least one patient[14] was given treatment for two years for pulmonary tuberculosis, until the true nature of the mass on the chest roentgenogram was revealed. A most important differential diagnosis is that of pulmonary arteriovenous fistula,[2,3,13,30] which may have an identical roentgenographic appearance. Partial anomalous pulmonary venous return should also be included in the differential diagnosis.[39]

The Müller maneuver enlarges the size of pulmonary varices, and the Valsalva maneuver decreases it in many, but not all patients.[2,16,19] The only safe criterion for the diagnosis of pulmonary varices is the opacification of the varices during the venous phase of pulmonary angiography. Demonstration of normal pulmonary arterial and venous pressures is necessary to exclude heart disease as an etiologic factor.

An important additional criterion is the non-growth of pulmonary varices over long periods of follow-up. Eleven patients[3,9,12,14,16,17,19,29,32] and our Case I had follow-up chest roentgenograms over periods ranging from two to 24 years, without any appreciable change in the size of the pulmonary varices.

TREATMENT

Treatment is not necessary in asymptomatic patients who are free of heart disease.[14,21,30,31] Patients with right heart failure, who show enlargement of the varices, can be expected to reduce the size of the lesions with treatment of their congestive failure.

Only rarely, as in the patient in whom hemoptysis exists and is directly related to pulmonary varices, should surgery be considered.[2]

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REFERENCES

1. AUER, J. Development of human pulmonary vein and its major variations. *Anat. Rec.*, 1948, *101*, 581–594.
2. BARTRAM, D., and STRICKLAND, B. Pulmonary varices. *Brit. J. Radiol.*, 1971, *44*, 927–935.
3. BEAMISH, W. E. Pulmonary venous varicosity. *J. Canad. A. Radiologists*, 1972, *23*, 136–139.
4. BENFIELD, J. R., GOTS, R. E., and MILLS, D. Anomalous single left pulmonary vein mimicking parenchymal nodule. *Chest*, 1971, *59*, 101–103.
5. BERNSTEIN, J., NOLKE, A. C., and REED, J. O. Extrapulmonic stenosis of pulmonary veins. *Circulation*, 1959, *19*, 891–897.
6. BHARATI, S., and LEV. M. Congenital anomalies of pulmonary veins. *Cardiovasc. Clin.*, 1973, *5*, 23–41.
7. BRYK, D. Dilated right pulmonary veins in mitral insufficiency. *Chest*, 1970, *58*, 24–27.
8. BRYK, D., and LEVIN, E. J. Pulmonary varicosity. *Radiology*, 1965, *85*, 834–837.
9. Case records of The Massachusetts General Hospital. *New England J. Med.*, 1951, *245*, 575–581.
10. DAVIA, J. E., GOLDEN, M. S., PRICE, H. L., HASTINGS, J. E., and CHEITLIN, M. D. Pulmonary varix: diagnostic pitfall. *Circulation*, 1974, *49*, 1011–1012.
11. Editorial. Pulmonary varicosity. *Radiology*, 1966, *86*, 116.
12. ELLIS, J. G., McCORMICK, W., and CLEVELAND, J. E. Absence of artery to right lung and varices of veins from left lung. *Angiology*, 1974, *25*, 428–431.
13. FRASER, R. G., and PARÉ, J. A. P. Varicosities of pulmonary veins. In: Diagnosis of Disease of the Chest. Volume I. W. B. Saunders Company, Philadelphia, 1970, pp. 576–577.
14. GIMES, B., and HORVATH, F. Über die Varikosität der Pulmonalvene. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1958, *89*, 545–548.
15. GOOD, C. A. Certain vascular abnormalities of lungs. Hickey Lecture, 1961. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1961, *85*, 1009–1024.
16. GOTTESMAN, L., and WEINSTEIN, A. Varicosities of pulmonary veins: case report and survey of literature. *Dis. Chest*, 1959, *35*, 322–327.
17. HAGEN, H., and HEINZ, K. Varixknoten im Lingulaast der Vena pulmonalis. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1960, *93*, 151–159.
18. HEDINGER, E. Demonstration eines Lungenvarix. *Verhandl. deutsch. Gesellsch. Pathol.*, 1907, *11*, 303–308.
19. HIPONA, F. A., and JAMSHIDI, A. Observations on natural history of varicosity of pulmonary veins. *Circulation*, 1967, *35*, 471–475.
20. JACOBS, R. C., GERSHENGORN, K., and CHAIT, A. Dysphagia associated with distended pulmonary vein. *Brit. J. Radiol.*, 1972, *45*, 225–228.
21. KELVIN, F. M., BOONE, J. A., and PERETZ, D. Pulmonary varix. *J. Canad. A. Radiologists*, 1972, *23*, 227–229.
22. KLINCK, G. H., and HUNT, H. D. Pulmonary varix with spontaneous rupture of death. *Arch. Path.*, 1933, *15*, 227–237.
23. KOZUKA, T., and TADAHARU, N. Pulmonary vein anomaly: unusual connection and tortuosity of right lower lobe vein. *Brit. J. Radiol.*, 1968, *41*, 232–234.
24. LUCAS, R. V. Congenital causes of pulmonary venous obstruction. *Cardiovasc. Clin.*, 1972, *4*, 1–51.
25. NAKIB, A., MOLLER, J. H., KANJUH, V. I., and EDWARDS, J. E. Anomalies of pulmonary veins. *Am. J. Cardiol.*, 1967, *20*, 77–90.
26. NASH, B., FREIDENBERG, R. M., and GOETZ, R. Aberrant insertion of pulmonary vein into left atrium simulating intrinsic lesion of esophagus. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1961, *86*, 888–891.
27. NAUWERK, D. Lungenvarix und Haemoptose München. *Med. Wochenschr.*, 1923, *70*, 1084–1086.
28. NEILL, C. A. Development of pulmonary veins. *Pediatrics*, 1956, *18*, 880–887.
29. NEIMAN, B. H. Varix of pulmonary vein. AM. J. ROENTGENOL. & RAD. THERAPY, 1934, *32*, 608–612.
30. NELSON, W. P., HALL, R. J., and GARCIA, E. Varicosities of pulmonary veins simulating arteriovenous fistula. *J.A.M.A.*, 1966, *195*, 13–17.

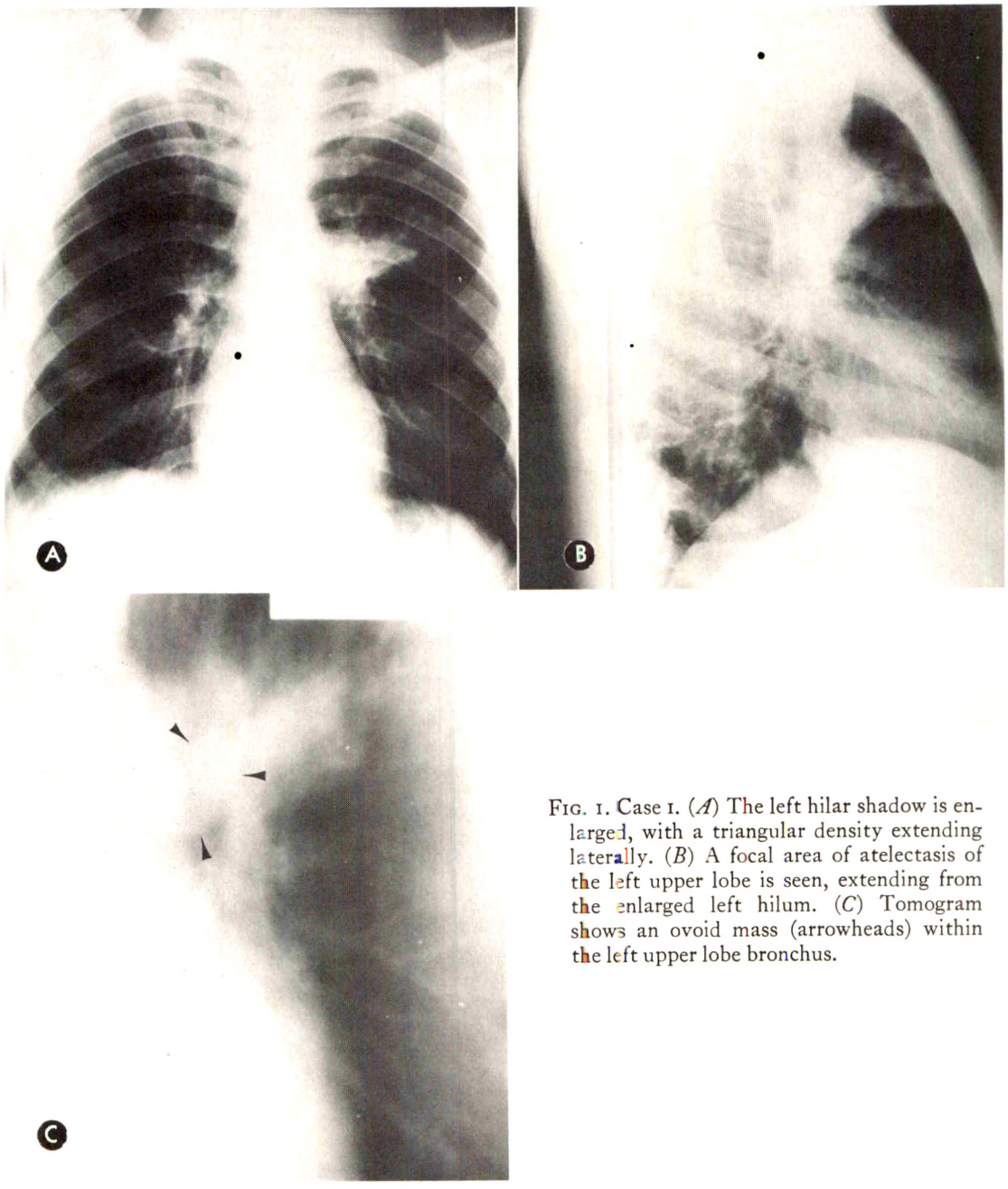

Fig. 1. Case 1. (*A*) The left hilar shadow is enlarged, with a triangular density extending laterally. (*B*) A focal area of atelectasis of the left upper lobe is seen, extending from the enlarged left hilum. (*C*) Tomogram shows an ovoid mass (arrowheads) within the left upper lobe bronchus.

contact with bundles of nerve tissue and appeared to derive from normal nerve tissue.

Case II. A 21 year old totally asymptomatic, healthy woman was found to have a solitary nodule in the right upper lobe on routine chest roentgenogram (Fig. 2*A*). Physical examination and all laboratory studies were negative, including skin test for tuberculosis and histoplasmosis. A tomogram (Fig. 2*B*) confirmed the presence of a somewhat lobulated, sharply demarcated mass about 1.5 cm in diameter (Fig. 3*A*). This appeared to be entirely outside the bronchial tree. Bronchoscopy and bronchography were normal. Pulmonary arteriography was performed and showed no arteriovenous malformation.

A right upper lobectomy was performed and 2 cm mass was found attached to the trifurcation of the upper lobe bronchus, grossly entirely

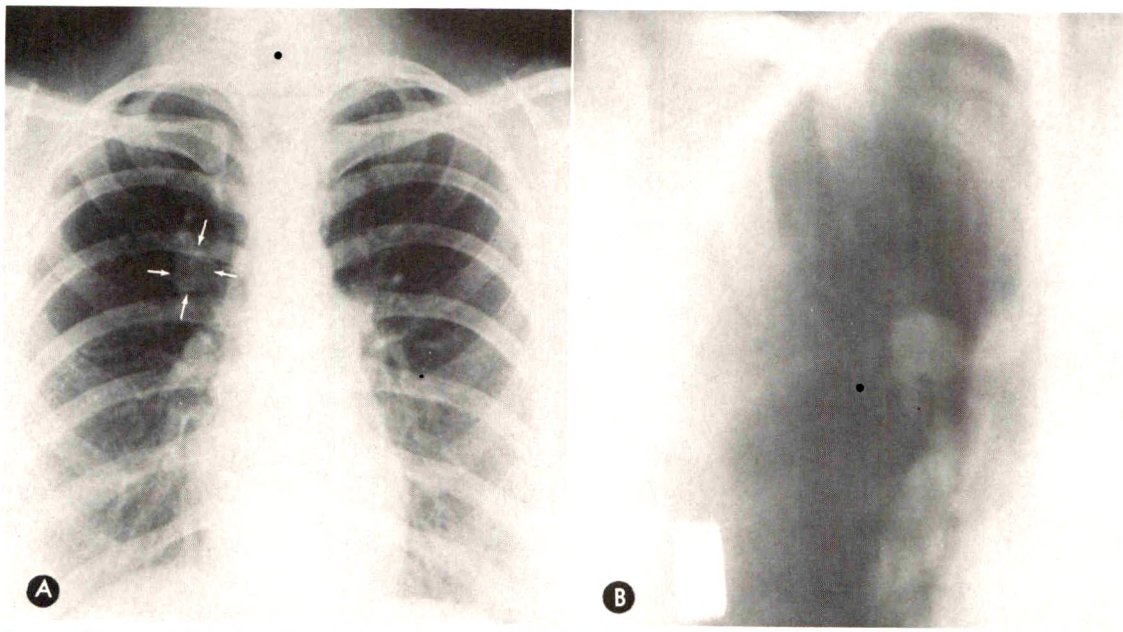

FIG. 2. Case II. (*A*) A sharply marginated rounded density (arrows) is seen above the right hilum. (*B*) Tomogram reveals a circumscribed nodule completely outside the bronchial lumen.

outside the bronchial lumen. The bronchial mucosa in the three surrounding bronchi was elevated. Histologically, the tumor was circumscribed but not encapsulated and compressed in the lung parenchyma. It infiltrated the bronchial submucosa and focally broke the mucosa. It merged focally with the bronchial smooth muscle. The tumor cells were polygonal to spindle with abundant granular brown cytoplasm and small round to vesicular nuclei. Lymph node biopsies showed reactive hypoplasia but no tumor cells. The diagnosis was granluar cell myoblastoma.

CASE III. E.F. is a 47 year old Black male with a history of night sweats, productive cough and recurrent hemoptysis of six months' duration. He was found to have an area of chronic pneumonitis in the right upper lobe (Fig. 4).

Bronchoscopy was performed and although no abnormalities were apparent in the right upper lobe bronchus, an endobronchial lesion approximately one cm in diameter was seen on the lateral wall of the left lower lobe mainstem bronchus. It was not occluding the bronchial lumen. A biopsy specimen was taken and the lesion proved to be a granular cell myoblastoma of the bronchus. No roentgen abnormalities were seen in the left hilum or lung, even after careful retrospective examination of the chest roentgenograms.

DISCUSSION

The uncommon granular cell myoblastoma is generally a small lesion, rarely greater than two to three cm, most frequently encountered in the skin, mucous membrane, and tongue. Unusual sites include the larynx, breast, thyroid, gallbladder, esophagus, and stomach.[8,9,13] The lesions of the tracheobronchial tree are exceedingly rare, representing less than two percent of all myoblastomas.[7,13] Of the approximately 50 reported cases of tracheobronchial myoblastoma,[1–7,9,11,12,14,15] the vast majority were in a major bronchus. A large number of these cases were incidental findings in unrelated surgery or autopsy.

The only reports in the radiologic literature are of a tracheal lesion[11] and two gastric tumors.[8]

Myoblastomas in the lung (bronchi) can occur at any age, although they are most often found in the 30–50 year old patients. There appears to be a predilection for Black females. Multiple bronchial lesions were found in about 14 percent of the

cases,[14] GCM is usually considered a benign tumor, although six percent of all myoblastomas may be malignant and show distant metastases.[13] However, distant metastases from endobronchial GCM have not been reported. The pulmonary myoblastoma is almost always within the lumen of a major bronchus, but about 20 percent can also extend submucosally along the bronchial tree and occasionally may penetrate into the surrounding lung parenchyma. Extension into the adjacent lymph nodes can also occur,[4] but this local spread does not necessarily indicate malignancy.

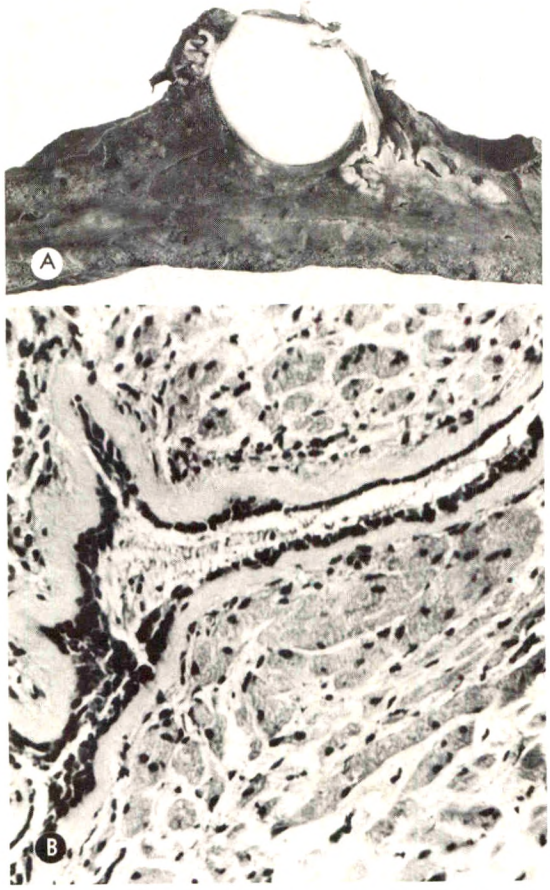

Fig. 3. (*A*) Gross section through tumor of Case II. The sharply circumscribed, 1.5 cm, whitish tumor is entirely within the lung parenchyma. (*B*) Histologic section of tumor of Case I. Collections of polygonal and ovoid cells, with reticulated cytoplasm and small dense nuclei, are scattered throughout the stroma. Their appearance is characteristic.

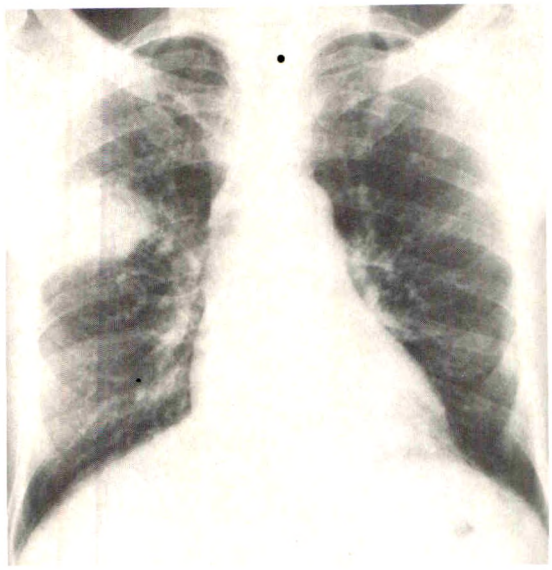

Fig. 4. Case III. The inflammatory infiltrate in the right upper lobe was entirely unrelated to the incidental finding of a granular cell myoblastoma in the left lower lobe bronchus. This endobronchial tumor has produced no apparent changes in the left hilum or the left lower lobe.

Grossly, the tumor is a well circumscribed glistening, whitish mass, rarely larger than 3 cm (Fig. 3*A*). The histologic appearance is characteristic and identical in all the lesions. Large round to polygonal cells with a granular acidophilic cytoplasm and small round or ovoid nuclei are packed in a scanty stroma (Fig. 3*B*). Rarely, the cells are pleomorphic and the tumor is considered malignant.[1,10] Whether the tumor cell is of muscle or neurogenic origin is uncertain.[3] The histologic picture in Case I suggests origin from nerve sheath cells.

Clinical symptoms from a GCM are usually completely absent when the tumor is small and unobstructive (Case III), or if the bronchial lesion is entirely extraluminal (Case II). The larger endobronchial lesions, which may cause atelectasis or recurring pneumonias can give rise to hemoptysis, cough, and expectoration (Case I).

The roentgenographic features of GCM of the lung can be surmised from case reports which contain roentgenographic illus-

trations.[1,3,4,9,12,15] Small asymptomatic nonobstructive lesions will cause no apparent findings on the chest roentgenograms (Fig. 4). Many of the smaller lesions might possibly be seen on tomography or bronchography, but in the absence of symptoms, these studies are not done and the tumors are unexpected findings either at autopsy or during unrelated bronchoscopy, as in our third patient. Larger lesions in a major bronchus may cause hilar enlargement often associated with focal pneumonitis or atelectasis (Fig. 1, *A* and *B*). Part of the hilar enlargement may be due to reactive hilar lymphadenopathy which frequently occurs. In one reported case[10] the hilar enlargement was entirely due to reactive adenopathy from a GCM of the trachea. Tomography will usually demonstrate the mass within the major bronchus (Fig. 1*C*). When the tumor growth extends entirely outside the bronchial lumen, as in Case II, there are usually no symptoms and the lesion will appear as a circumscribed parenchymal density (Fig. 2*A*) outside the bronchus on tomography (Fig. 2*B*). Bronchoscopy may then be entirely negative.

The roentgenographic findings of an endobronchial myoblastoma will most often simulate a bronchial adenoma or possibly a polyp. When the tumor is entirely outside the bronchial lumen, a host of other diagnostic possibilities must be considered including hamartoma, granuloma, arteriovenous malformation, and primary or secondary malignancy.

Successful treatment has usually been some form of surgical excision including bronchoscopic excision, sleeve resection,[12] lobectomy and even pneumonectomy. Although the lesion is benign, recurrence with local invasiveness can occasionally occur.[1,6,9,12] Radiation therapy has been tried with some degree of success.[11]

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We wish to thank Colonel Henry T. Uhrig of Tripler Army Medical Center for his cooperation with Case II.

REFERENCES

1. BENSON, W. R. Granular cell tumors (myoblastomas) of tracheobronchial tree. *J. Thoracic & Cardiovasc. Surg.*, 1966, *52*, 17–30.
2. BUSH, R. W., and PLAIN, G. L. Granular cell tumors (myoblastomas) involving bronchus and skin: report of case. *Am. J. Clin. Path.*, 1968, *50*, 563–567.
3. FRASER, R. G., and PARÉ, J. A. Diagnosis of Diseases of The Chest. Volume II. W. B. Saunders Company, Philadelphia, 1970, pp. 734.
4. GARANCIS, J. D., KOMOROWSKI, R. A., and KUZMA, J. F. Granular cell myoblastoma. *Cancer*, 1970, *25*, 542–550.
5. KRAUS, A. R., MELNICK, P. J., and WEINBERG, J. A. Myoblastoma of bronchus. *J. Thoracic Surg.*, 1948, *17*, 382–389.
6. MILLER, D. R. Benign tumors of lung and tracheobronchial tree. *Ann. Thoracic Surg.*, 1969, *8*, 542–650.
7. MURPHY, G. H., DOCKERTY, M. B., and BRODERS, A. C. Myoblastoma. *Am. J. Path.*, 1949, *25*, 1157–1181.
8. NAIDECH, H. J., AXELROD, R. S., and SELIGER, G. Granular cell tumor (myoblastoma) of stomach. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 245–247.
9. OSTERMILLER, W. E., COMER, T. P., and BARKER, W. L. Endobronchial granular cell myoblastoma. *Ann. Thoracic Surg.*, 1970, *9*, 143–148.
10. ROBBINS, S. L. Pathologic Basis of Disease. W. B. Saunders Company, Philadelphia, 1974, pp. 1429.
11. SARGENT, E. N., WILSON, R., GORDONSON, J., and GEORGE, F. W. Granular cell myoblastoma of trachea: response to radiation therapy. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *114*, 89–92.
12. SYMBAS, P. N., LOGAN, W. D., and VAKIL, H. C. Granular cell myoblastoma of bronchus. *Ann. Thoracic Surg.*, 1970, *9*, 136–142.
13. UMANSKY, C., and BULLOCK, W. K. Granular cell myoblastoma of breast. *Ann. Surg.*, 1968, 810.
14. VANCE, S. F., and HUDSON, R. P. Granular cell myoblastoma. *Am. J. Clin. Path.*, 1969, *52*, 208–211.
15. WEITZNER, S., and OSER, J. F. Granular cell myoblastoma of bronchus. *Am. Rev. Resp. Dis.*, 1968, *97*, 923–930.

VOL. 125, NO. 4

EVALUATION OF A METHOD FOR QUANTIFYING THE ANGIOGRAPHIC SEVERITY OF MAJOR PULMONARY EMBOLISM*

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ABSTRACT:

A method has been evaluated for the quantification of the angiographic severity of pulmonary embolism. There was close agreement between observers especially in the more severely affected cases. The method has been shown to be sensitive enough to demonstrate highly significant differences between treatment regimens and to assist in the selection of treatment at the outset.

PULMONARY angiography is a precise method of diagnosing pulmonary embolism.[2] The importance of this procedure is now widely accepted [5,9] especially in therapeutic trials.[3,8,11] Miller and his colleagues[8] have devised a system for quantifying the severity of pulmonary embolism demonstrated by pulmonary angiography. We have examined the value of this method and its application in clinical practice.

MATERIAL AND METHOD

The angiograms studied were from patients entered into the Brompton-Oxford pulmonary embolism trial which compared heparin with streptokinase.[11] Pulmonary angiography was performed before and after 72 hours of treatment, and in some cases six months later.

Right heart catheterization was carried out under local anesthesia but without premedication that might have had pharmacological action on the pulmonary vasculature. A catheter of maximum possible size, up to No. 8 French and with side holes, was maneuvered into the proximal portion of the main pulmonary artery. After right heart pressures had been recorded, pulmonary angiography was carried out. Single plane anteroposterior, serial roentgenograms were taken in full inspiration and while injecting 0.5–1.0 ml/kg body weight of contrast medium (usually Triosil 75 percent—sodium, calcium, and magnesium metrizoate*) using a pressure injector. The filming program was usually six exposures per second for four seconds and then two per second for six seconds.

Method of assessing pulmonary angiograms. The method described by Miller *et al.*[8] provided a score for the embolic involvement (objective score) and for the reduction of peripheral perfusion of the lungs as judged by the opacification of the blood vessels by contrast medium (subjective score). The right and left main pulmonary arteries were regarded as having nine and seven major segmental branches respectively (Fig. 1). The presence of a filling defect, irrespective of size, in any one of these branches scored one point. The presence of a filling defect proximal to the segmental branches scored a value equal to the number of segmental branches arising distally. Where a proximal artery was involved, more peripherally placed emboli were not scored in addition. The maximum possible score for involvement was sixteen points.

This method of quantifying emboli did not distinguish between large and small filling defects. In order to compensate for

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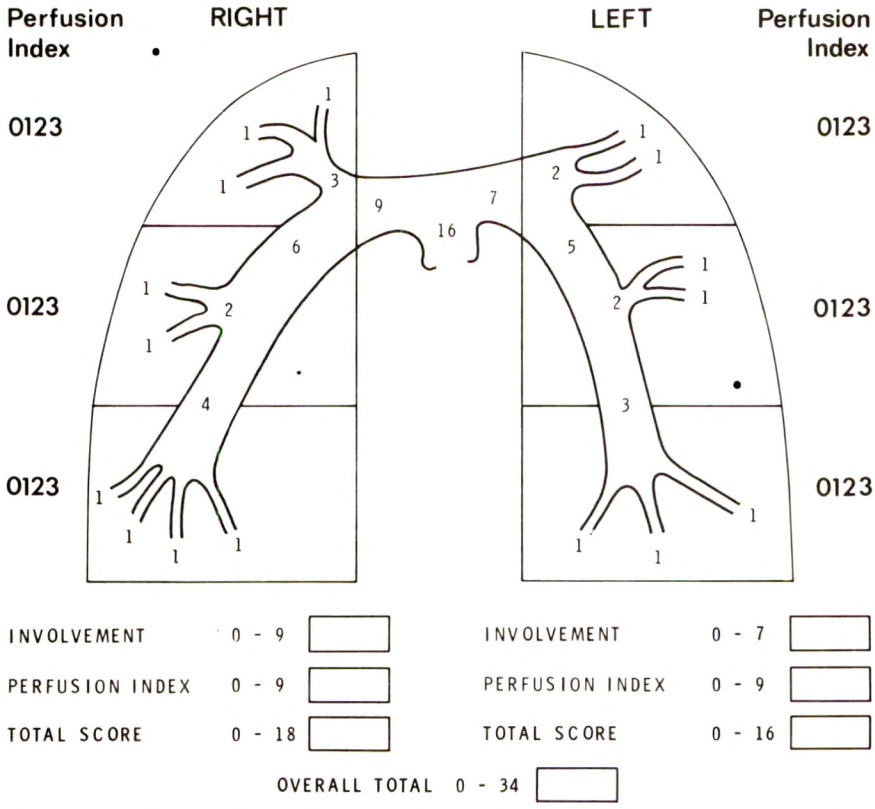

FIG. 1. Score sheet for recording the angiographic severity of pulmonary embolism. See text for explanation. (With permission from the *British Medical Journal*.)

this, a subjective assessment of the reduction in the peripheral perfusion of three zones for each lung ("perfusion index") was estimated from the opacification of the blood vessels by contrast medium. Normal opacification scored zero, mild or moderate reduction one point, severe reduction two points, and no opacification three points. The maximum score for the reduction in peripheral perfusion of each lung was nine points. Therefore the over-all maximum score possible was: 16 (embolic involvement) + 18 (perfusion index) = 34 (total angiographic score).

All pulmonary angiograms were scored by two radiologists independently (E.W.L.F. and M.L.T.). They had no knowledge of whether the roentgenograms being examined had been taken before or after treatment, and no knowledge of the clinical state of the patients, the treatment given, or their clinical response to treatment.

RESULTS

Sixty-eight angiograms were examined. The comparison between the scores from each radiologist for the embolic involvement, "perfusion index," and total angiographic scores is shown in Figure 2, *A–C*. The widest discrepancies were among the lower scores. This applied particularly to the embolic involvement score (Fig. 2*A*): Observer I considered twelve angiograms to show no vascular filling defects but Observer II agreed in only one case. The final score for each angiogram was taken as the mean of the scores from each observer. This was compared with the score given by the radiologists (Observer III) involved at the time of angiography (Fig. 3); there was close agreement and this improved as the score increased. The coefficients of

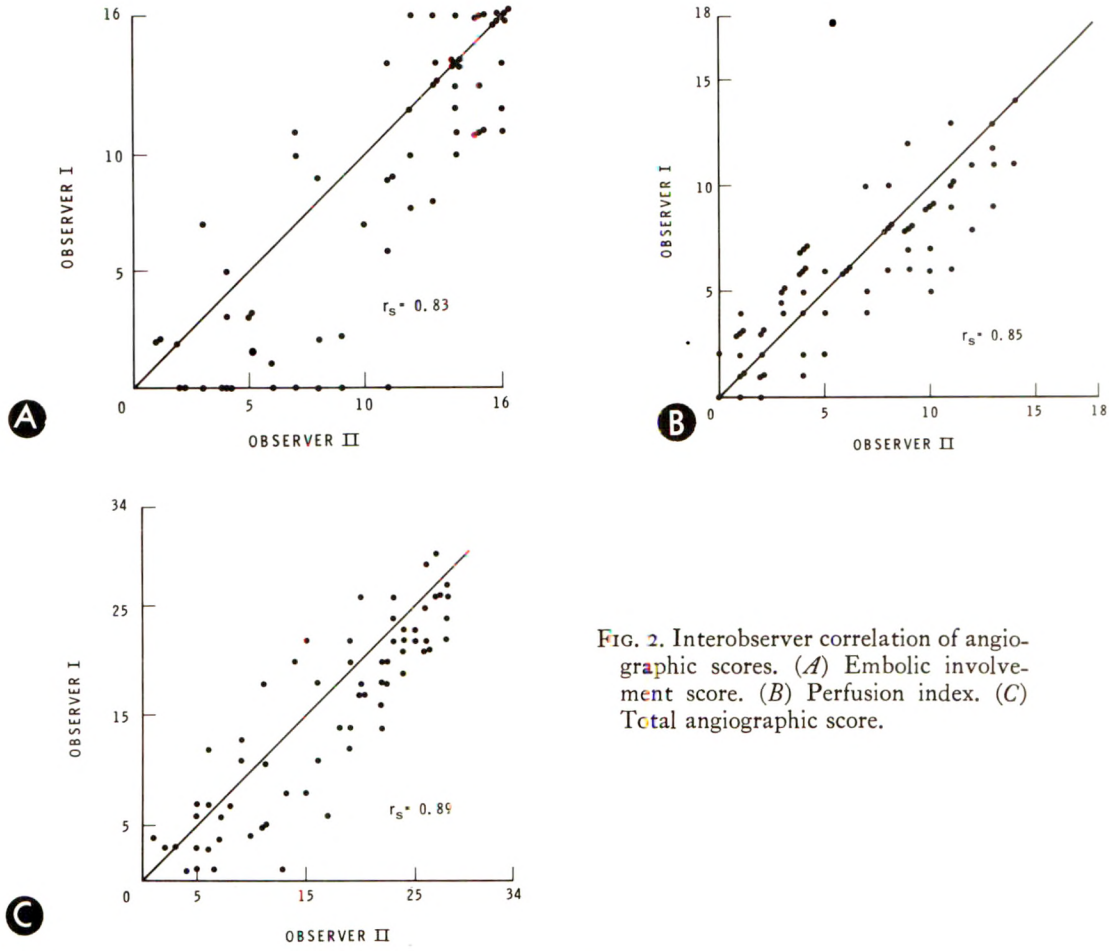

FIG. 2. Interobserver correlation of angiographic scores. (*A*) Embolic involvement score. (*B*) Perfusion index. (*C*) Total angiographic score.

correlation (Spearman rank correlation coefficient, r_s) between observers are shown on the relevant figures.

The difference between angiographic scores, before and after 72 hours of treatment, in a series of 23 patients was analyzed. The coefficient of correlation between the differences from each observer in angiographic scores before and after treatment was 0.79 (Fig. 4).

Attempts were made to relate the angiographic score to other measures of the embolic severity: no close relationship was found with right heart hemodynamics. However, although the initial systemic systolic pressure was not significantly correlated with the total angiographic score ($r_s = -0.26$), there was a significant inverse correlation between the initial systemic systolic blood pressure and the perfusion index ($r_s = -0.38$, $p. < 0.05$).

DISCUSSION

A similar method for the quantification of angiographic abnormalities was used in the Urokinase Pulmonary Embolism Trial (U.P.E.T.).[11] This method required the actual number and dimensions of filling defects to be measured. Miller's method is simpler in that the score for filling defects did not take account of their size and number, but this was compensated for by adding an estimate of the effect of emboli on the lung perfusion. The subjective aspect of the U.P.E.T. method used a four point scale for rating individual pulmonary

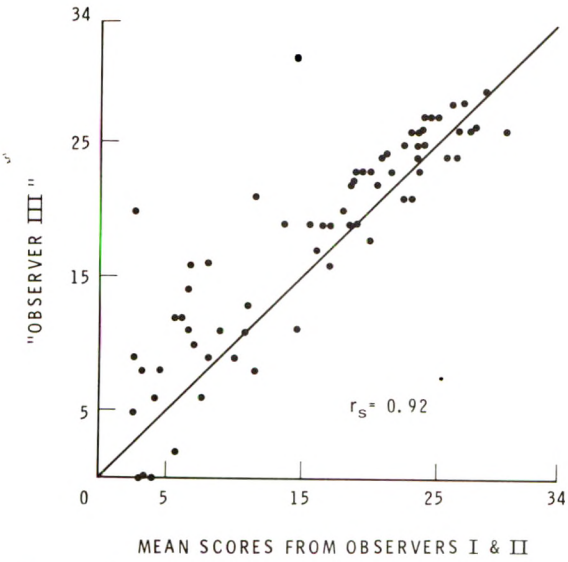

FIG. 3. Correlation of total angiographic scores from "Observer III" and mean scores from Observers I and II.

angiograms and a seven point scale for comparison of pre- and post-treatment angiograms. The subjective score for individual angiograms in Miller's system was probably made more sensitive by being built upon an 18 point scale. The maximum objective scores in these two methods were similar: 18 in the U.P.E.T. and 16 in Miller's method.

The interobserver agreement on the various scores analyzed in this paper is high (r_s=0.79–0.92) and this, coupled with the simplicity of the method, demonstrates its value.

There are three applications of a method for quantifying the angiographic severity of pulmonary embolism:[12]

1. Communication "vocabulary."
2. Assessment of resolution of pulmonary emboli and efficacy of treatment.
3. Classification of patients into severity groups with resulting aid in selection of treatment.

The method evaluated in this paper has proved itself useful in these respects. It has been used satisfactorily in a trial comparing streptokinase and heparin in the treatment of life-threatening pulmonary embolism:[11] the angiographic scores revealed a highly significant difference ($p<0.001$) between the treatment groups whereas other measures (*e.g.*, pulmonary arterial pressure) of improvement did not show such marked differences.

It has been frequently emphasized that pulmonary angiography is mandatory before embolectomy is attempted.[4,5,10] Cross and Mowlem[1] found a high (100 percent) mortality rate when no embolus was discovered at operation; most of the deaths were from myocardial infarction. Poor peripheral perfusion on the angiogram is associated with a poor prognosis,[6] with an increased failure rate of medical treatment, and with need for embolectomy.[11] The agreement between observers tended to be greater at higher angiographic scores (Fig. 2; and 3): this is of particular importance since it was with high initial scores (<24) and systemic arterial hypotension (systolic <100 mm Hg) that medical treatment failed.

In conclusion we would advocate the

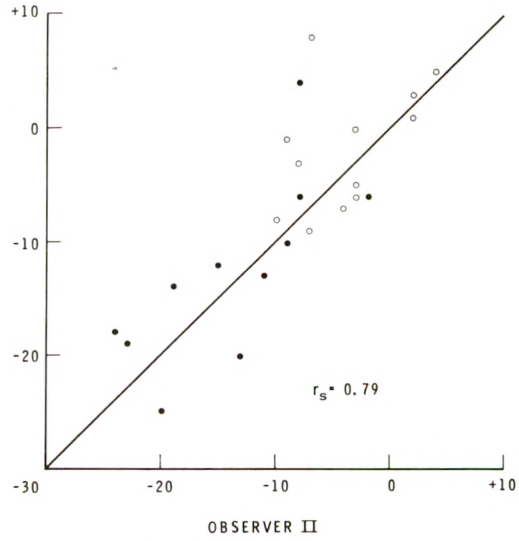

FIG. 4. Interobserver correlation of improvement scores (difference between pre- and post-treatment angiographic scores).

● Streptokinase. ○ Heparin.

quantification of the angiographic severity of major pulmonary embolism. The method described is acceptably reproducible, has clearly distinguished between the efficacy of two treatment regimens, and may help in the prediction of the likely response to medical treatment and of the need for surgery.

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REFERENCES

1. Cross, F. S., and Mowlem, A. Survey of current status of pulmonary embolectomy for massive pulmonary embolism. *Circulation*, 1967, *35*, Suppl. 1, 86–91.
2. Dalen, J. Pulmonary angiography in pulmonary embolism. *Bull. de Physiopath. Resp.*, 1970, *6*, 45–63.
3. Edwards, I. R., MacLean, K. S., and Dow, J. D. Low dose urokinase in major pulmonary embolism. *Lancet*, 1973, *2*, 409–413.
4. Gahagan, T., Gale, H. H., and Ormond, R. Pulmonary angiograms in management of pulmonary embolism. *Am. J. Surg.*, 1966, *111*, 718–727.
5. Hume, M., Sevitt, S., and Thomas, D. P. In: Venous Thrombosis and Pulmonary Embolism. Harvard University Press, Cambridge, 1970.
6. Kakkar, V. V., and Raftery, E. B. Selection of patients with pulmonary embolism for thrombolytic therapy. *Lancet*, 1970, *2*, 237–241.
7. Lowman, R. M., Rearden, J., Hipona, F. A., Stern, H., and Toole, A. L. Role of pulmonary angiography in pulmonary embolism: comprehensive view of roentgen findings. *Angiology*, 1971, *18*, 291–305.
8. Miller, G. A. H., Sutton, G. C., Kerr, I. H., Gibson, R. V., and Honey, M. Comparison of streptokinase and heparin in treatment of isolated acute massive pulmonary embolism. *Brit. M. J.*, 1971, *2*, 681–684.
9. Oakley, C. M. Diagnosis of pulmonary embolism. *Brit. M. J.*, 1970, *2*, 773–777.
10. Sabiston, D. C., Stein, M., Simon, M., and Littman, D. Pathophysiology of pulmonary embolism: relationships to accurate diagnosis and choice of therapy. *J. Thoracic & Cardiovasc. Surg.*, 1965, *50*, 339–351.
11. Tibbutt, D. A., Davies, J. A., Anderson, J. A., Fletcher, E. W. L., Hamill, J., Holt, J. M., Lea Thomas, M., Lee, G.de J., Miller, G. A. H., Sharp, A. A., and Sutton, G. C. Comparison by controlled clinical trial of streptokinase and heparin in treatment of life-threatening pulmonary embolism. *Brit. M. J.*, 1974, *1*, 343–347.
12. Walsh, P. N., Greenspan, R. H., Simon, M., Simon, A. L., Hyers, T. M., Woosley, P. C., and Cole, C. M. Angiographic severity index for pulmonary embolism. *Circulation*, 1973, *47* and *48*, Suppl. 2, 101–108.

PATHWAYS OF EXTRAPELVIC SPREAD OF DISEASE: ANATOMIC-RADIOLOGIC CORRELATION*

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ABSTRACT:

Extrapelvic spread of disease, particularly from gastrointestinal tract perforations which may be clinically occult, may first present in the buttock, hip, thigh, and even lower leg, and the extraperitoneal space of the abdomen itself. Clinical manifestations at these remote sites may be very misleading. Anatomic and roentgenologic observations establish the preferential pathways of extrapelvic spread. These are related to the insertions and fascial investments of the iliopsoas, pyriformis, and obturator internus muscles and the ensheathed penetrations of the superior gluteal arteries. Superiorly, extension from the pelvic tissues seeks out the posterior pararenal compartment of the extraperitoneal region of the abdomen. Roentgenologic signs may first identify the presence, extent, and localization of the primary process.

DISEASES arising from the pelvic contents may first manifest themselves by signs and symptoms remote from their source of origin. Gastrointestinal tract perforations in particular may dissect along anatomic planes of the pelvis to first present in the buttock, hip, thigh and even lower leg, and the retroperitoneal space of the abdomen itself. The occurrence of pain, mass, or crepitation at these sites may be very misleading since the origin of the underlying inflammatory condition or the neoplastic, traumatic, or foreign body perforation of the bowel within the pelvis often remains clinically occult. Roentgenologic evaluation may be crucial in redirecting the diagnostic and therapeutic approach as well as in documenting the extent of disease.

Basic to an appreciation of the clinical significance is an understanding of the anatomic pathways permitting spontaneous dissection of processes from the pelvis to these sites. These are dependent upon the course of certain pelvic muscles and vessels and their particular fascial relationships. The purpose of this report is: (1) to establish the preferential planes of extrapelvic spread, based upon detailed analysis of anatomic sections derived from frozen cadavers and recently reported injection studies;[14,16] and (2) to document the diagnostic roentgenologic features of extrapelvic spread which may be of critical clinical importance in identifying the source of origin.

ANATOMIC CONSIDERATIONS

The levator ani and coccygeus muscles form the floor of the pelvis (pelvic diaphragm). This is the most inferior portion of the body wall and closes the abdominopelvic cavity. The obturator internus and pyriformis muscles originate within the pelvis and thus form part of the pelvic wall, but they are really muscles of the lower limbs. Figure 1 demonstrates an anatomic section showing the relationships of the major muscles of the pelvis.

The endopelvic fascia is the internal investing fascia of the pelvis. Its parietal layer (Fig. 2) covers the levator ani, coccygeus, and the intrapelvic portions of the obturator internus and the pyriformis muscles. It is directly continuous above with the transversalis (endo-abdominal) fascia lining the abdominal cavity. The visceral layer of the endopelvic fascia covers the urinary bladder, lower third of the ureters, uterus, vagina, and the rectum.

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Four of the pelvic muscles have important extra-pelvic insertions. These are illustrated in Figure 3:

(1) The psoas major originates from the transverse processes and bodies of the lumbar vertebrae and inserts on the lesser trochanter of the femur.

(2) The iliacus originates primarily from the upper two-thirds of the iliac fossa and the ala of the sacrum and inserts with the tendon of the psoas major on the lesser trochanter of the femur. In its common insertion, it comprises the iliopsoas muscle group.

(3) The pyriformis originates from the sacrum and the margin of the greater sciatic foramen and inserts on the greater trochanter of the femur.

(4) The obturator internus originates from the margins of the obturator foramen and inserts on the greater trochanter of the femur.

It is also important to recognize that the pelvic blood vessels lie in front of the pelvic fascia, *i.e.*, superficial to the parietal layer of the endopelvic fascia. The superior and inferior gluteal branches of the internal iliac arteries passing out of the pelvis (Fig. 1) must perforate the fascia and carry extensions of the fascia with them to the gluteal region. For example, the fascia of the pyriformis is prolonged outward through the greater sciatic foramen and joins the gluteal fascia. The internal iliac vessels and their branches lie in the subperitoneal tissue in front of the fascia, and the branches to the gluteal region, particularly the superior gluteal artery (the largest branch of the hypogastric artery), emerge in special sheaths of this tissue, paralleling the border of the pyriformis, to reach and supply the gluteus muscles.

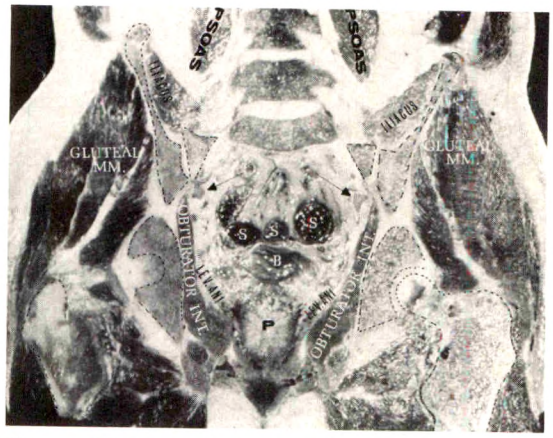

Fig. 1. Mid-coronal anatomic section through the pelvis at the level of the urinary bladder (B), prostate gland (P), and loops of sigmoid colon (S). The course and relationships of the psoas major, iliacus, obturator internus, levator ani, and gluteal muscles are shown. These are invested by fascia. The superior gluteal vessels (arrows) pass out of the pelvis at the level of the greater sciatic foramina.

In this way, the fascial investments of the iliopsoas, pyriformis and obturator internus muscles and the fascial sheaths of the superior gluteal arteries provide anatomic pathways from the pelvis to the buttocks, hips, and thighs. Superiorly, the pelvic tissues above the levator ani and coccygeus muscles are continuous with the extraperitoneal portion of the abdomen.

Report of Cases

Case 1. *Rectal extravasation complicating a barium enema study.* This 45 year old female with a questionable history of colitis in the past presented with recent change in bowel habits. Barium enema study disclosed no abnormalities. However, the post-evacuation film demonstrated extravasation into the wall of the rectum and pelvic tissues (Fig. 4*A*). Dissection of the contrast agent along the fascial sheath of the left superior gluteal artery was also noted (Fig. 4*B*).

Within several hours, the patient developed rectal bleeding and point tenderness in the left buttock. Proctoscopy identified a false lumen 15 cm from the anus. Early cellulitis of the pelvic tissues was surgically drained and the patient recovered uneventfully.

Comment. Dissection from the pelvis toward the gluteal area is strikingly illustrated in this case by the progress of the extravasated contrast agent into the fascial extension of the superior gluteal artery through the greater sciatic foramen. Indeed, the vessel is shown as a tubular "filling defect" within its opacified endopelvic sheath. Tenderness in the buttock

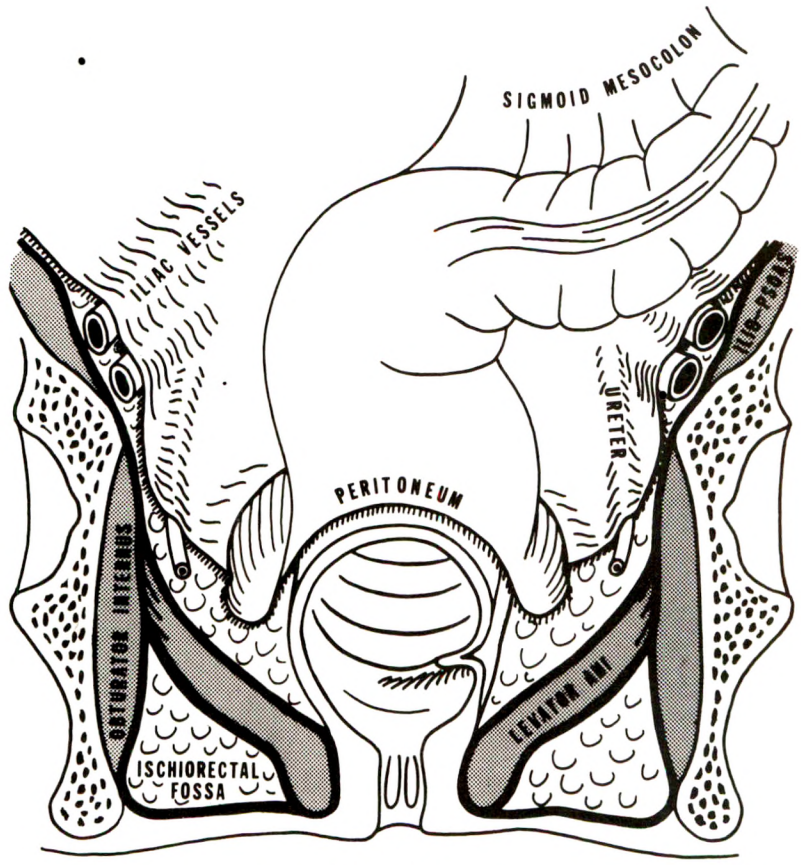

FIG. 2. Drawing of posterior coronal section at level of the rectum showing planes of pelvic fascia (bold lines). The endopelvic fascia covers the major muscles of the pelvis. The internal iliac arteries lie superficial to the fascia. Their gluteal branches perforate the endopelvic fascia and then continue within tunneled extensions as they pass to the gluteal region.

was a consequence of this anatomic extension.

CASE II. *Sigmoid diverticulitis perforating to the right buttock.* This 75 year old female presented with a two week history of fever and vague pain in the right buttock. Barium enema study revealed inflammatory narrowing of a segment of the sigmoid colon, from which a sinus tract extended along the course of the superior gluteal artery toward the right buttock (Fig. 5*A*). She was treated conservatively with antibiotics with a diagnosis of ruptured diverticulitis. Failure to improve led to a second barium enema examination ten days later. This now showed persistent opacification of the sinus tract with the development of a gluteal abscess (Fig. 5, *B* and *C*). Sigmoid resection with drainage of the abscess was accomplished. Postoperatively, however, the patient remained septic and expired.

Comment. Perforation of the sigmoid colon in this case is directed along the extension of the superior gluteal artery through the greater sciatic foramen to the right buttock. The initial symptom of pain in this site heralded the progression to a prominent gluteal abscess.

CASE III. *Sigmoid diverticulitis perforating to the left buttock and hip.* This 57 year old male presented with fever and pain in the left hip. Previous history included several episodes of left lower quadrant pain. Barium enema study demonstrated inflammatory narrowing of the distal sigmoid colon. Loculated extravasation

from this area along the plane of the pyriformis muscle extended to the left buttock and hip (Fig. 6, *A* and *B*). Subsequently, sigmoid resection and drainage confirmed the diagnosis of perforating diverticulitis.

Comment. The presenting symptom of pain in the hip was secondary to dissection from the perforation of the sigmoid colon within the fascial investment of the pyriformis muscle. The tract can be seen to lead toward the greater trochanter of the femur.

Case IV. *Appendicitis perforating to the right thigh.* This 69 year old male was admitted because of right flank pain and tenderness. There was a negative psoas sign, but pain was elicited on straight leg raising and adduction of the right thigh. Digital insertion into the right inguinal ring caused pain radiating to the right hip. White blood cell count was 15.2 million/mm³. Fever and hip pain increased and tenderness developed over the greater trochanter of the femur. A clinical diagnosis of bursitis of the right hip was made and the area was injected with cortisone and xylocaine. This was followed by sepsis, progressive hip pain, and tenderness in the right lower quadrant of the abdomen. The pain then extended to the right knee, and

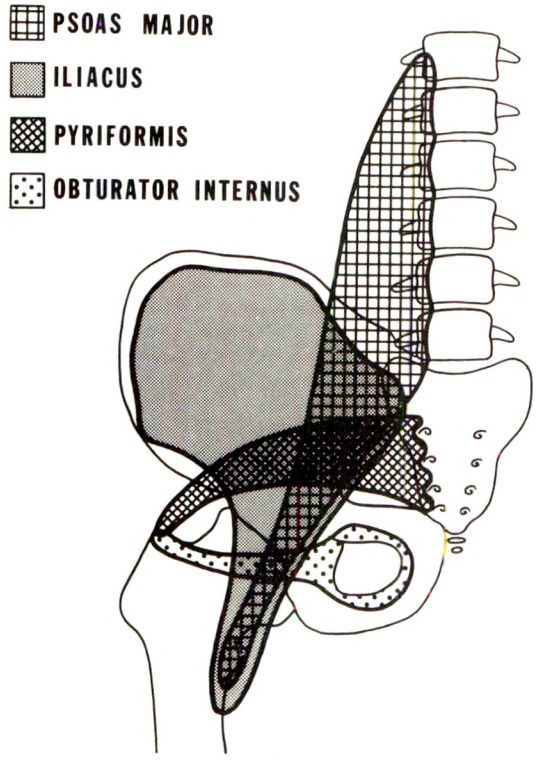

Fig. 3. Extrapelvic insertions of abdominal and pelvic muscles. The iliopsoas muscle group inserts medially on the lesser trochanter of the femur, and the pyriformis and obturator internus insert laterally on the greater trochanter.

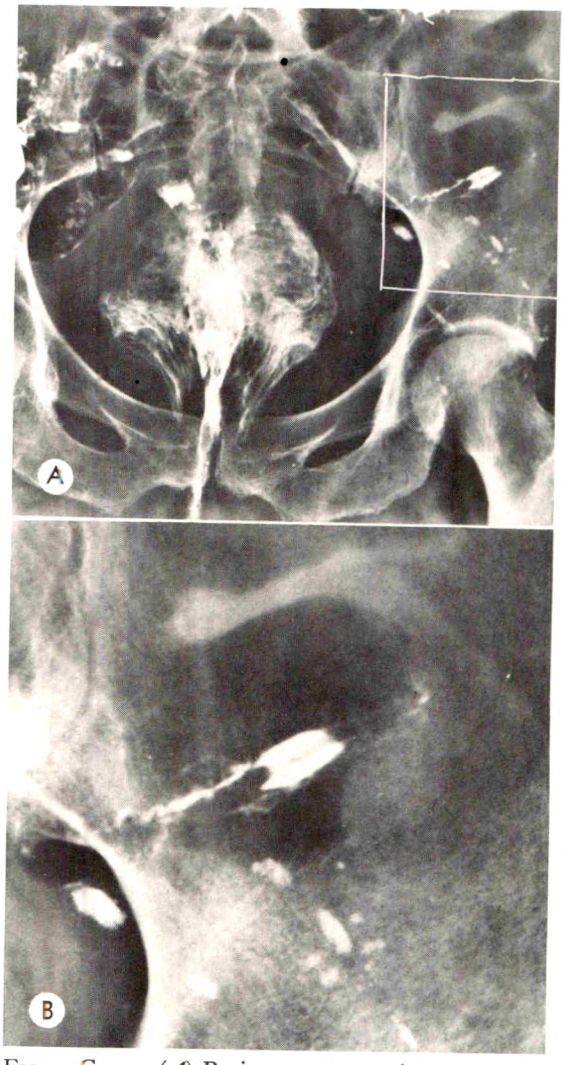

Fig. 4. Case I. (*A*) Barium enema study, postevacuation roentgenogram. Contrast agent has infiltrated the intramural and extrarectal pelvic tissues. (*B*) Magnification of area outlined in *A* demonstrates extravasation within the tunneled fascial sheath of the superior gluteal artery progressing through the greater sciatic foramen toward the buttock.

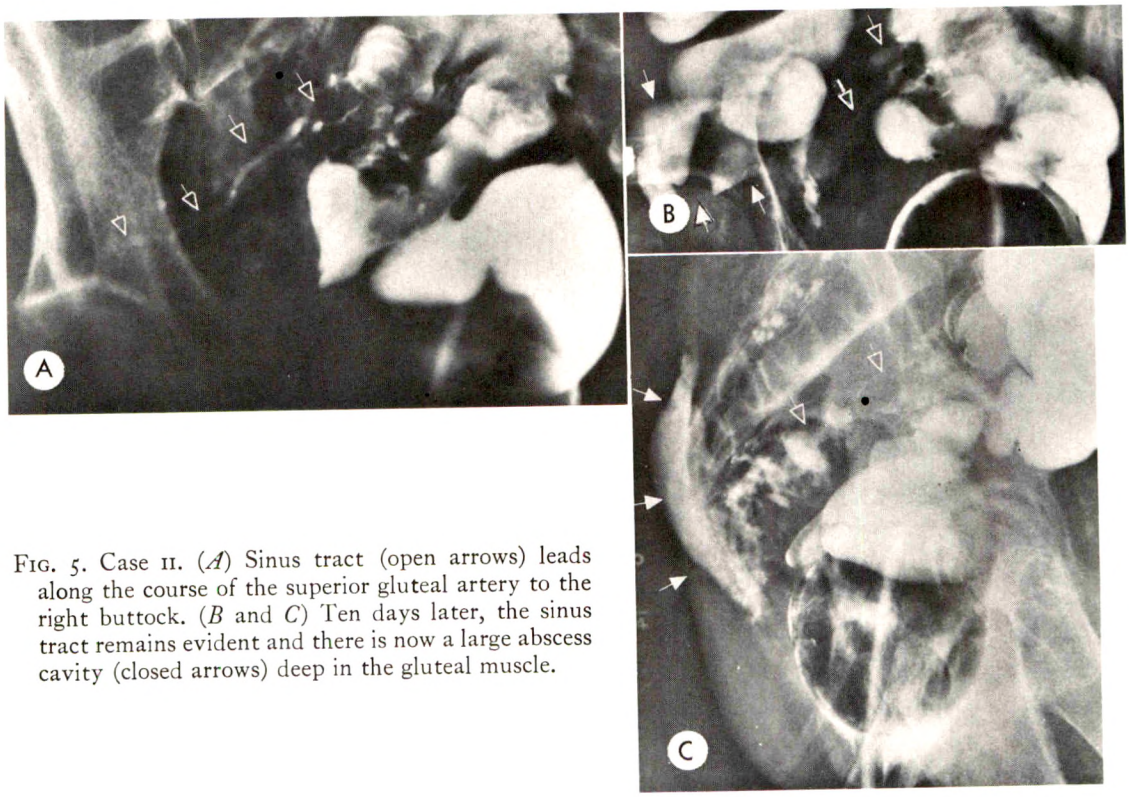

FIG. 5. Case II. (*A*) Sinus tract (open arrows) leads along the course of the superior gluteal artery to the right buttock. (*B* and *C*) Ten days later, the sinus tract remains evident and there is now a large abscess cavity (closed arrows) deep in the gluteal muscle.

increased temperature of the right lower extremity was noted. A violaceous discoloration below the right inguinal ligament and induration of the lateral aspect of the thigh quickly developed. A roentgenogram of the right lower extremity disclosed interstitial emphysema lateral to the femur (Fig. 7*A*). Antibiotics were begun and aspiration of the thigh cultured *E. coli*.

Barium enema study one month after admission demonstrated reflux into the terminal ileum but no opacification of the appendix. A prominent filling defect near the base of the appendix at its junction with the cecum was present (Fig. 7*B*).

The patient subsequently developed respiratory acidosis and died of cardiac arrest five weeks after admission.

Autopsy disclosed that the appendix, which was located retrocecally between the lateral margin of the psoas muscle and the floor of the right iliac fossa, was inflamed and ruptured. A path of inflammatory exudate dissected the fascial planes and extended along the iliopsoas muscle under the inguinal ligament and into the right thigh where it collected between the necrotic iliopsoas muscle and the sartorius muscle anterior to the femoral canal.

Comment. Necrotic cellulitis and interstitial emphysema of the right thigh progressed *via* the iliopsoas muscle group from a perforated appendicitis which was not diagnosed ante mortem. The ultimate isolation of *E. coli* indicated origin from the gastrointestinal tract. The findings of the barium enema examination reflect obstruction of the appendiceal lumen.

CASE V. This 62 year old male was admitted for cardiac catheterization following mitral valve replacement. Difficulty in passing the catheter through the iliac artery was encountered. Contrast agent was introduced to evaluate the cause of the difficulty. This demonstrated localized loculated extravasation. He developed right lower quadrant guarding and tenderness.

An abdominal roentgenogram now documented, in addition to the localized extravasation from the right external iliac artery, a large extraperitoneal hematoma within the right posterior pararenal compartment (Fig. 8).

At surgery, repair of the perforated iliac ar-

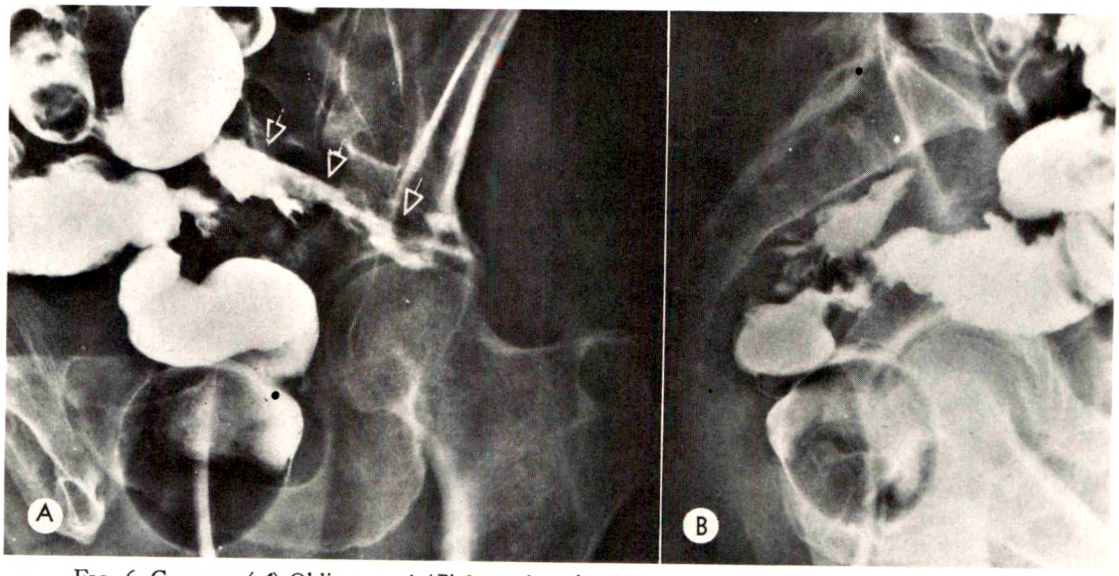

Fig. 6. Case III. (*A*) Oblique and (*B*) lateral projections of barium enema study demonstrate extension along the course of the pyriformis muscle (arrows).

tery was done and the extraperitoneal hematoma of approximately 1,800 cc was drained.

Comment. Superior flow from the pelvis can be traced to seek preferentially the posterior pararenal compartment[16] of the extraperitoneal tissues of the abdomen. The characteristic features allow precise roentgenologic localization.

DISCUSSION

The most common extra-abdominopelvic extension of gastrointestinal tract perforations, beyond the development of ischiorectal abscesses, is to the buttocks, hips, and lower extremities. Review of the literature shows that 21 cases have been reported as subcutaneous emphysema of the leg arising from intestinal perforations (Table 1). Diverticulitis and appendicitis with perforation and fistula formation accounted for eleven of the twelve cases resulting from infection. Carcinoma of the colon with perforation accounted for six, with uterine carcinoma, foreign body, and trauma to the rectum causing the remaining.

Most of the cases of extrapelvic spread reported have created serious diagnostic problems with admitting diagnoses of thrombophlebitis, sciatica, gas gangrene, inguinal abscess, Spigelian hernia, and even fractured hip, until surgery or autopsy uncovered a definitive diagnosis. The clinical presentation is often pain in the hip or buttock.[4,11–13,18,19] With the development of crepitation, particularly in the thigh,[4,11,18,21] incision may then display severe necrotizing fasciitis. It is often not until blunt dissection is then carried out along the femoral canal beneath the inguinal ligament that an extraperitoneal abscess is revealed as evidence of a more severe process extending from the pelvis.

Surgical findings have not often localized the particular pathway of spread in the face of a pelvic abscess with frequent necrotizing dissection toward the inguinal region, perineum, thigh and hip joint, buttock, and paravertebral gutter. Only six previous reports document the development of an iliopsoas abscess as the major source of the extra-pelvic spread under the inguinal ligament to the leg.[2,4,11,13,19,22] In two, a sinus tract was noted extending from a perforated lesion of the sigmoid colon through the sacrosciatic notch to an abscess cavity with emphysematous cellulitis lateral to the hip and down the thigh.[1,13]

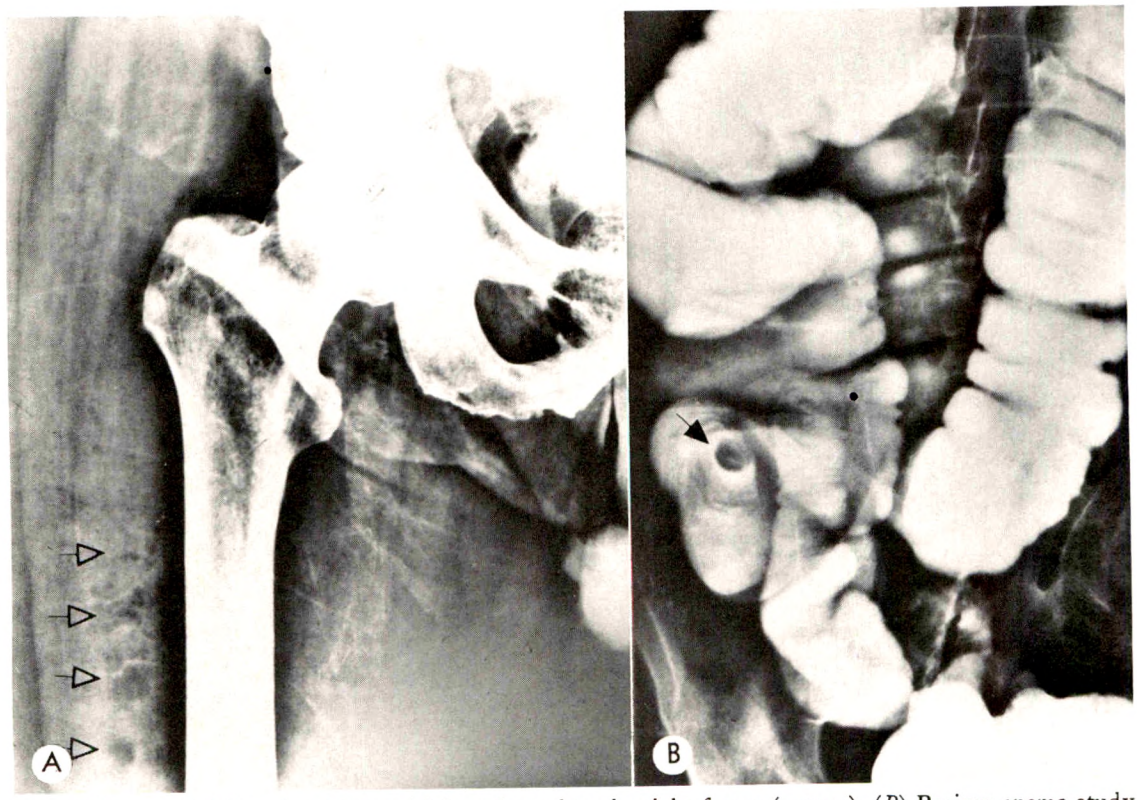

FIG. 7. Case IV. (*A*) Loculated interstitial gas lateral to the right femur (arrows). (*B*) Barium enema study demonstrates non-opacification of the appendix with a prominent defect near its confluence with the cecum (arrow).

The pathogenesis of subcutaneous cellulitis and emphysema of gastrointestinal origin is dependent upon four basic factors:[17] (1) perforation of the bowel; (2) an adequate pressure gradient between the lumen of the bowel and ultimately the subcutaneous space; (3) the anatomic site of perforation; and (4) infection.

The major mechanism of gas formation is not the gas-forming organisms present in the feces, but the pressure gradient between the gut lumen and the surrounding tissue. Quigley and Brody[20] have recorded large pressure gradients associated with vigorous peristaltic contractions. Gas pressures in the intestinal lumen may rise to more than 60 cm of H_2O during peristalsis while soft-tissue tension is usually about 5 cm H_2O. The intra-abdominal pressure may be greatly increased by the contraction of the diaphragmatic and abdominal muscles. This increased pressure is transmitted to the contents of the intestinal tract and promotes their evacuation, especially at sites of perforation when the sphincters are intact.[6,20] This mechanism, enhanced by bacterial gas formation, accounts for the rapidity of its accumulation.

The most common offending organism is *E. coli*. Occasionally, cultures isolate *Clostridium welchii*,[1,21] *Aerobacter aerogenes*,[4,21] and *Proteus*.[10,13]

The anatomic site of perforation largely determines the pathway of spread of the sinus tract to the subcutaneous position. The usual route of extravasation from the bowel is directly through a pathologic defect in the parietal peritoneum or fascia contiguous with this defect into the intermuscular planes and subcutaneous spaces. Solid parenchymatous organs and serous membranes have a relatively great resistance to the diffusion of gas, whereas loose areolar and fascial structures readily allow the passage of gas.

This report establishes the correlation

between the anatomic pathways and the clinical documentation of the extrapelvic spread of disease. The insertions of the iliopsoas, pyriformis and obturator internus muscles, within their fascial investments, and the ensheathed penetrations of the superior gluteal arteries provide avenues of dissection to the buttocks, hips, and thighs.

Roentgenologic signs may first identify the presence, extent, and localization of the primary process. Subcutaneous emphysema presents as gas in the fascial planes between the muscles and interstitial tissues in contrast to gas gangrene where the gas lies within the muscles. In the hip, it may first appear about the greater or lesser trochanter.[4,11,18,21,22] Linscheid *et al.*[13] identified a gas-filled sinus tract extending from a sigmoid perforation externally through the sciatic notch to emphysematous cellulitis about the trochanter and neck of the femur. Contrast studies readily demonstrate extravasation along the anatomic planes.

It is apparent that the first site of appearance or area of maximum development of cellulitis or subcutaneous emphysema should indicate the approximate anatomic level and likely nature of perforation of the bowel. Cases of perforated diverticulitis or carcinoma of the sigmoid or descending colon (Case III) thus may present in the left leg or buttock,[1–4,8] and appendicitis or perforation of the cecum (Case IV) in the right leg and buttock.[10,19,23] Because of variations in position and length of the appendix and in redundancy of the sigmoid, perforations from these sites may extend to the side opposite to that ordinarily expected (Case II). Traumatic or neoplastic perforation of the rectum may present in either[5] or both[9] legs. Spread may occur also into the abdominal wall[4,22,23] and upwards as a retroperitoneal space abscess.[1–3,21] Three clearly demarcated compartments within the extraperitoneal region of the abdomen have been recently described anatomically and identified roentgenologically.[14–16] These constitute the anterior pararenal, perirenal, and posterior pararenal spaces. Fluid or gas collections within each present distinguishing characteristics which are often clinically helpful in localizing the primary site of disease. As Case V illustrates, the preferential spread upwards from the subperitoneal pelvic

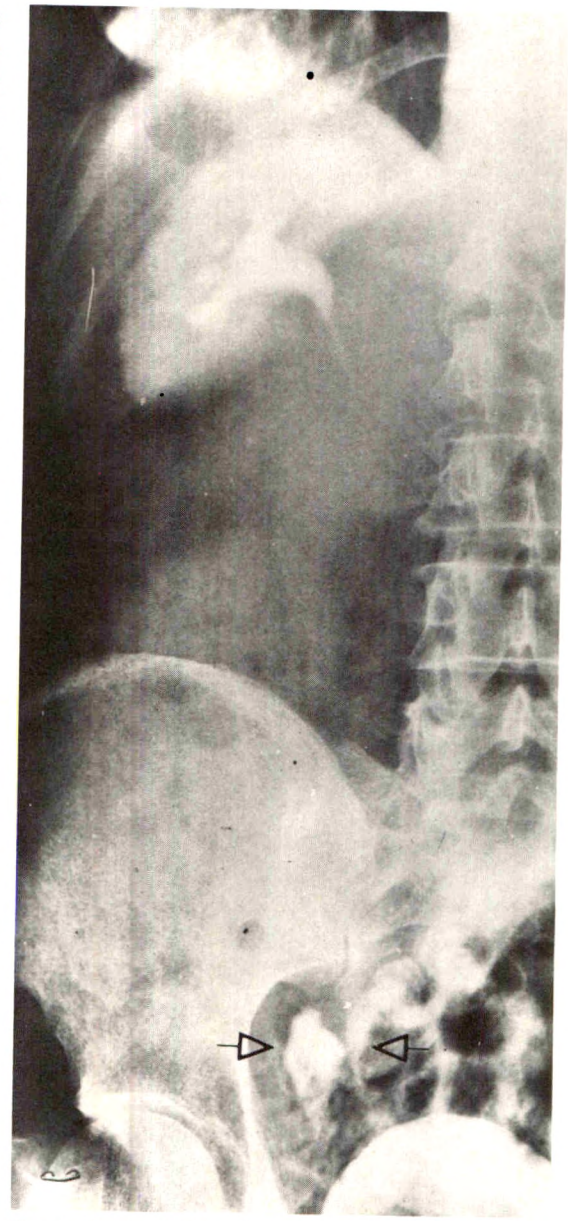

Fig. 8. Case V. Delayed roentgenogram demonstrates persistent localized extravasation (arrows) from the right external iliac artery. An acute soft-tissue mass occupies the right abdomen. This displaces the lower pole of the kidney laterally, obstructs the ureter, and obliterates the lateral edge of the psoas muscle and the flank stripe. These features localize the hematoma to the extraperitoneal posterior pararenal compartment.

TABLE I

SUMMARY OF 21 REPORTED CASES OF SUBCUTANEOUS EMPHYSEMA OF LEG ARISING FROM GASTROINTESTINAL ORIGIN

Author	Age and Sex	Cause
Barrington and Gardham 1932[3]	52 M	Diverticulitis of descending colon
Gillies 1941[9]	63 M	Carcinoma of rectum
Dawson and Hardy 1948[8]	73 F	Diverticulitis of sigmoid colon
Spring and Kahn 1951[23]	49 M	Gangrenous appendicitis
Gow 1951[10]	58 F	Cecal perforation secondary to periureteral abscess after abdominal hysterectomy
Bernstein *et al.* 1952[5]	49 F	Traumatic perforation of rectum
Beaconsfield 1953[4]	35 F	Carcinoma of sigmoid colon
Pierleoni and Johnson 1955[19]	74 M	Ruptured retrocecal appendix
	39 F	Ruptured appendix
Ainsworth 1959[1]	56 F	Diverticulitis of sigmoid colon
	62 F	Carcinoma of rectosigmoid colon
Lee 1960[12]	72 F	Carcinoma of cecum
Altemeier and Alexander 1961[2]	72 F	Carcinoma of colon
Shambaugh and Burgert 1962[22]	70 F	Carcinoma of descending colon
Linscheid *et al.* 1963[13]	66 F	Perforation of sigmoid colon secondary to recurrent uterine carcinoma
	41 F	Diverticulitis of sigmoid colon
Case Records of the Massachusetts General Hospital 1965[7]	60 M	Diverticulitis of sigmoid colon
Shaffer 1969[21]	51 M	Foreign body perforation of malrotated, retroperitoneal terminal ileum
Korsten *et al.* 1973[11]	65 F	Ruptured diverticulum of ascending colon
Pickels *et al.* 1974[18]	76 M	Ruptured diverticulum of transverse colon
Meyers and Goodman 1975	69 M	Ruptured appendix

tissues is into the posterior pararenal compartment. Extravasation of fluid or early abscess formation in this space is revealed by the inferolateral axis of the soft-tissue density with lateral displacement of the lower renal pole, and obscuration of the psoas margin and flank stripe.[14,16] Extraluminal gas similarly shows localizing anatomic distribution and roentgenographic features.[15] This may be seen with perforations of the rectum or, rarely, of the sigmoid colon between the leaves of its mesocolon.

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REFERENCES

1. AINSWORTH, J. Emphysema of leg following perforation of pelvic colon or rectum. *Brit. J. Radiol.*, 1959, *32*, 54–55.
2. ALTEMEIER, W. A., and ALEXANDER, J. W. Retroperitoneal abscess. *A.M.A. Arch. Surg.*, 1961, *83*, 512–524.
3. BARRINGTON, F. J. F., and GARDHAM, A. J. Diverticulitis of large intestine. *Clin. J.*, 1932, *61*, 337–343.
4. BEACONSFIELD, P. Carcinoma of sigmoid presenting as emphysematous cellulitis of leg. *Ann. Surg.*, 1953, *138*, 919–921.
5. BERNSTEIN, W. C., GHENT, C. H., and REA, C. E. Emphysema of leg following perforation of rectum. *Minnesota Med.*, 1952, *35*, 1138–1139.
6. BURT, C. V. Pneumatic rupture of intestinal canal with experimental data showing mechanism of perforation and pressure required. *Arch. Surg.*, 1931, *22*, 875–902.
7. Case records of The Massachusetts General Hospital. *New England J. Med.*, 1965, *273*, 387–393.
8. DAWSON, R. L. G., and HARDY, R. H. Diverticulitis presenting as emphysematous cellulitis of leg: clinical report on 2 cases. *Brit. M. J.* 1948, *1*, 498–499.

9. Gillies, G. I. Interstitial emphysema in diabetes mellitus due to colon bacillus infections: report of four cases. *J.A.M.A.*, 1941, *117*, 2240–2242.
10. Gow, J. C. Case of reno-inguinal fistula due to periureteric abscess. *Brit. J. Surg.*, 1951, *39*, 91–92.
11. Korsten, J., Mattey, W. E., Bastidas, J., and Sicat, L. C. Subcutaneous emphysema of thigh secondary to ruptured diverticulum of ascending colon. *Radiology*, 1973, *106*, 555–556.
12. Lee, J. B. Cystitis emphysematosa. *A.M.A. Arch. Int. Med.*, 1960, *105*, 618–626.
13. Linscheid, R. L. Kelly, P. J., and Symmonds, R. E. Emphysematous cellulitis of hip and thigh resulting from enteric fistula. *J. Bone & Joint Surg.*, 1963, *45*, 1691–1697.
14. Meyers, M. A. Acute extraperitoneal infection. *Seminars in Roentgenol.*, 1973, *8*, 445–464.
15. Meyers, M. A. Radiologic features of spread and localization of extraperitoneal gas and their relationship to its source: anatomical approach. *Radiology*, 1974, *111*, 17–26.
16. Meyers, M. A., Whalen, J. P., Peelle, K., and Berne, A. S. Radiologic features of extraperitoneal effusions: anatomic approach. *Radiology*, 1972, *104*, 249–257.
17. Oetting, H. K., Kramer, N. E., and Branch, W. E. Subcutaneous emphysema of gastrointestinal origin. *Am. J. Med.*, 1955, *19*, 872–886.
18. Pickels, R. F., Karmody, A. M., Tsapogas, M. J., and Griffin, P. Subcutaneous emphysema of lower extremity of gastrointestinal origin: report of case. *Dis. Colon & Rectum*, 1974, *17*, 82–86.
19. Pierleoni, E., and Johnson, J. Perforation of vermiform appendix into retroperitoneal space. *Gastroenterology*, 1955, *29*, 308–317.
20. Quigley, J. P., and Brody, D. A. Physiologic and clinical consideration of pressures developed in digestive tract. *Am. J. Med.*, 1952, *13*, 73–81.
21. Shaffer, R. D. Subcutaneous emphysema of leg secondary to toothpick ingestion. *A.M.A. Arch. Surg.*, 1969, *99*, 542–545.
22. Shambaugh, P., and Burgert, P. H. Subcutaneous emphysema in cancer of colon. *A.M.A. Arch. Surg.*, 1962, *85*, 344–347.
23. Spring, M., and Kahn, S. Nonclostridial gas infection in diabetic. *Arch. Int. Med.*, 1951, *88*, 373–377.

RECTAL AND SIGMOID INVOLVEMENT SECONDARY TO CARCINOMA OF THE PROSTATE*

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ABSTRACT:

Three types of involvement of the rectum and recto-sigmoid by carcinoma of the prostate are reviewed through an analysis of eight cases. A fourth type with subserosal metastatic implant of the proximal sigmoid may occasionally be encountered. The roentgenographic findings are not pathognomonic, but are characteristic of extrinsic involvement of the bowel wall.

When clinical symptoms are predominantly related to the bowel, carcinoma of the prostate is usually advanced. All patients presented with bone metastases, ureterohydronephrosis, lack of function of one kidney, or both bone metastases and urinary tract obstruction. Rectoscopy and biopsy are helpful. However, biopsy specimens often show non-diagnostic features in secondary malignancy.

Correct diagnosis is important, since there is a difference in treatment of primary carcinoma and of secondary involvement of the rectum by prostatic carcinoma. A diagnostic challenge exists if the patient is evaluated by barium enema examination for primary bowel symptoms, in particular, large bowel obstruction.[4,7,13] At this time intravenous pyelography and bone survey for metastases may not be available to suggest the correct diagnosis. More widespread use of barium enema examinations in the evaluation of advanced carcinoma of the prostate is suggested, since the type of rectal disease shown on barium enema study was not clinically suspected in five of eight patients.

The prognosis is usually unfavorable because of advanced carcinoma. Survival often does not exceed several months to one year. However, one of our patients is still well after three years of hormonal therapy.

CARCINOMA of the prostate is a frequent disease in the elderly and is now the third most common cause of death from cancer in the male. Diagnosis is readily made by rectal examination and biopsy. In some cases, posterior extension to the rectum and sigmoid colon may create confusion with primary rectal carcinoma, especially in cases with predominant bowel symptoms such as large bowel obstruction. The correct diagnosis is essential, since carcinoma of the prostate with recto-sigmoid involvement indicates advanced carcinoma which is not considered a surgical lesion, but requires hormone therapy.

Eight cases are reported which emphasize the importance of barium enema examination in recognizing the diagnosis of involvement of the rectum and sigmoid by carcinoma of the prostate.

Three types of rectal involvement are described by Lazarus:[10] (1) anterior rectal mass, compressing or even occluding the rectal lumen proximally, at the level of the prostate. The rectal mucosa is normal or edematous; (2) annular rectal stricture with circumferential infiltration of the rectum or recto-sigmoid. The stenosis is usually long with irregularly scalloped margins. The mucosa is normal or edematous; and (3) anterior rectal mass with ulceration of the mucosa. Invasion of the anterior bowel mucosa may result in a fungating, ulcerated mass. This lesion simulates most

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primary rectal carcinomas. A fourth type has been added by Winter:[21] separate metastatic implant to the more proximal sigmoid.

MATERIAL

Between 1969 and 1975, 257 patients with histologically proved carcinoma of the prostate were seen at St. Luke's Hospital Center. Eight of 56 patients who had barium enema studies showed abnormalities of the rectum and signoid which could be ascribed to carcinoma of the prostate. These will be discussed in detail.

Barium enema studies are not routinely performed to evaluate pelvic extension of advanced cases of carcinoma of the prostate. Patients with a frozen pelvis secondary to carcinoma of the prostate are not included in this report, since these patients were not evaluated by barium enema studies and do not present a diagnostic challenge.

Among the eight cases reported, five had recto-sigmoid involvement when first seen at St. Luke's Hospital. Three patients (Cases 6, 5, 7) developed involvement of the recto-sigmoid, seven, ten, and 14 months after the diagnosis of carcinoma of the prostate was made. Of these three patients, two were evaluated by barium enema examination for bowel obstruction, and the third for recent constipation and rectal bleeding.

The age range of the eight patients was between 60 and 87 years, with an average age of 69. The initial symptoms were related to the urinary tract in three patients, to the bowel in one, and to both urinary tract and bowel in four (Table 1). Pelvic examination showed a hard, nodular prostate in seven patients with evidence of pelvic fixation in one (Case 2). One patient was considered to have benign prostatic hypertrophy (Case 5) and one was thought to have carcinoma of the rectum. The latter patient was admitted for rectal bleeding and had a rectal mass at 5 cm (Case 8). He also had urinary frequency and dysuria. Another patient with evidence of prostatic carcinoma on rectal examination had no urinary symptoms and was admitted for increasing constipation and small fragmented stools (Case 4). Two patients had clinical evidence of pelvic extension: edema of left leg in one patient (Case 2), right hydrocele and supra-pubic mass due to anterior extension of carcinoma of prostate in the other (Case 3). A third patient had inguinal lymphadenopathy (Case 4).

According to the criteria of Lazarus,[10] the eight abnormal barium enema studies may be classified as follows: Type I—two

Table I

Case	Age	Urinary Tract Symptoms*	Bowel Symptoms*			Intravenous Pyelography	Type of Rectal Involvement	Bone Metastases*
			Constipation	Obstruction	Bleeding			
1	69	X	X				I	X
2	85	X	X			Hydronephrosis Left non-visualization	I	X
3	72	X				Hydronephrosis	II	X
4	70		X			Left non-visualization	II	X
5	60	X		X		Hydronephrosis	III	
6	68	X				Hydronephrosis Right non-visualization	II	
7	60	X					II	X
8	64	X			X	Stenosis Left ureter	II	

* At time of first visit.

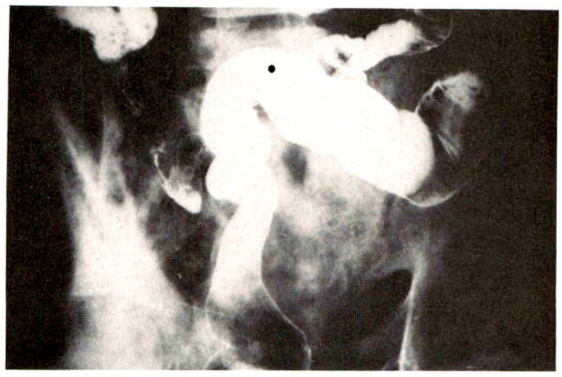

FIG. 1. Case 1. Smooth concave mass effect on left lateral aspect of proximal rectum (Type I). Anterior mass effect was also present on lateral view. Osteoblastic bone metastases.

cases, Type II—five cases, and Type III—one case.

One patient presented additional roentgenographic evidence of a subserosal metastatic implant of the proximal sigmoid (Case 5). In addition, this patient had symptoms of intermittent small bowel obstruction nine months after the diagnosis of carcinoma of the prostate was established. Barium enema examination showed involvement of the recto-sigmoid and cecum with lack of retrograde filling of the terminal ileum. Surgery for small bowel obstruction revealed metastatic involvement of the terminal ileum and cecum by carcinoma of the prostate.

Sigmoidoscopy was only performed in two patients, one Type II and one Type III. Rectal involvement was suspected in the Type III patient in that nodularity of the mucosa was observed. However, the biopsy showed normal mucosa. The second patient with a Type II barium enema examination had a normal sigmoidoscopy, but the biopsy showed malignant invasion of the mucosa.

Recto-sigmoid involvement was always associated with evidence of pelvic extension or distant metastasis. Five patients had bilateral hydronephrosis or visualization of only one kidney on intravenous pyelographic study. One patient presented with medial displacement of the ureters bilaterally associated with a left ureteral stricture at the level of the fifth lumbar transverse process.

Intravenous pyelography, cystography, or both, showed infiltration of the bladder floor in one patient (Case 6) and lateral compression and displacement of the urinary bladder in another (Case 4). A third patient presented with bullous cystitis similar in roentgenographic appearance to invasion of the urinary bladder by carcinoma of the cervix (Case 8). Four of these

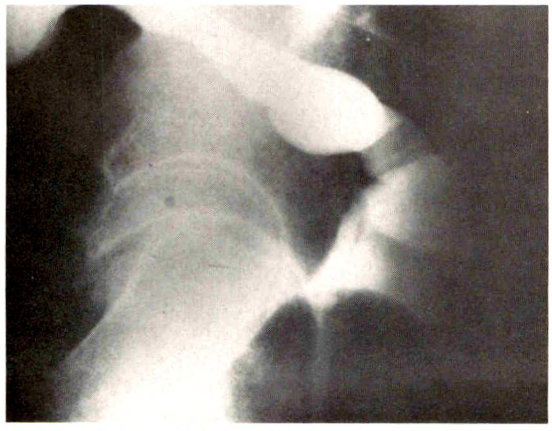

FIG. 2. Case 2. Lateral view with marked anterior compression of recto-sigmoid (Type I).

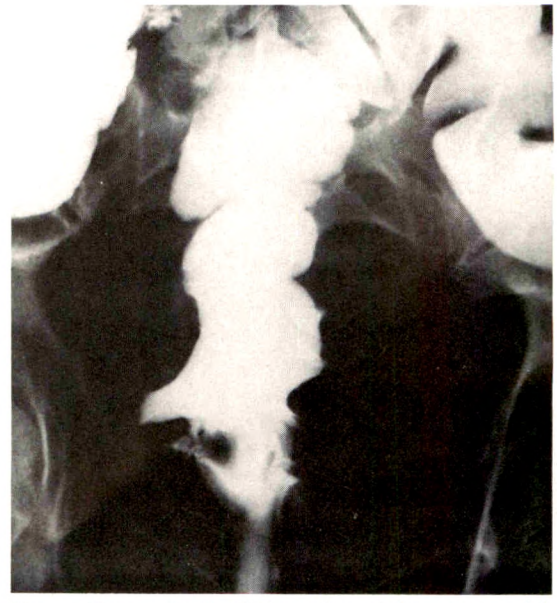

FIG. 3. Case 3. Lack of distention of the rectum with scalloped margins showing extrinsic infiltration of the bowel wall (Type II).

also had trabeculations of the bladder.

Osteoblastic bone metastases were present in three patients at the time the diagnosis of carcinoma of the prostate with recto-sigmoid involvement was established. Subsequently two other patients developed bone metastases and one patient had a liver scan consistent with metastatic disease.

DISCUSSION

The frequency of prostatic carcinoma over the age of 50 is estimated at 20–25 percent.[21] However, only a few of these patients present with an atypical appearance. Among these, posterior extension of carcinoma of the prostate may be significant enough to result in bowel symptoms. Anterior extension presenting as a hypogastric mass is less common and may simulate extension from carcinoma of the urinary bladder.

The more frequent posterior extension may be explained by the origin of the carcinoma in the posterior lobe of the prostate which frequently breaches the capsule. Denonvillier's fascia is easily involved, and carcinoma may spread between the two layers of the fascia. This results in circumferential seeding of the carcinoma around the rectum and distal sigmoid. The anterior mass effect on the recto-sigmoid is seen in more localized prostatic tumors. In most cases the metastatic spread to the rectal mucosa is prevented by the muscular layers, but it may occur and present as mucosal ulceration.

The frequency of documented recto-sigmoid involvement by carcinoma of the prostate differs depending on whether the rectal symptoms were part of the initial

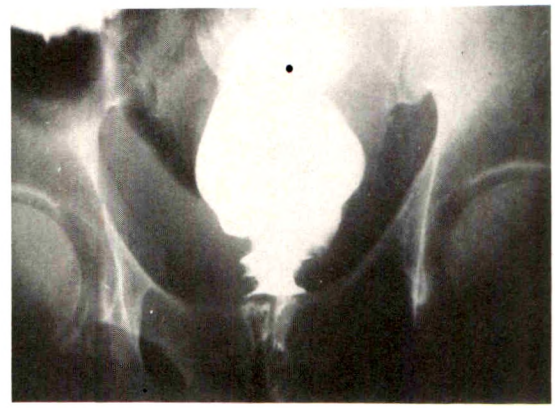

Fig. 5. Case 6. Concentric narrowing with scalloped margins of distal rectum (Type II).

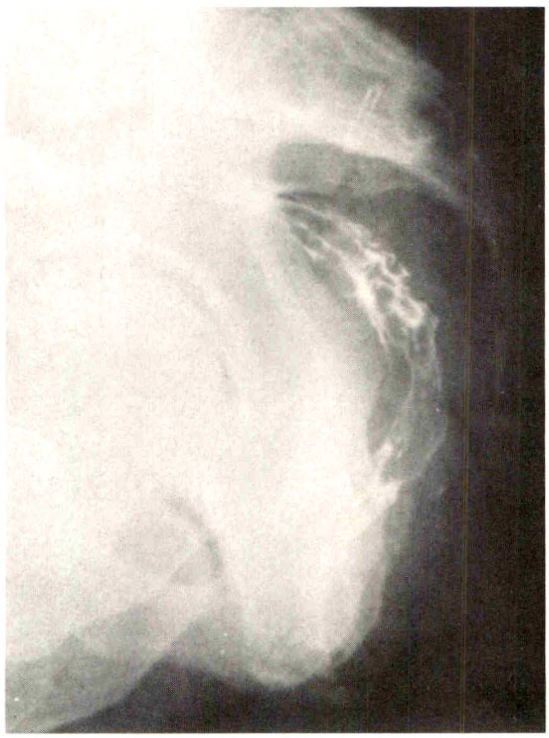

Fig. 4. Case 4. Postevacuation roentgenogram of recto-sigmoid in lateral view. Swollen nodular mucosa with anterior mass effect on proximal rectum (Type II).

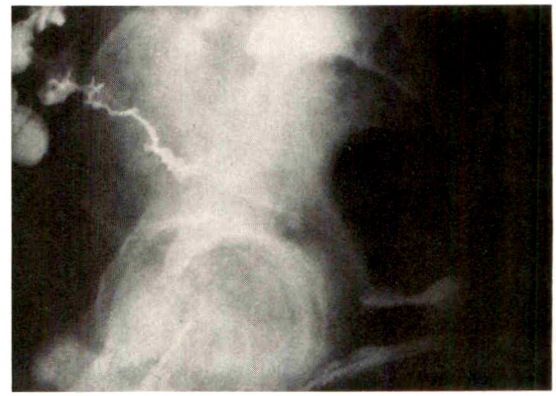

Fig. 6. Case 7. Severe narrowing of recto-sigmoid with very wide presacral space in lateral view (Type II). Osteoblastic bone metastases.

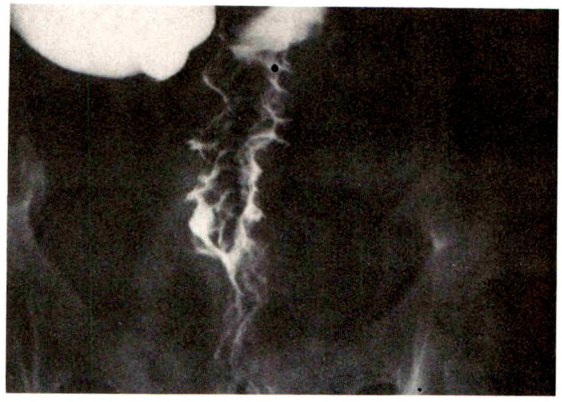

FIG. 7. Case 8. Postevacuation roentgenogram shows swollen mucosa and scalloped margins of rectum (Type II).

presenting signs or whether the rectal symptoms occurred after the diagnosis of carcinoma of the prostate had been established, since barium enema examinations are not routinely done on patients with carcinoma of the prostate. Estimates range between 1.5 percent[23] and 11.5 percent.[21] In our series the incidence is 3.1 percent which probably reflects the fact that patients were evaluated with barium enema examinations only when the differential diagnosis was difficult.

Three types of roentgenographic appearances were observed on barium enema examinations.

Type I: Anterior rectal mass (Fig. 1; and 2).

Frontal and oblique views usually show a smooth, large concave pressure defect on the left lateral aspect of the recto-sigmoid without evidence of a tumor edge. This mass effect is best demonstrated in the cross table lateral projection, with the patient prone.[3] The compression may be severe enough to result in recto-sigmoid obstruction. This appearance mimics benign prostatic hypertrophy (Fig. 10; and 11).

Type II: Annular rectal stricture (Fig. 3–7).

This is the most frequent presentation and shows circumferential, asymmetrical stricture of the proximal rectum or recto-sigmoid. Occasionally only the distal rectum is involved (Fig. 5). The margins of the stricture show irregular scalloping either by intramural tumor nodules or as a result of edema infiltrating the bowel wall (Fig. 3). The presacral space is abnormally wide. The rectal mucosa is best evaluated on postevacuation roentgenograms and air studies. Although no mucosal destruction is seen, the mucosal pattern is abnormal

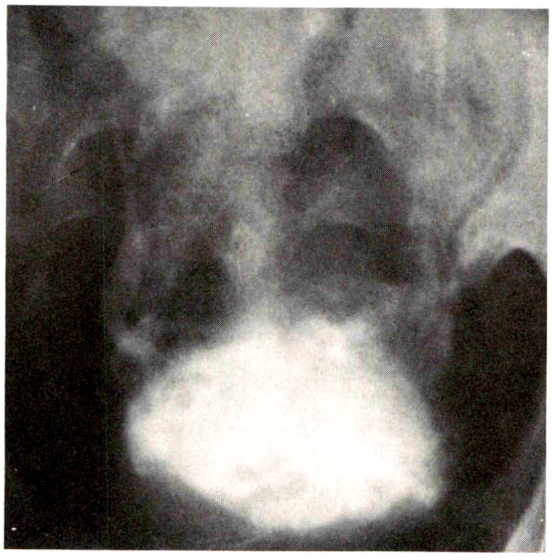

FIG. 8. Case 8. Cone-down view of bladder after intravenous pyelogram shows bullous cystitis.

FIG. 9. Case 5. Irregular narrowing of proximal rectum with destruction of the mucosa of right proximal rectal wall (Type III) (arrow). Abnormal nodular mucosal areas at rectoscopy. In addition, there is a serosal metastatic implant on proximal sigmoid (double arrow).

(Fig. 4; and 7). The edema may be so prominent as to mimic cobblestoning and may even simulate inflammatory colitis.[12] This is probably related to lymphatic and venous obstruction. In one patient there was additional roentgenographic evidence of bullous cystitis (Fig. 8), which may be explained by the same mechanism involving the bladder. In this type no ulceration of the bowel mucosa is seen.

Type III: Anterior rectal mass and ulcer (Fig. 9).

This type of involvement presents as an anterior mass with infiltration of the bowel wall. The presentation is difficult to identify by barium examination alone as the mucosal destruction is often only seen at sigmoidoscopy. Edema as seen in Type II is also present. The posterior aspect of the recto-sigmoid is usually not involved.

A fourth type of bowel involvement considered by Winter[21] is metastatic infiltration of the more proximal sigmoid which also occurs in other malignant tumors (Fig. 9, arrow).[11,12,17]

DIFFERENTIAL DIAGNOSIS

Concurrent rectal and prostatic carcinomas may occur[6,21] and have been included in Winter's[21] classification. The differential diagnosis between primary carcinoma or secondary involvement may be difficult. However, the primary carcinoma usually shows a tumor edge (Fig. 12), while secondary involvement shows tapering margins. A primary lesion is less extensive and the presacral space is usually in the normal range (Fig. 12). Evidence of advanced prostatic carcinoma as documented by infiltration of urinary bladder, hydronephrosis, and osteoblastic metastases favors secondary involvement of the rectum. A high serum acid phosphatase activity indicates extension of prostatic carcinoma beyond its capsule.

Proctoscopy with rectal biopsy confirms the diagnosis of carcinoma of the rectum in the majority of cases. However, it may be difficult to establish whether an undifferentiated carcinoma is primarily or secondarily involving the rectum. In secondary involvement of the bowel, the biopsy specimen

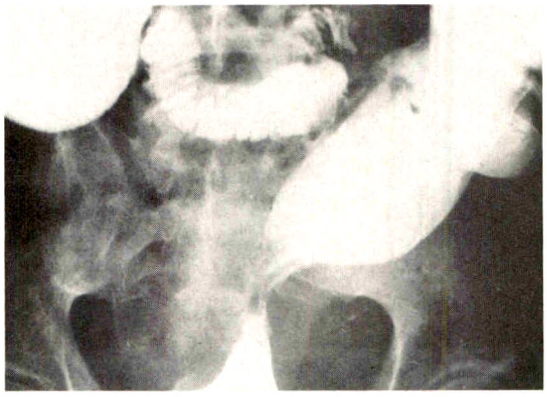

Fig. 10. Smooth compression of rectum by large benign prostatic hypertrophy. Differentiate from Type I.

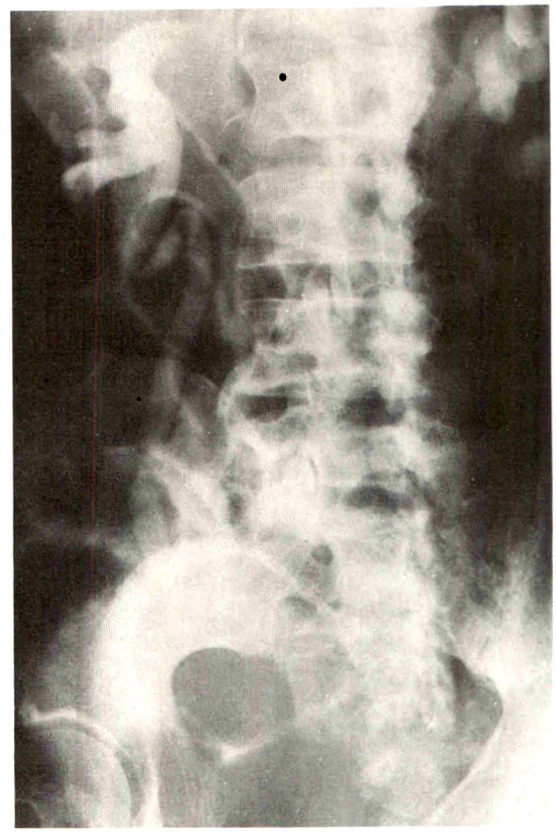

Fig. 11. Same patient as in Figure 10. Intravenous pyelogram shows giant prostate protruding into the urinary bladder, bilateral hydronephrosis (benign prostatic hypertrophy, 280 gm at surgery).

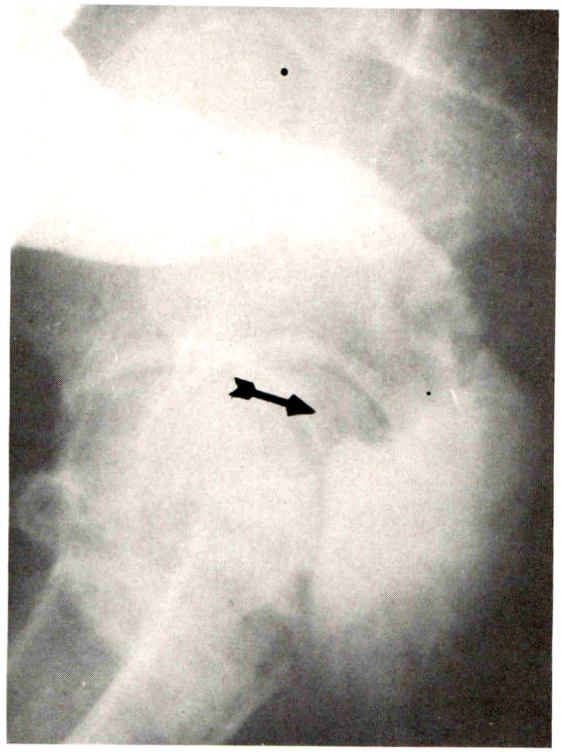

FIG. 12. Primary adenocarcinoma of rectum. Note tumor shelf at inferior aspect of mass (arrow) which is not present in secondary prostatic cancer involvement (Type II or III).

often is not diagnostic, because of extrinsic involvement of the serosal and muscular layers while the mucosa is normal. The roentgenographic findings on barium enema examination are a definite clue to the diagnosis, since secondary involvement of the rectum and recto-sigmoid has a characteristic appearance.

Beside primary rectal carcinoma, the differential diagnosis includes Blumer's shelf secondary to abdominal carcinomatosis[11,12,17] or testicular carcinoma,[19] carcinoma of the bladder and ureter with posterior extension, pelvic lipomatosis[14] in patients with smooth extrinsic compression of the recto-sigmoid, and granuloma inguinale or inflammatory colitis[12] which may simulate a Type II or III involvement. Benign prostatic hypertrophy as well as a markedly distended urinary bladder may result in anterior rectal compression and may simulate a Type I change (Fig. 10; and 11).

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REFERENCES

1. BAILEY, H. D., CHUNG, E. B., and WHITE, J. E. Prostatic malignancy presenting as rectal malignancy. *J. Nat. M. A.*, 1968, *60*, 294–296.
2. BAUM, W. C., and MCCLENNAN, R. E. Prostatic annular constricting lesion of rectum: diagnostic challenge. *Am. Surgeon*, 1955, *141*, 91–94.
3. BECKER, J. A. Prostatic carcinoma involving rectum and sigmoid colon. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *94*, 421–428.
4. DAVIES, A. L. Prostatic carcinoma presenting as acute obstruction of large bowel. *Cancer*, 1967, *20*, 1035–1037.
5. DESAI, S. E. Carcinoma of prostate: local extension following perineal needle biopsy. *Urology*, 1974, *3*, 87–88.
6. DEVAN, W. T. Metastatic (prostatic) carcinoma of rectum coexisting with primary adenocarcinoma of rectum. *Pennsylvania M. J.*, 1964, *67*, 41–44.
7. GRAVES, R. C., and MILITZER, R. E. Carcinoma of pancreas with metastases. *J. Urol.*, 1935, *33*, 235–251.
8. GUILLEMIN, P., L'HERMITE, J., PARIETTI, R., and RAVEY, M. Extensions atypiques du cancer de la prostate. *J. d'Urol. Nephrol.*, 1972, *18*, 866–871.
9. JACKMAN, R. J., and ANDERSON, J. R. Proctologic manifestations of carcinoma of prostate. *Am. J. Surg.*, 1952, *83*, 491–495.
10. LAZARUS, J. A. Complete rectal occlusion necessitating colostomy: due to carcinoma of prostate. *Am. J. Surg.*, 1935, *30*, 502–505.
11. MEYERS, M. A., and MCSWEENEY, J. Secondary neoplasms of bowel. *Radiology*, 1972, *105*, 1–11.
12. MEYERS, M. A., GLIPHANT, M., TEIXIDOR, H., H., and WEISER, P. Metastatic carcinoma simulating inflammatory colitis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1975, *123*, 74–83.
13. MIR, M., DIKRANIAN, H., and COGBILL, C. L. Carcinoma of prostate presenting as obstructive carcinoma of rectum. *Am. Surgeon*, 1973, *39*, 582–586.
14. NUSSBAUM, P. S. Carcinoma of prostate pre-

senting as pelvic lipomatosis. *Surg. Clin. North America*, 1972, *52*, 405–414.

15. Olsen, B. S., and Carlisle, R. W. Adenocarcinoma of prostate simulating primary rectal malignancy. *Cancer*, 1970, *25*, 219–222.
16. Reay, E. R., and Utley, W. L. F. Rectal manifestations of malignant prostate. *Australian & New Zealand J. Surg.*, 1955, *24*, 272–278.
17. Scheid, J. E., and Crile, G., Jr. Metastases of carcinoma of stomach to submucosa of rectum. *Am. J. Surg.*, 1952, *83*, 827–829.
18. Stein, C. A., and Herbst, W. P. Cancer of prostate simulating cancer of rectum: digital dilemma. *Med. Ann. District Columbia*, 1971, *40*, 162–164.
19. Steinbach, H. L., and Minagi, H. Endocrines. In: Atlas of Tumor Radiology. Year Book Publishers, Inc., Chicago, p. 273.
20. Stuart, J. R., and Klutz, W. S. Rectal obstruction due to carcinoma of prostate. *Rhode Island M. J.*, 1971, *54*, 211–212.
21. Winter, C. C. Prostatic carcinoma involving rectum: problem of differentiation from other malignant lesions. *California Med.*, 1955, *82*, 85–90.
22. Winter, C. C. Problem of rectal involvement by prostatic cancer. *Surg., Gynec. & Obst.*, 1957, *105*, 136–140.

THE PALEOEPIDEMIOLOGY OF POROTIC HYPEROSTOSIS IN THE AMERICAN SOUTHWEST: RADIOLOGICAL AND ECOLOGICAL CONSIDERATIONS

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CLEVELAND, OHIO AND TEMPE, ARIZONA

ABSTRACT:

Porotic hyperostosis was observed in 34 percent of 539 crania excavated from sites in Arizona and New Mexico. Common causes of this cranial pathology in the Old World (thalassemia, sickle cell anemia, and malaria) do not explain its occurrence in the American Southwest, as malaria and hemoglobinopathies are not known to have existed in the New World prior to European contact. Iron deficiency anemia which may also be associated with porotic hyperostosis occurs on a mass level only with hookworm infestation or nutritionally-related iron deficiency. Since hookworm infestation is rare in the American southwest and has not been reported in prehistoric southwestern American Indians, the hypothesis of nutritional anemia was examined. In canyon bottom sites where the diet was heavily dependent on maize, which is low in iron and also contains an inhibitor of iron absorption, significantly more crania had porotic hyperostosis than in sage plain sites, where the diet included ample animal protein rich in easily absorbable iron ($p < .001$). Furthermore, canyon bottom children, who were more susceptible to iron deficiency anemia, had a higher incidence of porotic hyperostosis lesions than adults ($p < .0001$).

THIS study describes the occurrence of porotic hyperostosis, a cranial lesion associated with hematologic abnormalities, in 539 crania excavated from sites in the American Southwest. Porotic hyperostosis (also known as symmetrical osteoporosis, cribra orbitalia, cribra cranii, or hyperostosis spongiosa) is a descriptive term for small apertures in the outer table of the orbits or cranial vault, which give a coral or sieve-like appearance. Roentgenographically, this condition is characterized by widening of the diploic spaces, thinning or obliteration of the outer table, and a radial pattern of bone spiculae in the diploe known as "hair-on-end" striations.[33,34]

Porotic hyperostosis was first described by Welcker in 1885.[35] Initial attempts to explain its pathogenesis and significance were handicapped by the absence of similar observations in circumstances where etiology could be determined.[35] The first documented cause for porotic hyperostosis was thalassemia major, described by Cooley and Lee[9] on roentgen studies in 1925. Subsequently, similar roentgen changes were found in patients with sickle cell anemia and other hemoglobinopathies.[22,30,40,42] These findings have also been noted in cases of hereditary spherocytosis and elliptocytosis,[33] pyruvate kinase deficiency,[6] polycythemia vera,[16] and cyanotic congenital heart disease.[5,36,37]

On the basis of these observations, it was then hypothesized that in Africa and the Mediterranean basin, hemolytic anemias associated with malaria and hemoglobinopathies were the cause for these cranial lesions in human remains.[3,4] However,

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porotic hyperostosis was also detected in pre-Colombian remains in the New World[19,35] where neither human malaria nor hemoglobin variants associated with hemolytic anemias are known to have existed prior to European contact.[13,20,41]

As porotic hyperostosis has been found throughout the world, relatively uncommon or geographically limited causes could not explain its distribution. In 1936, Sheldon[39] described the first case of the roentgenographic findings of porotic hyperostosis in iron deficiency anemia; its significance, however, was not appreciated until the second case was reported by Eng in 1958.[16] Since then some 35 additional children with iron deficiency anemia and typical roentgen findings have been described.[1,2,7,8,24,32,37] With confirmation that porotic hyperostosis occurs in the common problem of iron deficiency anemia, it has been hypothesized that this is a major factor in producing the skull changes.[18,33,34,37]

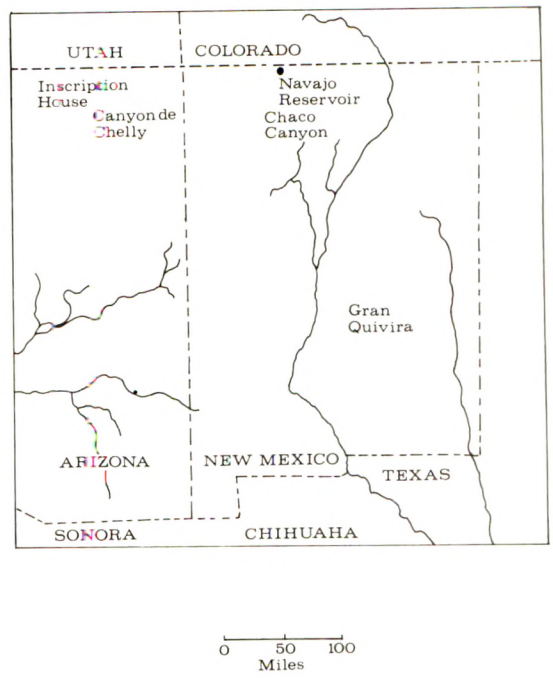

Fig. 1. Geographic distribution of the six skeletal series examined in this study.

Table I

SITE IDENTIFICATION AND ECOLOGY

Site	Cultural Period	Date A.D.	Environment	Diet
Canyon de Chelly	Basketmaker II–IV	400–700	Canyon bottom	Predominantly maize diet. Animal food sources scarce. Cactus fruits, pinon nuts, chenopods, squash, and beans occasionally found[31]
	Pueblo II–III	900–1300	Canyon bottom	
Chaco Canyon	Pueblo II–III	900–1300	Canyon bottom	Maize 75 percent of the diet, supplemented by beans, pumpkin, and animal foods[21]
Inscription House	Pueblo III	1250–1300	Canyon bottom	Predominantly maize diet supplemented by squash and gourds, prickley pear and bee weed. Beans rare[27]
Navajo Reservoir	Pueblo I–II	700–1100	Sage plains	Abundant variety of food sources (ten species of fish, 41 animal species, 52 bird species). Maize, squash, and beans also cultivated[11,12]
Gran Quivira	Pueblo IV–V	1315–1673	Sage plains	Deer, antelope, and small game animals very common. Maize, squash, and beans frequently utilized[12]

FIG. 2. Porotic hyperostosis in the orbits and frontal region.

MATERIAL

A total of 539 crania in six series of human remains were examined. All were southwestern Anasazi Indian, derived from sites within a 120 mile radius (Fig. 1). Four series are from canyon bottom sites where diet was heavily dependent on maize. Two series are from sage plain sites where diet was based on a wide variety of animal and vegetable food sources. Table I summarizes the site, cultural period, date, environment, and dietary content for each series of crania.

METHOD

All crania were inspected for the presence of small porous openings on the external surface of the orbits and cranial bones. Skulls with one or more clusters (>5 mm in diameter) of such lesions were considered to have porotic hyperostosis. Widened diploe or thinned outer tables were not considered criteria for porotic hyperostosis unless accompanied by apertures. All observations were made by one person (M. El-Najjar). The Inscription House material was additionally observed by Dennis Ryan. Aging and sexing was based on the standard techniques suggested by Krogman in 1962.[23]

RESULTS

Of the 539 crania, 200 were from children under ten years of age. Among the crania from individuals over ten years of age, 159 were male and 180, female.

Porotic hyperostosis was observed in crania from each of the series, and 34.3 percent of all skulls were affected. Lesions were noted primarily in the orbits but also occurred in the frontal and parietal bones (Fig. 2; and 3); roentgenographically, the changes are similar to those described in hemolytic states (Fig. 4, *A* and *B*). Orbital lesions, always found in both orbits, were typically localized to the lateral anterior orbital roof (Fig. 5). No lesions of porotic hyperostosis were noted on the endocranial surface or below the temporal or nuchal muscle lines.

The age and sex distribution of porotic hyperostosis in each series is summarized in Table II.

The prevalence of porotic hyperostosis in the sage plains and canyon bottom sites is compared in Table III. Porotic hyperostosis was much more common in crania from canyon bottom than from sage plains sites. In the canyon bottom crania, porotic hyperostosis was much more prevalent in children than in adults, but in the sage plains crania no age differences were observed. Sex differences were not statistically significant. Comparisons of three temporal periods at Gran Quivira and two cultural periods at Canyon de Chelly showed no statistically significant differences in the prevalence of porotic hyperostosis.

DISCUSSION

Porotic hyperostosis has been described

FIG. 3. Porotic hyperostosis in the parietal bone.

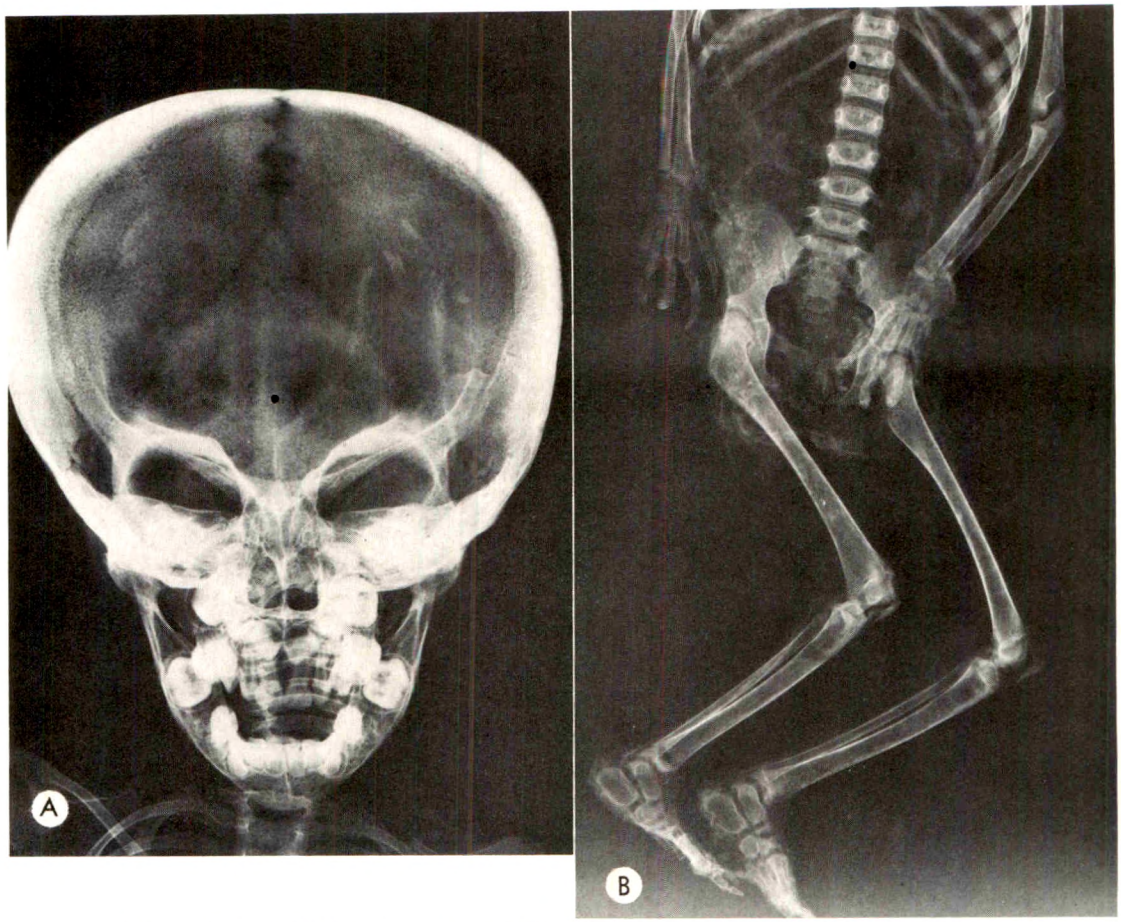

Fig. 4. (*A* and *B*) Roentgenograms of the same individual shown in Figure 2 and 3. Skeletal changes resemble those in hemolytic anemias.

only in conditions associated with erythroid hyperplasia of the bone marrow—hemolytic anemias, polycythemic states, and iron deficiency anemia. In these states the bone marrow space becomes physically larger due to both an increase in the proportion of larger, immature red cell precursors, and also an increase in the number of red cells.[43] In the skull, marrow tissue is found in the diploe throughout life. Recently, mechanisms have been suggested whereby hyperplastic marrow may produce the physical alterations of bony structure observed in porotic hyperostosis.[6,28,38] Once the skull changes have occurred, they persist for years. Although some healing may occur,[7] details of the process of repair in porotic hyperostosis have not been described.

The high frequency of porotic hyperostosis in the skeletal series from the American Southwest suggests that a condition associated with bone marrow hyperplasia must have been common. Of the established causes, iron deficiency anemia seems the most likely explanation, as malaria and hemoglobinopathies were unknown in the area and other causes for porotic hyperostosis are extremely rare. Iron deficiency anemia may occur as a result of either blood loss or nutritionally-related iron deficiency. Chronic blood loss occurring in a substantial proportion of a population is found only with hookworm infestations. Since parasitic infestations were extremely rare in southwestern American Indians,[17] a dietary cause for the postulated iron deficiency anemia must be sought.

FIG. 5. In the orbits, porotic hyperostosis is always found on the lateral anterior orbital roof.

Recent mineral analyses showed that ground water, soil, and prehistoric maize in this area were not low in iron.[14] However, the absorbable iron in food, not the total iron content, determines the dietary iron balance. The iron content of maize is relatively low, and less than five percent of this iron is absorbed;[29] the availability of iron in foods taken with maize is also decreased.[25] By contrast, animal foods both supply relatively large amounts of absorbable iron and also increase the uptake of iron from foods taken simultaneously. Thus, populations subsisting on maize diets with little animal protein are in a "critical situation in attempting to meet normal iron requirements."[26] In groups with staple diets so limited in absorbable iron, certain individuals are more likely to develop iron deficiency anemia due to their increased iron needs. Young children, because of rapid growth, and women of childbearing age, because of menstrual losses and pregnancy demands, are particularly susceptible.

If iron deficiency anemia accounts for the presence of porotic hyperostosis in these crania then: (1) porotic hyperostosis will be more common in populations with diets consisting largely of maize with little animal protein; and (2) individuals with increased iron needs will display more porotic hyperostosis when absorbable dietary iron is limited.

The findings of the present series are generally consistent with these expectations. The sage plains groups had ready access to a variety of animal foods in addition to maize; porotic hyperostosis was relatively uncommon. The canyon bottom peoples were heavily dependent on maize and porotic hyperostosis was widespread. In the canyon bottom sites where dietary constituents are known to interfere with iron absorption, porotic hyperostosis was much more common in one group with increased iron needs (*i.e.*, children).

It was also anticipated that adult females, another group with increased iron needs, would have more porotic hyperostosis than males when adsorbable dietary iron was marginal. Although females were more affected than males in canyon bottom

TABLE II

AGE AND SEX DISTRIBUTION OF POROTIC HYPEROSTOSIS (PH) IN THE SIX SKELETAL SERIES

Series	Total		Children (0–10)		Total Adults (11+)		Adult Males		Adult Females	
	n	Percent with PH	*n*	Percent with PH	*n*	Percent with PH	*n*	Percent with PH	*n*	Percent with PH
Canyon de Chelly										
a. Basketmakers	*136*	49.3	*50*	72.0	*86*	42.3	*34*	26.5	*52*	42.3
b. Pueblos	*78*	55.1	*17*	88.0	*61*	45.9	*39*	41.0	*22*	54.5
Chaco Canyon	*32*	71.8	*12*	83.3	*20*	65.0	*7*	57.1	*13*	69.2
Inscription House	*24*	54.2	*11*	63.6	*13*	46.2	*4*	50.0	*9*	44.4
Navajo Reservoir	*92*	13.0	*44*	15.9	*48*	10.4	*28*	10.7	*20*	10.0
Gran Quivira	*177*	15.3	*66*	18.2	*111*	13.5	*47*	15.0	*64*	12.5

Table III

GEOGRAPHIC, AGE AND SEX DISTRIBUTION OF THE PREVALENCE OF POROTIC HYPEROSTOSIS (PH) IN THE TWO ECOLOGICAL ZONES

	Percent	No. with PH/ Total No.	Percent	No. with PH/ Total No.	X^2	p
1. Geographic Distribution	Canyon Sites		Sage Plains Sites			
Total	54.1	146/270	14.5	39/269	93.5*	0.0001
Children	75.6	68/ 90	17.3	19/110	68.4*	0.0001
2. Age Distribution	Children		Adults			
Canyon sites	75.6	68/ 90	43.3	78/180	25.0*	0.0001
Sage plains	17.3	19/110	12.6	20/159	1.19	0.20
3. Sex Distribution	Males		Females			
Canyon sites	36.9	31/ 84	49.0	47/ 96	2.65	0.10
Sage plains	13.3	10/ 75	11.9	10/ 84	0.08	0.90

* Statistically significant findings.

sites, statistically significant differences were not observed. These adult findings may result from properties of adult bone as well as iron status. Bone marrow hyperplasia may more readily produce porotic hyperostosis in children because the bones are thin and not fully mineralized. It is therefore possible that some adult lesions are partially healed lesions of childhood. The understanding of adult findings awaits adequate description of the effect of bone maturation and repair on the lesions of porotic hyperostosis. Thus, the hypothesis that iron deficiency anemia due to lack of absorbable iron in the diet accounts for not only the occurrence of porotic hyperostosis in the American Southwest but also its geographic distribution within the region and its predominance in children in certain sites.

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We would like to thank Drs. Kenneth Warren and Gary Brittenham for their suggestions and comments in the preparation of the manuscript. We would like to thank Mrs. Patricia Ann El-Najjar for her assistance in recording the original data, Miss Deborah Vaiksnoras who prepared the map, and Miss Alice Siman who typed the final manuscript.

REFERENCES

1. Agarwal, K. N., Dhar, N., Shah, M. M. and Bhardwaj, O. P. Roentgenologic changes in iron deficiency anemia. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 635–637.
2. Aksoy, E., Kalmi, N., and Erdem, S. Roentgenographic bone changes in chronic iron deficiency anemia. *Blood*, 1966, *27*, 677–686.
3. Angel, J. L. Porotic hyperostosis, anemias, malarias and marshes in prehistoric Mediterranean. *Science*, 1966, *153*, 750–763.
4. Angel, J. L. Porotic hyperostosis or osteoporosis symmetrica. In: Diseases in Antiquity. Edited by D. Brothwell and A. T. Sandison. Charles C Thomas, Publisher, Springfield, Ill., 1967.
5. Ascenzi, A., and Marinozzi, V. Sur le crâne en bosse au cours des polyglobules secondaires à l'hypoxemie chronique. *Acta haemat.*, 1958, *19*, 253–262.
6. Becker, M., Geniesen, N., Piomelli, S., Dave, D., and Mendoza, R. Roentgenographic manifestations of pyruvate kinase deficiency hemolytic anemia. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 481–491.
7. Britton, H. A., Canby, J. P., and Mohler, C. M. Iron deficiency anemia producing evidence of marrow hyperplasia in calvarium. *Pediatrics*, 1960, *25*, 621–628.
8. Burko, H., Mellins, H. Z., and Watson, J. Skull changes in iron deficiency anemia. Am.

J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1961, *86*, 447–452.
9. COOLEY, T. B., and LEE, P. Series of cases of splenomegaly in children with anemia and peculiar bony changes. *Tr. Am. Pediat. Soc.*, 1925, *33*, 29–30.
10. D'ERAMO, N., and DE GAETANO, G. Le alterzioni ossee nelle emopatie. *Soc. Ed. Univ.*, Rome, 1957.
11. DITTERT, A. E., HESTER, S. J., and EDDY, F. W. Archaeological survey of Navajo Reservoir district, northwestern New Mexico. *Monographs of the School of American Research and the Museum of New Mexico*, 1961, *23*.
12. DITTERT, A. E. Personal communication, 1974.
13. DUNN, F. L. On antiquity of malaria in western hemisphere. *Human Biol.*, 1965, *37*, 385–393.
14. DUTT, G. R., and MCCREARY, T. W. Quality of Arizona's domestic, agricultural and industrial waters. University of Arizona, Tucson, Agricultural Experimental Station, 1970, Report 256.
15. DYKSTRA, O. H., and HALBERTSMA, T. Polycythemia vera in childhood: report of case with changes in skull. *Am. J. Dis. Child.*, 1940, *60*, 907–916.
16. ENG, L. Chronic iron deficiency anemia with bone changes resembling Cooley's anemia. *Acta haemat.*, 1958, *19*, 263–268.
17. FRY, E. Personal communication, 1974.
18. HENGEN, C. P. Cribra orbitalia; pathogenesis and probable etiology. *Homo*, 1971, *22*, 57–75.
19. HRDLICKA, A. Anthropological work in Peru in 1913, with notes on pathology of ancient Peruvians. *Smithsonian Misc. Coll.*, 1914, *61*, 1–69.
20. JARCHO, S. Some observations on disease in prehistoric North America. *Bull. Hist. Med.*, 1964, *38*, 1–19.
21. JUDD, N. M. Material culture of Pueblo Bonito. *Smithsonian Misc. Coll.*, 1954, *124*, 1–398.
22. KARSHNER, R. G. Roentgen studies of bones in certain diseases of blood and certain hematopoietic systems. AM. J. ROENTGENOL. & RAD. THERAPY, 1928, *20*, 433–439.
23. KROGMAN, W. M. The Human Skeleton in Forensic Medicine. Charles C Thomas, Publisher, Springfield, Ill., 1962.
24. LANSKOWSKY, P. Radiological features of iron deficiency anemia: *A.M.A. Am. J. Dis. Childhood*, 1968, *116*, 16–29.
25. LAYRISSE, M., COOK, J., MARTINEZ-TORRES, C., ROCHE, M., KUHN, I., WALKER, R., and FINCH, C. Food iron absorption: comparison of vegetable and animal foods. *Blood*, 1969, *33*, 430–443.
26. LAYRISSE, M., and MARTINEZ-TORRES, C. Food iron absorption: iron supplementation of food. *Progr. Haemat.*, 1971, *7*, 137–160.
27. LINDSAY, A. J., JR. The Tsegi phase of the Kayenta culture tradition in northeastern Arizona. Ph.D. dissertation, University of Arizona, Tucson, 1969.
28. MAGGIONI, G., and ASCENZI, A. Morbodi Cooley. Edited by Abruzzini. Roma, 1948.
29. MOORE, C. W. Iron. In: Modern Nutrition in Health and Disease. Fourth edition. Edited by Wohl and Goodhart. Lea & Febiger, Philadelphia, 1968.
30. MOORE, S. Bone changes in sickle cell anemia with note of similar changes observed in skulls of ancient Maya Indians. *J. Missouri Med. A.*, 1929, *26*, 561.
31. MORRIS, D. P. Personal communication.
32. MOSELEY, J. E. Patterns of bone changes in leukemia and myelosclerosis. *J. Mt. Sinai Hosp.*, 1961, *28*, 1–31.
33. MOSELEY, J. E. Paleopathologic riddle of "symmetrical osteoporosis." AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *95*, 135–142.
34. MOSELEY, J. E. Radiographic studies in hematologic bone disease; implications for paleopathology. In: Human Paleopathology. Edited by S. Jarcho. Yale University Press, New Haven, 1966.
35. NATHAN, H., and HAAS, N. Cribra orbitalia, bone condition of orbit of unknown nature. *Israel J. Med. Sc.*, 1966, *2*, 171–191.
36. NICE, C. M., DAVES, M., and WOOD, G., II. Changes in bone associated with cyanotic congenital cardiac disease. *Am. Heart J.*, 1964, *68*, 25–31.
37. POWELL, J. W., WEENS, S., and WENGER, N. K. Skull roentgenogram in iron deficiency anemia and in secondary polycythemia. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *95*, 143–147.
38. REIMANN, F., and KURAN, S. Ursache, Entstehung und Wesen des Bürstensymptoms am Schädel bei schweren Erkrankungen des Blutes, VI, Untersuchungen über die Veränderungen des Skelettsystems bei schweren Bluterkrankungen. *Virchows Arch. path. Anat.*, 1973, *358*, 173–191.
39. SHELDON, W. Anemia with bony changes in skull. *Proc. Roy. Soc. Med.*, 1936, *29*, 743.
40. SMITH, C. H. Anemias of infancy and childhood: diagnostic and therapeutic considerations. *Bull. New York Acad. Med.*, 1954, *30*, 155–183.
41. STEWART, T. D. Effect of pathology on skeletal populations. *Am. J. Phys. Anthropol.*, 1969, *30*, 443–450.
42. VOGT, F. C. and DIAMOND, C. K. Congenital anemias, roentgenologically considered. AM. J. ROENTGENOL. & RAD. THERAPY, 1930, *23*, 625–627.
43. WINTROBE, M. W. Clinical Haematology. Sixth edition. Lea & Febiger, Philadelphia, 1967.

Vol. 125, No. 4

IMPROVED ROENTGENOLOGIC DIAGNOSIS OF OSTEOMALACIA BY MICRORADIOSCOPY OF HAND BONES*

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ABSTRACT:

Microradioscopic study of metacarpals in 24 osteomalacic and 34 osteoporotic patients revealed excessive intracortical resorption (striations) in approximately 60 percent of patients suffering from osteomalacia, but in none of the osteoporotics. Phalangeal periosteal resorption was found less frequently in osteomalacia than metacarpal striations, and was absent in osteoporotics. On the other hand, quantitative evaluation of cortical thickness, percent cortical area, bone mineral mass, and density did not show any clear differences between the two study groups. It is concluded that the differential diagnosis between osteomalacia and osteoporosis may be considerably improved by microradioscopy of hand bones.

WE have previously shown that the use of industrial (fine-grain) roentgenographic films and a simple magnification procedure, microradioscopy, enabled detection of abnormal intracortical resorptive changes ("tunnels," "cutting cones")[11] in the metacarpal bones in seven of ten female patients suffering from vitamin D deficiency osteomalacia.[17] We have now examined additional cases of osteomalacia and the findings will be compared to those obtained in a group of patients suffering from severe osteoporosis. The results indicate that the study of intracortical and periosteal resorptive changes by microradioscopy is a useful adjunct to the roentgenologic differential diagnosis between osteoporosis and osteomalacia.

MATERIAL AND METHOD

From our accumulated files of over 1,500 fine-detail roentgenograms of the hands, we extracted all those with a definite or suspected diagnosis of osteomalacia and osteoporosis. For inclusion in the study, one of the following diagnostic criteria was to be satisfied.

1. For *osteomalacia:* Positive histologic diagnosis from iliac crest biopsy (eleven cases), or presence of pseudofractures (Looser's zones) in skeletal roentgenograms (ten cases), or a serum Ca×P product <23 mg percent in patients with clinical features known to be commonly associated with osteomalacia (ten cases) (Table 1), or a combination of these. Nineteen female and five male patients fulfilled these criteria after pa-

TABLE I

ASSOCIATED DIAGNOSES AND FEATURES IN OSTEOMALACIC PATIENTS

	No. of Cases
1. Positive iliac crest biopsy	11
2. Roentgenologic findings:	
(a) Looser's zones (pseudofractures)	10
(b) Vertebral compressions	9
(c) Femoral neck fractures	6
(d) Other spontaneous fractures	2
3. Clinical diagnoses:	
(a) Previous gastrectomy	7
(b) Proved intestinal malabsorption	4
(c) Chronic pancreatitis	3
(d) Long-term anticonvulsant therapy	3
(e) Cirrhosis of the liver	2
(f) Renal tubular acidosis	2
(g) Hypophosphatemia	2

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This study was supported by Medical Research Council of Canada, Grant MA 3889.

TABLE II

AVERAGE AGE, STATURE, BODY-WEIGHT, AND MUSCLE AND FAT THICKNESS OF THE FOREARM IN PATIENTS WITH OSTEOMALACIA AND OSTEOPOROSIS

	Number	Age (years)	Age (range)	Stature (cm)	Body-weight (kg)	Muscle (cm)	Fat (cm)
Female patients							
Osteomalacia	19	59.7	(34–82)	160.7(N=12)	50.0(N=13)	6.96(N=15)	0.79(N=15)
Osteoporosis	20	64.8	(41–83)	157.3(N=14)	49.2(N=14)	7.13(N=16)	0.98(N=16)
Male patients							
Osteomalacia	5	46.4	(28–66)	154.3	55.3	8.50	0.53(N=4)
Osteoporosis	14	62.1	(45–89)	171.7(N=11)	69.2(N=12)	8.68(N=12)	0.73(N=12)

N=Normal.

tients with chronic azotemic renal disease were excluded.

2. For *osteoporosis:* Finding of one or more vertebral compressions in lateral roentgenograms of the thoracic or lumbar spine without previous or recent history of known trauma, metabolic or endocrine bone disease (osteomalacia, azotemic renal disease, thyrotoxicosis, Cushing's syndrome, hyperparathyroidism) or malignancy. There were 20 female and 14 male patients with osteoporosis thus defined.

The age distribution, together with available information on body-weight, stature, and forearm muscle and subcutaneous fat thickness,[20] are given in Table II. There were no significant differences between the variables in female osteoporotics and osteomalacics. The male osteomalacic group had considerably lower values for average age, stature, body-weight and both soft tissue measurements (but because of small numbers, these differences were not analyzed statistically in the males).

The technique of fine-detail roentgenography has been previously described[19] and normal and abnormal resorptive patterns defined.[16]

Identification of patients was removed from fine-detail roentgenograms of the hands and the roentgenograms were randomly numbered for a "blind" microradioscopic reading by one author (H.E.M.). The following observations were made from the roentgenograms:

1. Metacarpal cortical striations: 0 and 1+ (normal variations) or 2+ and 3+ (abnormal).
2. Subperiosteal resorption in phalanges: 0 and 1+ (normal variation) or ++ and +++ (abnormal).
3. On the same roentgenograms, the mid-metacarpal combined cortical thickness (C_{met}) was measured with a measuring magnifier, placed directly on the roentgenogram. This permits measurements to 0.1 mm.[14] The combined cortical thickness is the difference between external width (W) and marrow cavity width (m) or $C_{met} = W - m$.
4. From measurements of W and m, the percent cortical area was now calculated:

$$\text{percent CA} = \frac{W^2 - m^2}{W^2} \times 100.^7$$

In normal populations, the percent CA has a smaller coefficient of variation than C_{met} and is therefore diagnostically more sensitive for detecting early endosteal bone loss than the other simple mid-metacarpal measurements.[7,21,24]

Roentgen-ray photodensitometric studies of bone mineral content in the proximal radial shaft[15] had been performed in the majority of the patients. Of the three parameters, the bone mineral mass (M in g/cm²) and the combined cortical thickness (C in cm) are direct measurements in one

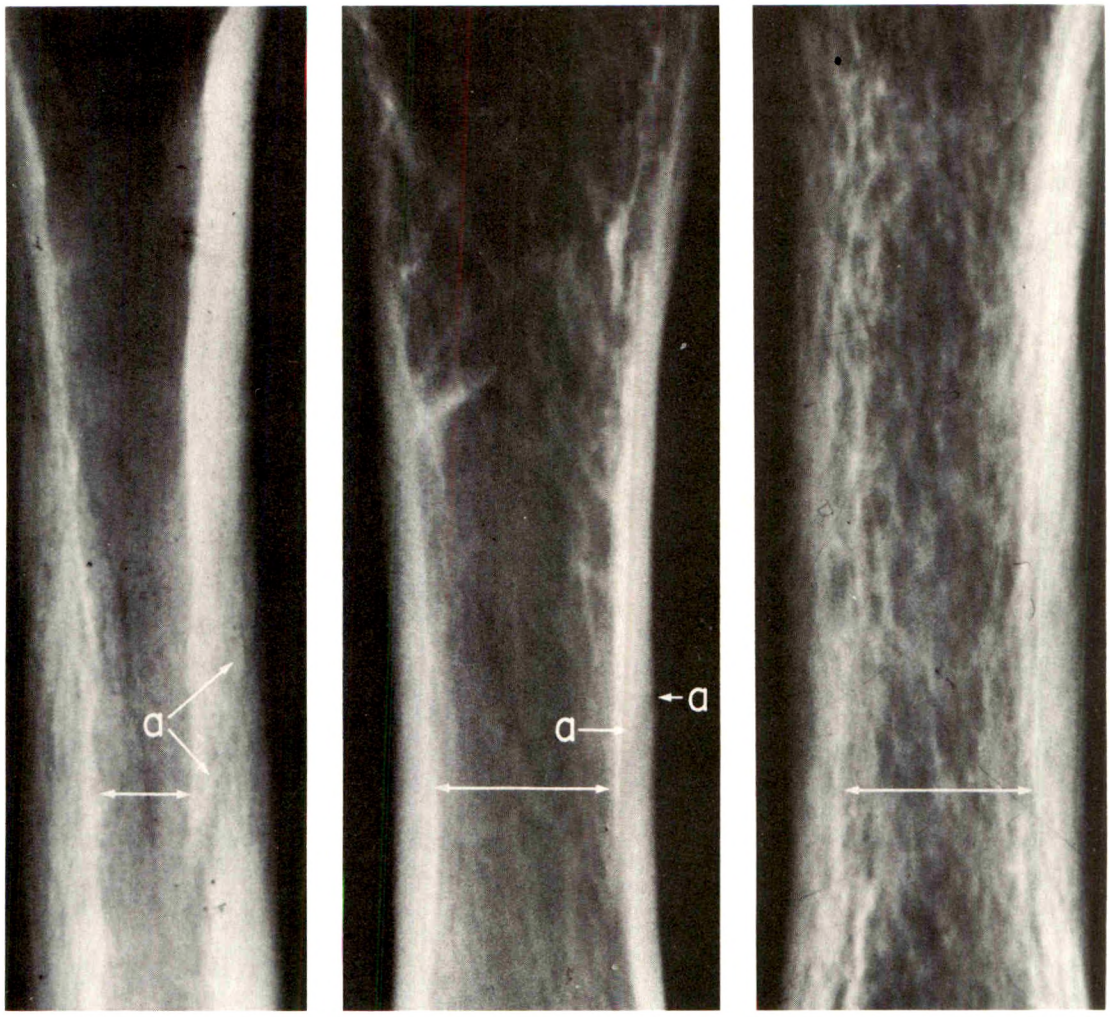

Fig. 1. Magnified roentgenograms of metacarpals from three female patients (original magnification ×7, reduced 30 percent for reproduction). *Left:* 39 years, osteomalacia. *Middle:* 74 years, osteoporosis. *Right:* 69 years, osteomalacia. Endosteal resorption in the two older patients has resulted in the enlargement of the marrow cavity width (arrows) but intracortical resorption (longitudinal striations) is present only in the osteomalacic patients. Arrows at "a" point at the canal of the nutrient artery.

centimeter longitudinal scan-area, while the bone mineral density (*i.e.*, the amount of bone mineral per unit volume of cortical bone in g/cm³) is the ratio M/C.

RESULTS

The most striking difference in findings between the two study groups was that by microradioscopy abnormal metacarpal cortical striations were detected in twelve of 19 osteomalacic women (63 percent) and in two of five osteomalacic men, while no excessive striations were recorded in any of the patients suffering from severe osteoporosis (Fig. 1). Similarly, abnormal periosteal resorption ("subperiosteal resorption") was found only in the osteomalacic patients, although in only three females and two males. In the females, subperiosteal resorption was found only in those who also had abnormal metacarpal cortical striations. In one male patient, however, the abnormal periosteal resorption in the phalanges was present without metacarpal striations. Thus, the combined number of osteomalacic men with distinctly increased net intracortical

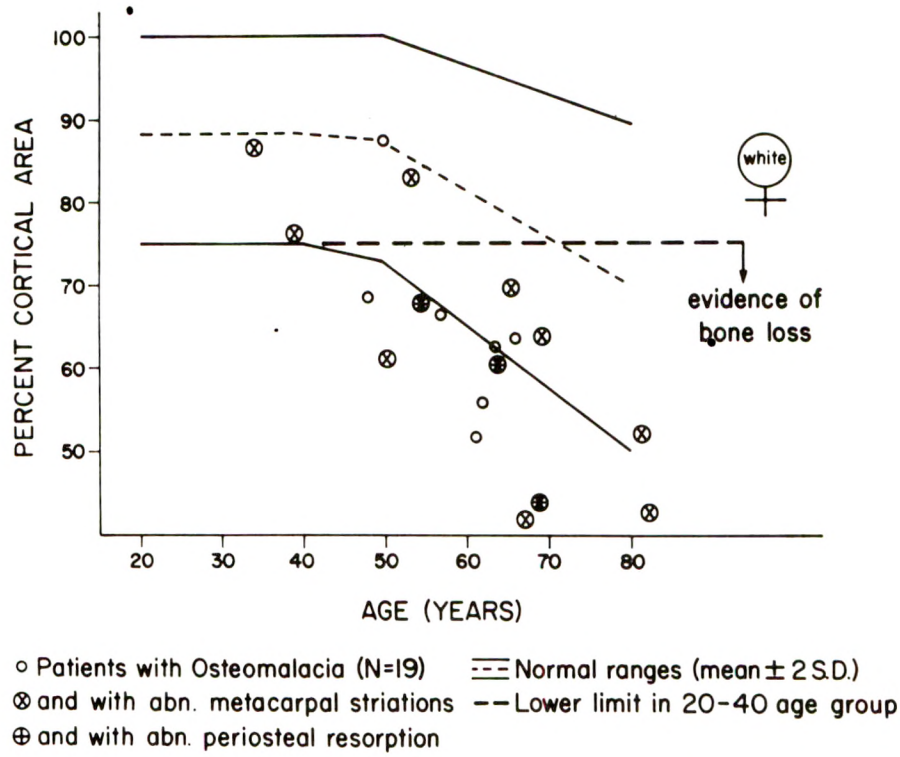

FIG. 2. Percent cortical area of the second metacarpal in female osteomalacic patients (in relation to normal ranges).

(=striations) or periosteal resorption in hand bones was three, the proportion 3:5 of the resorptive abnormalities being similar to that found by microradioscopy for the female patients.

Of ten patients who showed Looser's zones in their skeletal surveys, seven had abnormally increased metacarpal striations. On the other hand, there were seven patients with increased metacarpal striations without evidence of Looser's zones. Thus, by combining cases with Looser's zones and those with increased metacarpal striations, and considering these separately as signs of osteomalacia, a roentgenologic diagnosis of osteomalacia would have been made in 17 patients (71 percent).

An estimate of the increased net endosteal resorption in the metacarpals may be obtained by measuring either the marrow cavity width (enlarges) or the combined cortical thickness (diminishes), or one of the several other calculated metacarpal parameters (diminish).[7,21] Although the simple measurements are in principle preferable, it has been previously suggested[7,21,24] that diagnostically the most sensitive parameter for assessment of endosteal bone loss in the metacarpal is the percent cortical area (percent CA). The percent cortical area for osteomalacic (Fig. 2) and osteoporotic (Fig. 3) women, and for all male patients (in relation to age-related normal ranges according to Garn)[7] (Fig. 4) are shown.

It is seen from Figure 2 that endosteal bone loss in excess of that due to aging is demonstrable by percent CA in eleven of 19 osteomalacic women. Of the eight patients whose values were within normal ranges, six had abnormal intracortical striations. With only one exception (a 50 year old woman with renal tubular acidosis, a normal Ca×P product, but histologic diag-

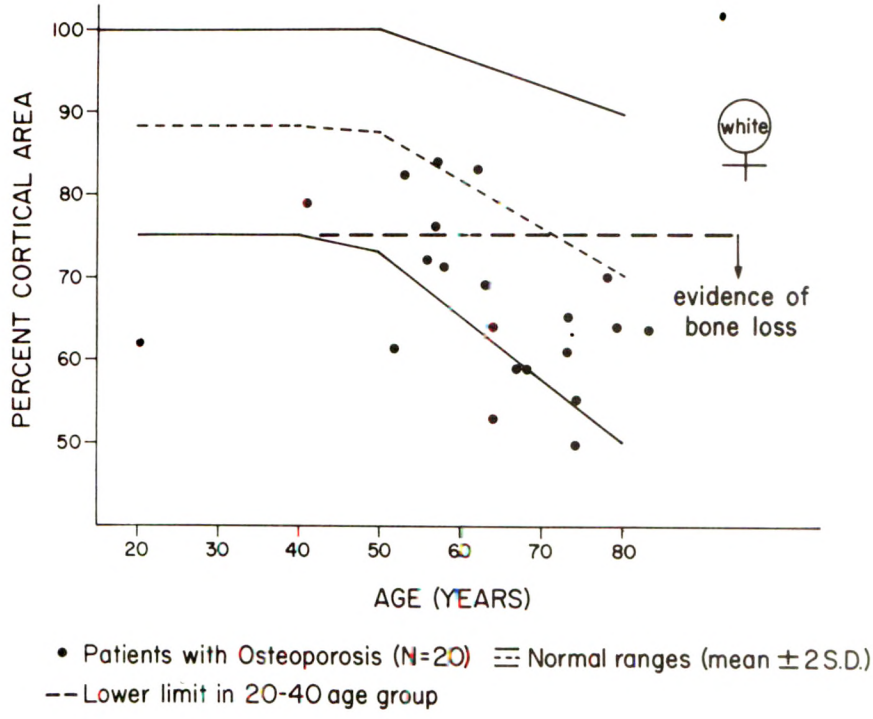

Fig. 3. Percent cortical area of the second metacarpal in female osteoporotic patients (in relation to normal ranges).

nosis of osteomalacia) either intracortical or endosteal bone loss was clearly demonstrable from industrial hand roentgenograms in these patients.

Figure 3 shows the percent CA in women with severe osteoporosis. Only 25 percent of these showed distinctly abnormal values although, with two exceptions, the values were below the age-related mean values.

Figure 4 shows the percent CA for osteomalacic and osteoporotic men in relation to normal ranges. Of the five osteomalacic patients, two showed no evidence of endosteal bone loss, but both of these had abnormally increased metacarpal cortical striations.

For comparison of the diagnostic sensitivity of percent CA *vs.* C_{met}, we calculated the percentages of those osteomalacics and osteoporotics whose values fell below the lower age-related normal limits. Excessive bone loss was thus demonstrable by C_{met} in 46 percent of osteomalacic and in nine percent of osteoporotic patients, and by percent CA in 58 percent of osteomalacics and in 32 percent of osteoporotics. Thus percent CA was more sensitive than C_{met} in diagnosing endosteal bone loss in both conditions, and both parameters revealed greater endosteal bone loss in osteomalacic than in osteoporotic patients.

The results of photodensitometric bone mineral measurements in the proximal radius for females are shown in Figure 5; and 6. It is seen that the distribution of values in relation to normal ranges (either by normal young standards or age-related) are similar to those obtained with percent CA for both osteomalacic and osteoporotic patients (Fig. 2; and 3).

DISCUSSION

The most important single finding of this study is that microradioscopy of hand bones revealed abnormal intracortical resorptive patterns in approximately 60 per-

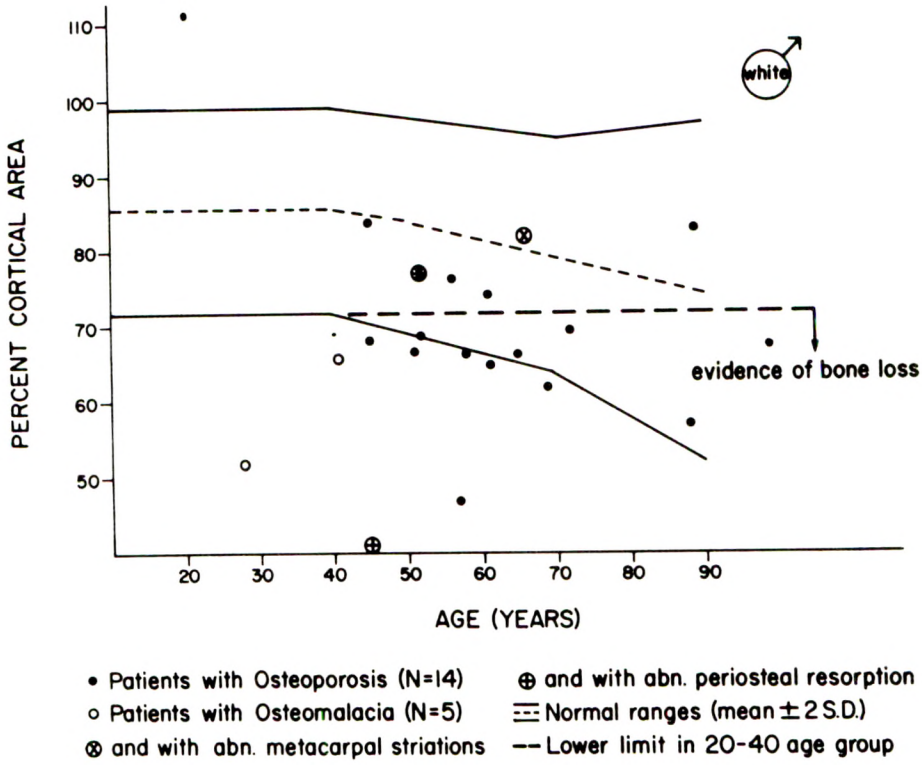

FIG. 4. Percent cortical area of the second metacarpal in male osteomalacic and osteoporotic patients (in relation to normal ranges).

cent of the patients suffering from osteomalacia, but in none of those with severe involutional (postmenopausal, senile) osteoporosis. On the other hand, photodensitometric bone mineral studies in the proximal radius failed to separate these two patient populations, although the values tended to be generally low (Fig. 5; and 6). Furthermore, simple morphometric measurements at the second metacarpal midshaft revealed patterns of bone loss similar to those obtained by photodensitometric measurements of the bone mineral mass in the radius (Fig. 2; and 3).

Thus, at least for the differential diagnosis between osteomalacia and osteoporosis, the rather complex and often costly roentgenologic measurements of bone mineral content[15,27] can be replaced by the simpler and less costly fine-detail hand roentgenography, suitable for microradioscopic detection of subtle resorptive changes as well as for accurate morphometric measurements.[14] The tabulated normally occurring variations of mid-metacarpal combined cortical thickness (C_{met}) are now available not only from the original sources,[3,7,30] but also from the widely used "Atlas of Roentgenographic Measurements" by Lusted and Keats,[13] and from the recent monograph of Steinbach *et al.*[28] A value of the C_{met} below the normal age-related values (-2 S.D. of the mean) is indicative of pathologic bone loss and should lead to a thorough metabolic investigation.

However, when the C_{met} falls into the lower normal ranges, excessive endosteal resorption may still be diagnosed by calculating the percent cortical area (percent CA) and relating this to its normal ranges.[7] The suggested better diagnostic sensitivity

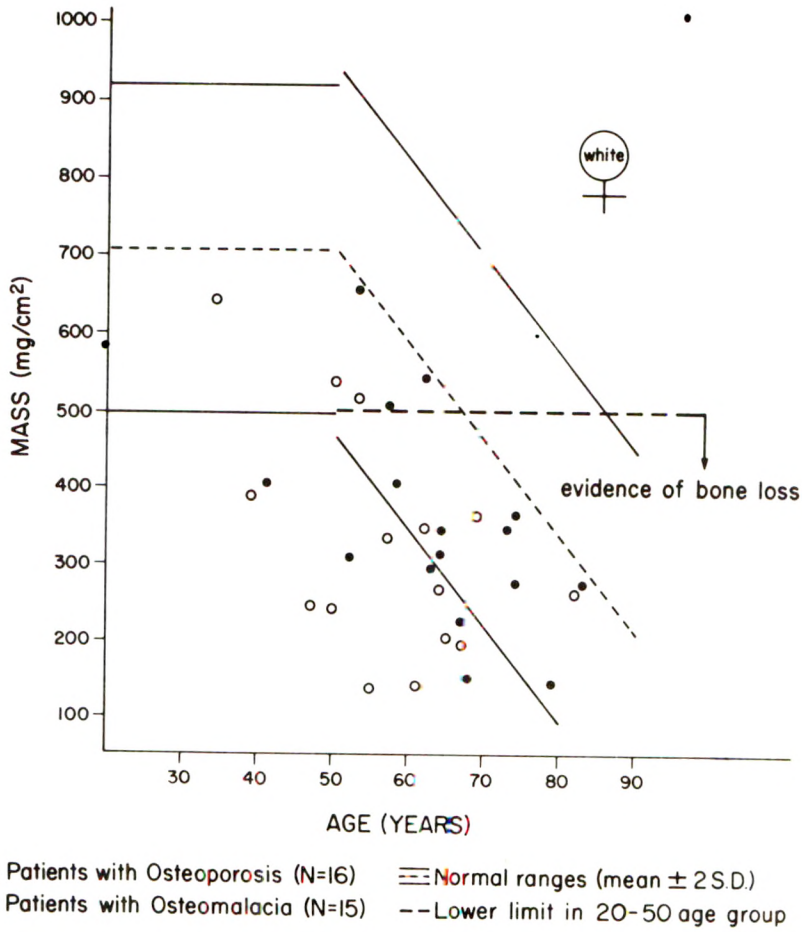

Fig. 5. Roentgen-ray photodensitometric measurements of bone mineral mass in the proximal radial shaft in female osteomalacic and osteoporotic patients (in relation to normal ranges).

of percent CA over C_{met} in the diagnosis of endosteal bone resorption[7,23] has been confirmed in this study for both osteoporosis and osteomalacia. While previously calculation of the percent CA would have been considered as being too time-consuming and tedious in routine roentgenologic practice, the availability of low-cost electronic calculators has removed this obstacle, and determination of percent CA should now be an integral part in the assessment of bone loss in metabolic bone diseases. The comparison with normal ranges of percent CA (and of C_{met}) may be further facilitated by preparing graphs (such as those of Figure 3; and 4) for routine use.

We have recently examined the diagnostic sensitivity of various cortical measurements in both second metacarpal and proximal radius and found that the single most sensitive parameter for diagnosing endosteal bone loss in osteoporosis[21] and osteomalacia[17] is the minimum combined cortical thickness of the proximal radial shaft, at least in women. Thus, when both C_{met} and percent CA fall into the lower normal ranges, it may still be worth while taking a roentgenogram of the proximal radius for measurement of the minimum combined cortical thickness.[21]

Our present findings should be viewed against the background of modern concepts of bone remodelling and the present knowledge of pathophysiology of bone.

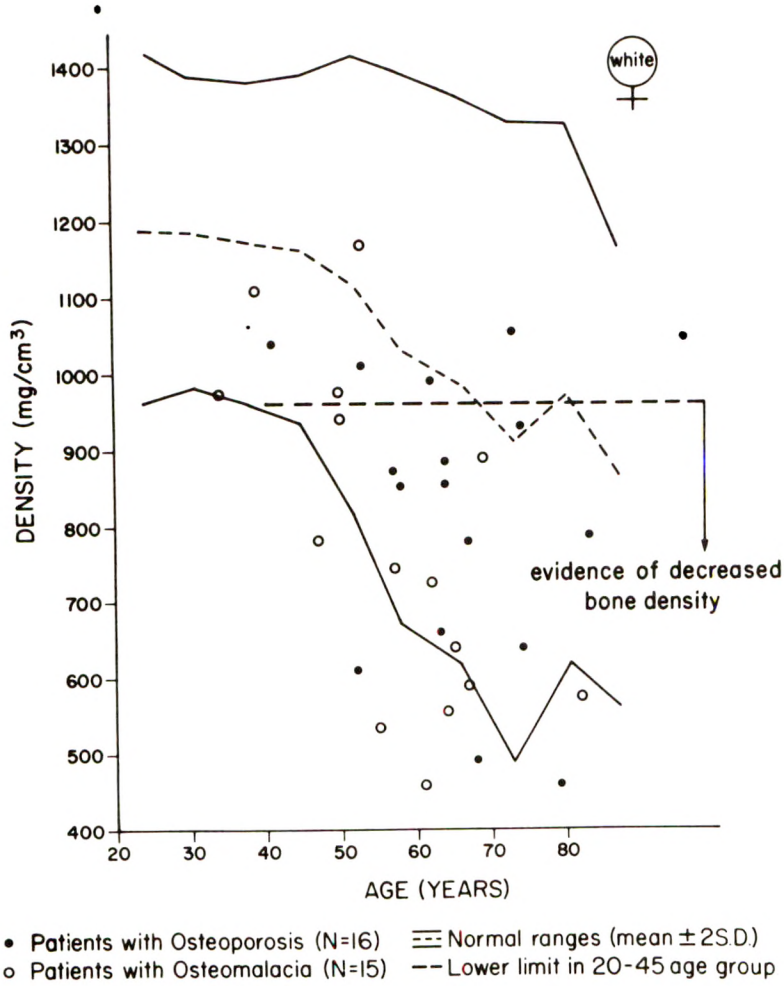

FIG. 6. Roentgen-ray photodensitometric measurements of cortical bone mineral density in the proximal radial shaft in female osteomalacic and osteoporotic patients (in relation to normal ranges).

According to the triple-envelope concept of bone remodelling dynamics,[6] bone resorption normally tends to predominate in the endosteal zone (envelope), there is balanced remodelling in the intracortical (haversian) bone, while bone formation predominates at the periosteal envelope. In conditions with high bone turnover, bone remodelling processes are activated in all three envelopes, but relatively more so in the normally quiescent haversian bone,[29] where increased numbers of resorptive foci appear as osteoclastic "cutting cones" or tunnels. These have an essentially longitudinal orientation in the cortical bone, measure a few millimeters to centimeters in length, and are approximately 200 to 500 microns in diameter,[11] *i.e.*, have physical characteristics similar to the metacarpal striations shown by microradioscopy in osteomalacia (Fig. 1; and 7).

One of the conditions with high bone turnover is hyperparathyroidism. At least some degree of secondary hyperparathyroidism probably always accompanies vitamin D deficiency osteomalacia,[10] the low serum calcium stimulating parathyroid glands to hypertrophy.[1,4] The increased parathyroid hormone output is apparently

designed to promote bone resorption and intestinal absorption of calcium and to decrease renal tubular reabsorption of calcium in order to raise the inherently low serum calcium in osteomalacia.[26]

Increased intracortical resorption in osteomalacia has previously been observed in the histologic studies of Frost[5] and Ramser *et al.*[25] and in microradiographic studies of Heuck,[9] but no previous roentgenologic studies have demonstrated this abnormality *in vivo*, probably because the intracortical striations are not distinctly seen in ordinary (regular-grain) roentgenograms and without magnification.

Although subperiosteal resorption is nearly pathognomonic for hyperparathyroidism, and its recognition greatly improved by microradioscopy,[8,18,31] in this series it was detected only in 20 percent of the cases as opposed to the 60 percent incidence of abnormal intracortical resorption. This relationship is the reverse of that observed in secondary hyperparathyroidism of azotemic chronic renal disease, where subperiosteal resorption was found in 53 percent, and abnormal intracortical resorption only in 16 percent.[16] On the other hand, microradioscopic studies by Genant and associates[8] of primary hyperparathyroidism have shown more frequent occurrence of intracortical than subperiosteal bone resorption.

Endosteal bone resorption, on the other hand, occurs commonly in all types of metabolic and endocrine bone diseases, thus being rather non-specific and of little value in the differential diagnosis. However, in conditions with low bone turnover, *e.g.*, involutional osteoporosis, bone loss occurs mainly in the endosteal and immediately subendosteal zone,[12,29] the central and peripheral cortical areas remaining relatively solid. Thus, while cortices are usually thinner than normal in both osteoporosis and osteomalacia (Fig. 2–4), in osteoporotics the central and outer portions of the cortex appear solid on microradioscopic inspection while in osteomalacia a striated pattern is found throughout most

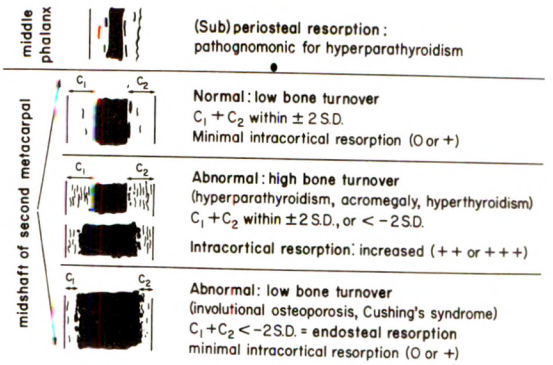

FIG. 7. Schematic presentation of microradioscopic (periosteal and intracortical resorption) and morphometric (endosteal resorption) findings in some conditions with high *vs.* low bone turnover.

C_1+C_2 is the combined cortical thickness, and ± 2 S.D. represents the normal ranges in the 20–40 year age group.

of the cortical thickness (Fig. 1; and 7).

The generally low bone mineral density of cortical bone (*i.e.*, the amount of bone mineral per volume of cortical bone) found in osteoporotic patients (Fig. 6) is partly explained by subendosteal resorption cavities or tunnels. Another factor contributing to this low bone mineral density in osteoporosis is the increased size of haversian canals throughout the cortex.[12] This type of intracortical porosity, as opposed to the osteoclastic tunneling in conditions with high bone turnover, is not distinctly visible on microradioscopy, the diameter of the enlarged haversian canals in the central and peripheral cortical zones usually not exceeding 100 microns. Finally, the fraction of poorly mineralized bone in older (and osteoporotic) patients is greater than that in young normal persons.[12]

Although Looser's zones are nearly pathognomonic for osteomalacia, they may occur relatively late in the course of the disease.[10] In the present study, approximately 40 percent of the patients had Looser's zones, but 60 percent had increased metacarpal striations. When both signs were combined and considered separately as signs of osteomalacia, the incidence of roentgenologically diagnosed osteomalacia in this series of 24 patients would be 71 percent.

The suggestion that trabecular bone "fuzziness" is a good roentgenologic sign of osteomalacia[2] has not been confirmed by us,[17] and is usually considered too subjective and unreliable.[22] On the other hand, vertebrae sometimes appear disproportionately dense in relation to the markedly thinned cortices of the peripheral bones in osteomalacia, while no such discrepancy is found in severe osteoporosis.[23]

Since involutional osteoporosis is very common after age 55 to 60, especially in women, there is a tendency among the radiologists to label patients with obvious generalized bone loss as osteoporotics. While they will be correct in a large majority of cases statistically, this practice is unfortunate since it suggests to the clinician a more-or-less normal aging phenomenon, and implies that further laboratory and other studies are probably superfluous. In these situations, when the rather non-specific symptoms of early osteomalacia continue in spite of reassurance and supportive treatment, the patient may eventually be labelled as "neurotic," "rheumatic," etc., until, after some years of suffering, a correct diagnosis of osteomalacia is finally made, perhaps following a vertebral collapse, femoral neck fracture, or discovery of a Looser's zone later in the course of the disease.

It is therefore suggested that in all cases of bone pain, muscular aches and weakness, and of course in all those suspected to have some metabolic or endocrine condition affecting bone, a posteroanterior roentgenogram of both hands be taken on industrial (fine-grain) film and inspected at 6–8× magnification by the simple technique of microradioscopy. Although increased intracortical striations are found in other conditions with high bone turnover, (thyrotoxicosis, acromegaly, chronic renal disease), the primary disease here is usually obvious clinically. A combination of a low Ca×P product (<23 mg percent) together with abnormal metacarpal striations is nearly pathognomonic for osteomalacia, and bone biopsy may then not be absolutely necessary to establish this diagnosis. Instead, trial therapy with vitamin D and calcium may alleviate the symptoms of osteomalacia quickly, and when a recheck microradioscopy of hand bones, a few months later, shows that metacarpal striations or subperiosteal resorption or both, are healing, and the Ca×P product is normalizing, the diagnosis of osteomalacia may then be considered as having been confirmed.

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REFERENCES

1. BARTOS, H. R., and HENNEMANN, P. H. Parathyroid hyperplasia in osteomalacia. *J. Clin. Endocrinol.*, 1965, *25*, 1522–1523.
2. CALENOFF, L., and NORFRAY, J. Magnification digital roentgenography: method for evaluating renal osteodystrophy in hemodialyzed patients. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *118*, 282–292.
3. DEQUEKER, J. Bone Loss in Normal and Pathological Conditions. Leuven University Press, 1972, p. 16.
4. FOURMAN, P., and ROYER, P. Calcium Metabolism and the Bone. Blackwell Scientific Publications, Oxford, 1968, p. 285.
5. FROST, H. M. The Bone Dynamics of Osteoporosis and Osteomalacia. Charles C Thomas, Publisher, Springfield, Ill., 1966.
6. FROST, H. M. Bone Remodelling and Its Relationship to Metabolic Bone Diseases. Charles C Thomas, Publisher, Springfield, Ill., 1973.
7. GARN, S. M. The Earlier Gain and the Later Loss of Cortical Bone in Nutritional Perspective. Charles C Thomas, Publisher, Springfield, Ill., 1970.
8. GENANT, H. K., HECK, L. L., LANZL, L. H., *et al.* Primary hyperparathyroidism. *Radiology*, 1973, *109*, 513–524.
9. HUECK, F. Der Knochen bei gastrointestinalen Erkrankungen. In: Aktuelle Gastroenterologie. Edited by H. Bartelheimer and N. Heisig. Georg Thieme Verlag, Stuttgart, 1968, pp. 174–190.
10. JAWORSKI, Z. F. G. Pathophysiology, diagnosis and treatment of osteomalacia. *Orthop. Clin. N. Am.*, 1972, *3*, 623–652.

11. Johnson, L. C. Morphologic analysis in pathology: kinetics and general biology of bone. In: Bone Biodynamics. Edited by H. M. Frost. J. & A. Churchill, Ltd., London, 1964, pp. 543–654.
12. Jowsey, J. Age changes in human bone. *Clin. Orthop.*, 1960, *17*, 210–218.
13. Lusted, L. B., and Keats, T. E. Atlas of Roentgenographic Measurements. Year Book Medical Publishers, Chicago, 1972, pp. 138–140.
14. Meema, H. E. Combined use of morphometric and microradioscopic methods in diagnosis of metabolic bone disease. *Der Radiologe*, 1973, *13*, 111–116.
15. Meema, H. E., Harris, C. K., and Porrett, R. E. Method for determination of bone-salt content of cortical bone. *Radiology*, 1964, *82*, 986–997.
16. Meema, H. E., and Meema, S. Comparison of microradioscopic and morphometric findings in hand bones with densitometric findings in proximal radius in thyrotoxicosis and in renal osteodystrophy. *Invest. Radiol.*, 1972, *7*, 88–96.
17. Meema, H. E., and Meema, S. Intracortical porosity in osteomalacia. In: Bone Morphometry. Proceedings of the First Workshop on Bone Morphometry, Ottawa, Ontario, March 28–31, 1973. Edited by Z. F. G. Jaworski. To be published.
18. Meema, H. E., Rabinovich, S., Meema, S., Lloyd, G. J., and Oreopoulos, D. G. Improved radiological diagnosis of azotemic osteodystrophy. *Radiology*, 1972, *102*, 1–10.
19. Meema, H. E., and Schatz, D. L. Simple radiologic demonstration of cortical bone loss in thyrotoxicosis. *Radiology*, 1970, *97*, 9–15.
20. Meema, S., Reid, D. B. W., and Meema, H. E. Age trends of bone mineral mass, muscle width, and subcutaneous fat in normal and osteoporotics. *Calc. Tiss. Res.*, 1973, *12*, 101–112.
21. Meema, S., and Meema, H. E. Improved recognition of bone loss by concurrent morphometric measurements in second metacarpal and radius. In: Bone Morphometry. Proceedings of the First Workshop on Bone Morphometry, Ottawa, Ontario, March 28–31, 1973. Edited by Z. F. G. Jaworski. To be published.
22. Morgan, B. Osteomalacia, Renal Osteodystrophy and Osteoporosis. Charles C Thomas, Publisher, Springfield, Ill., 1973, pp. 75–76.
23. Nordin, B. E. C. Metabolic Bone and Stone Disease. J. & A. Churchill Ltd., London, E. & S. Livingstone, Ltd., Edinburgh, 1973.
24. Nordin, B. E. C., Young, M. M., Bulusu, L., *et al.* Osteoporosis reexamined. In: Osteoporosis. Edited by U. S. Barzel. Grune & Stratton, Inc., New York, 1970, pp. 47–67.
25. Ramser, R., Villanueva, A. R., and Frost, H. M. Cortical bone dynamics in osteomalacia, measured by tetracycline bone-labeling. *Clin. Orthop.*, 1966, *49*, 89–102.
26. Rasmussen, H., and Bordier, P. The Physiological and Cellular Basis of Metabolic Bone Disease. Williams & Wilkins Company, Baltimore, 1974.
27. Sorenson, J. A., and Cameron, J. R. Reliable *in vivo* measurement of bone mineral content. *J. Bone & Joint Surg.*, 1967, *49-A*, 481–497.
28. Steinbach, H. L., Gold, R. H., and Preger, L. Roentgen Appearance of the Hand in Diffuse Disease. Year Book Medical Publishers, Inc., Chicago, 1975, p. 321.
29. Villanueva, A. R., Ilnicki, L., Duncan, H., *et al.* Bone and cell dynamics in osteoporoses: review of measurements by tetracycline bone-labeling. *Clin. Orthop.*, 1966, *49*, 135–150.
30. Virtama, P., and Helelä, T. Radiographic measurements of cortical bone. *Acta radiol.*, 1969, Suppl. 293.
31. Weiss, A. Incidence of subperiosteal resorption in hyperparathyroidism studied by fine detail bone radiography. *Clin. Radiol.*, 1974, *25*, 272–276.

THE WEISSENBACHER-ZWEYMULLER SYNDROME OF MICROGNATHIA AND RHIZOMELIC CHONDRODYSPLASIA AT BIRTH WITH SUBSEQUENT NORMAL GROWTH*

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ABSTRACT:

Two patients are reported with the clinical and roentgenographic findings of the Weissenbacher-Zweymuller syndrome. The features are neonatal micrognathia (Pierre Robin syndrome), rhizomelic chondrodysplasia with dumbbell shaped femur and humeri. The bone changes tend to regress and there is normal growth on serial studies.

A third case seemed at birth to show similar roentgenographic changes but was dwarfed; serial observations revealed him to be a case of the Kniest syndrome with micrognathia as an added feature.

The term "micrognathic dwarfism" suggested by Maroteaux is probably best avoided since it will inevitably include a variety of conditions, only some of which are associated with dwarfism.

IN 1964 Weissenbacher and Zweymuller[9] reported the "simultaneous occurrence of Pierre Robin syndrome and fetal chondrodysplasia" in a newborn male. The patient clinically was felt to have rhizomelic proportions, and the diagnosis of achondroplasia was suspected. The roentgenograms of their patient actually showed bulbous enlargement of the proximal and distal femurs and proximal humeri with spinal changes, none of which resembled achondroplasia. The coexistence of chondrodysplasia and micrognathia were next noted by Maroteaux *et al.*[4] in 1970; referring to Weissenbacher and Zweymuller's case, they reported three patients and suggested the term "micrognathic dwarfism."

In this communication two patients are presented whose clinical and roentgenographic findings in the first months of life parallel those noted by Weissenbacher and Zweymuller. Neither of the patients, however, was dwarfed at birth and serial observations in the first case showed normal growth despite the clinical impression of rhizomelic proportions. Weissenbacher and Zweymuller's patient had also grown in normal fashion.[10]

A third patient is included who at birth seemed to combine rhizomelic proportions and micrognathia; however, this patient has shown dwarfed growth and has roentgenographic findings now compatible with the Kniest syndrome.[3]

The cases reflect the spectrum of growth disturbances (with and without dwarfism) that can be associated with micrognathia and the need for serial observations with clinical measurements in prediction of growth and in classification for purposes of genetic advice.

REPORT OF CASES

CASE 1. This white girl weighed seven pounds,

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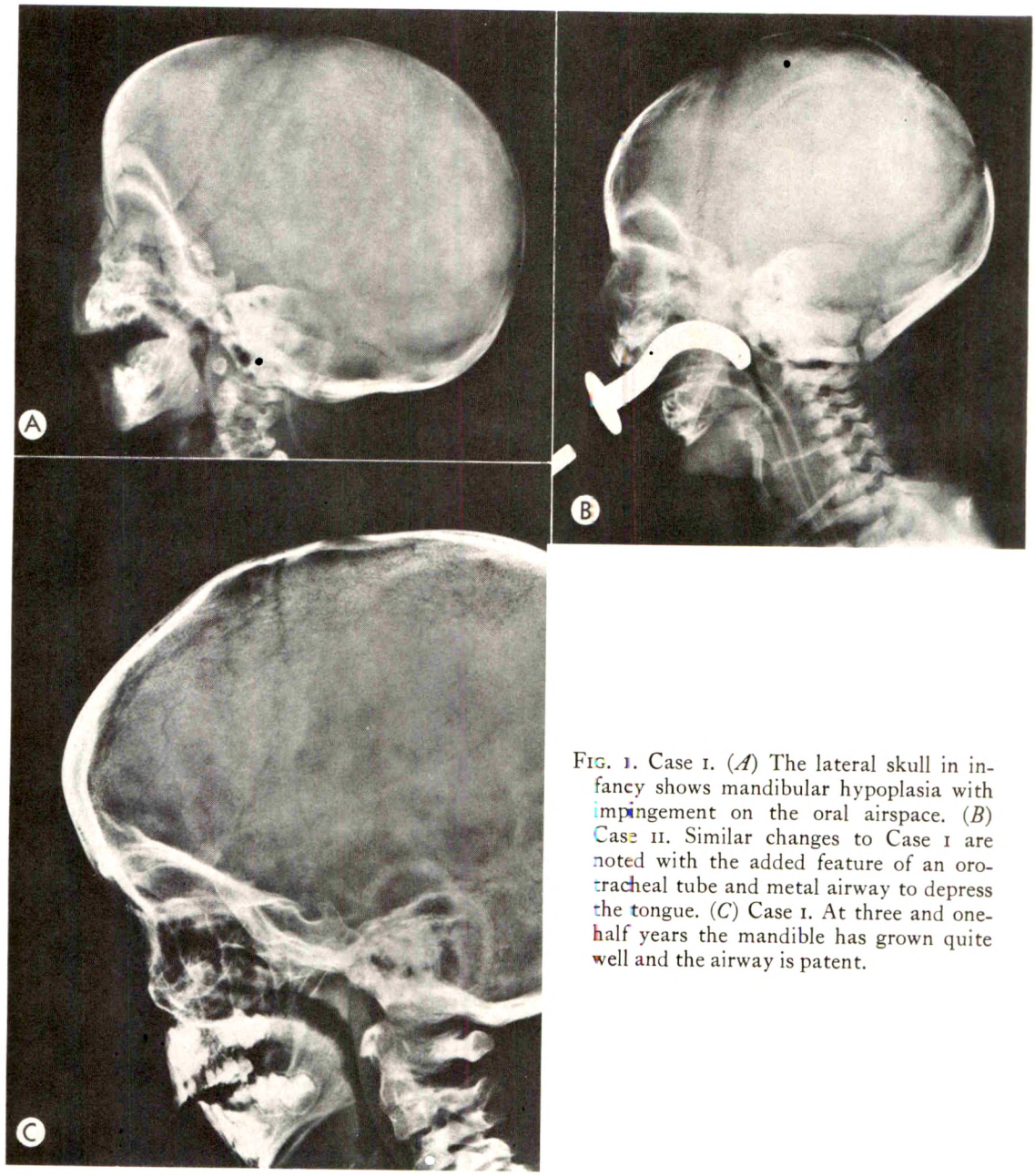

Fig. 1. Case I. (*A*) The lateral skull in infancy shows mandibular hypoplasia with impingement on the oral airspace. (*B*) Case II. Similar changes to Case I are noted with the added feature of an orotracheal tube and metal airway to depress the tongue. (*C*) Case I. At three and one-half years the mandible has grown quite well and the airway is patent.

six ounces at birth and was noted to have a cleft involving the soft and hard palate, and micrognathia. Respiratory distress was not present after birth. At two months of age progressive respiratory problems developed including pneumonia and sternal retractions. The infant was transferred to Babies Hospital where she was noted to be in a fixed opisthotonic position, to be odd-looking with a small mandible, pointed nose and large eyes. She appeared to have disproportionately short arms and legs, especially in the proximal root segments. Head circumference was normal. Her length of 50.5 centimeters was one standard deviation below normal. Roentgenographic findings will be discussed below. Among the initial diagnoses considered were metatropic dwarfism, as well as an atypical diastrophic dwarfism. Genetic consultation was sought since a sibling with micrognathia had died at two months of age; the

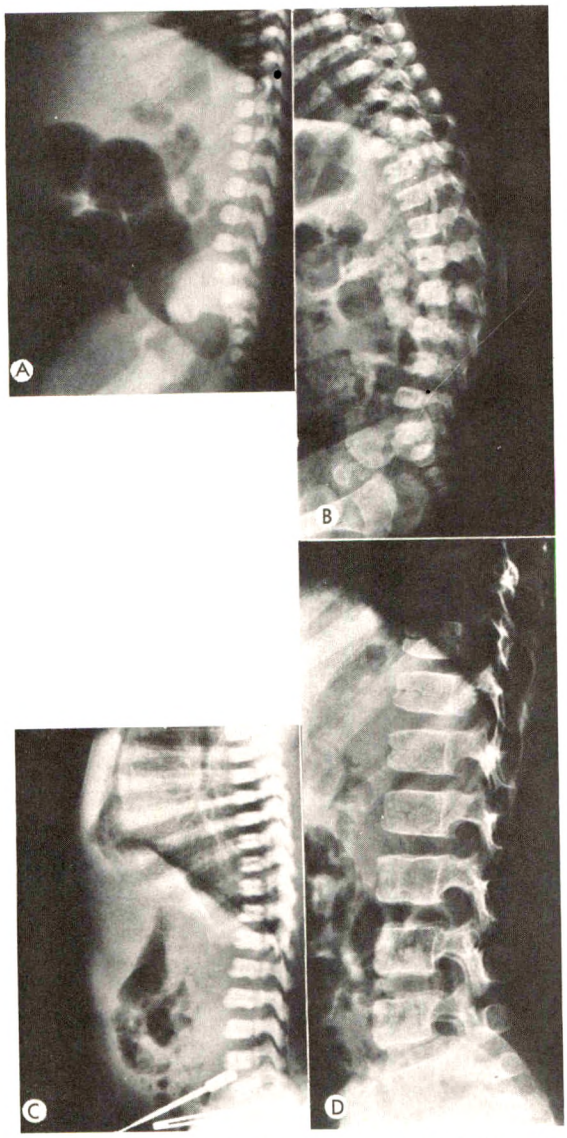

FIG. 2. (*A*) A normal spine is seen in lateral view; the infant has rectal atresia with rectourethral fistula which accounts for the colon distention. (*B*) Case I. Note the incomplete coronal clefts which result in a slight bulbous contour to the anterior and posterior halves of each body. (*C*) Case II. Similar although less marked changes are present. (*D*) Case I. At three and one-half years the spine has returned to normal.

consultant raised the possibility again of diastrophic dwarfism. The retractions and respiratory distress were felt to be secondary problems of secretions plus a large posteriorly placed tongue; the palatal repair was deferred until three years of age. The child, having walked at ten months of age and talked at two and three-quarter years, was noted at three years to be normal in height (96 centimeters). A repeat skeletal survey was obtained (to be discussed below). The palatal defect was repaired.

Comment. The odd appearance and seeming rhizomelic proportions led to suggestions of several types of dwarfism despite her initial length being only one standard deviation below normal; at three years of age she was of normal height.

CASE II. At birth this eight pound male had a cleft palate, micrognathia, and a large posteriorly placed tongue. The neonatal course was characterized by episodes of retractions, nasal flaring, and duskiness, most marked after feedings. His progressive respiratory distress led to tracheostomy. His head circumference was normal and length of 55 centimeters was normal. Skeletal findings as in Case I were noted. He was discharged and no follow-up measurements are available.

ROENTGENOGRAPHIC FINDINGS

Roentgenographic studies were performed in Cases I and II in infancy, and Case I was re-studied at three and one-half years of age.

Skull: (Fig. 1, *A–C*) There is mandibular hypoplasia with impingement on the oral airway. Case I at three and one-half years shows growth of the mandible with the oral airspace now well seen.

Spine: (Fig. 2, *A–D*) When compared with a normal infant the lateral lumbar spine shows an incomplete coronal cleft with a slight bulbous contour of the two halves of the bodies. Case I at three and one-half years of age shows a return to normal.

Pelvis and Femurs: (Fig. 3, *A–E*) When compared with a normal infant, roentgenograms of the pelvis and femurs show a bulbous contour of the ischial and pubic bones; the iliac bones are broad with a smooth acetabular roof and low acetabular angle. The femurs appear dumbbell shaped with prominent trochanters. Similar although lesser changes are in the lower legs. Case I at three and one-half years shows the

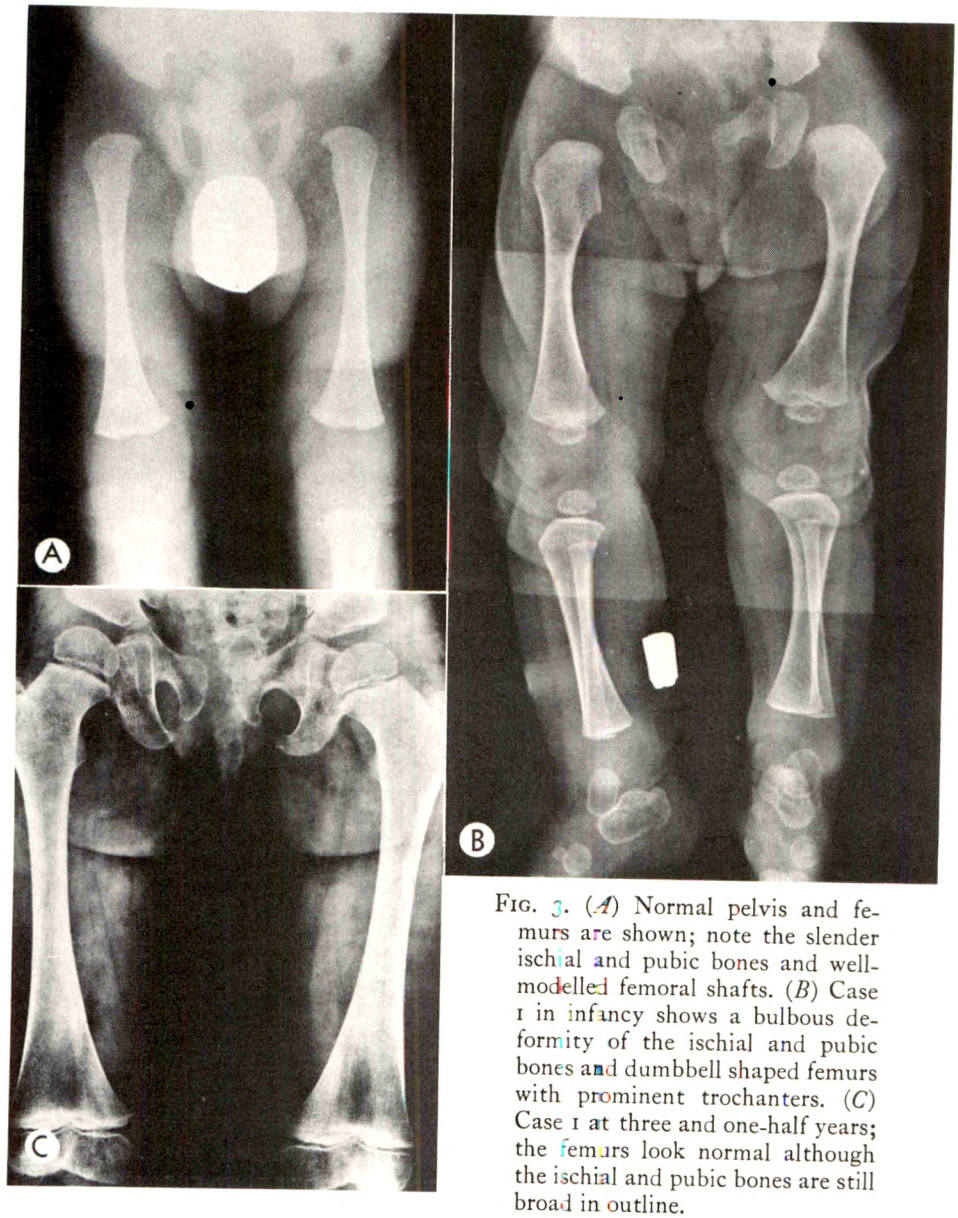

Fig. 3. (*A*) Normal pelvis and femurs are shown; note the slender ischial and pubic bones and well-modelled femoral shafts. (*B*) Case 1 in infancy shows a bulbous deformity of the ischial and pubic bones and dumbbell shaped femurs with prominent trochanters. (*C*) Case 1 at three and one-half years; the femurs look normal although the ischial and pubic bones are still broad in outline.

femurs to appear normal although the ischial and pubic bones remain broad in outline.

Humeri: (Fig. 4, *A* and *B*) The proximal and distal humeri in Case 1 now look quite normal at three and one-half years of age. Case 11 had similar changes.

Comment. The most curious and unexpected aspect of the above studies is that, while the patients appeared to have oddly short arms and legs in infancy, their growth was normal and the prominent roentgenographic changes were not associated with progressive deformity. We lack detailed measurement of length of the proximal *versus* distal bone segments as well as similar measurement in controls of the same age and weight.

DISCUSSION

The patients reported by Robin[6] had respiratory distress and micrognathia with-

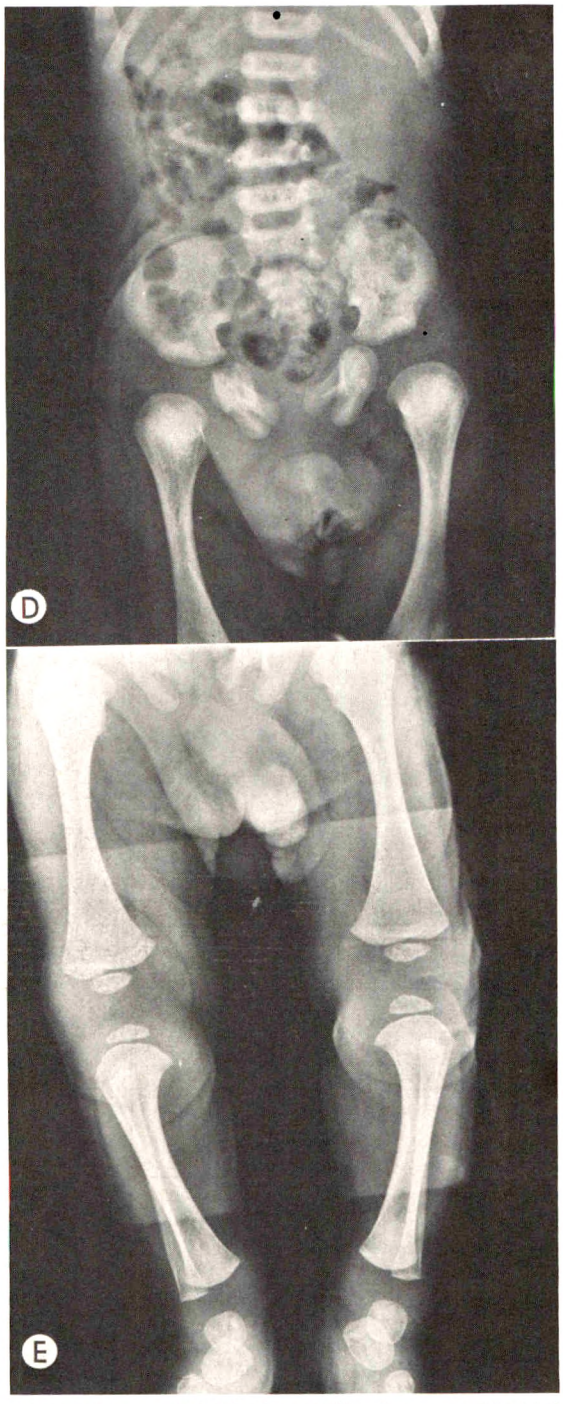

FIG. 3. Case II. (*D*) The broad iliac bones and ischial and pubic changes are well seen; note the femoral shaft contour. (*E*) The tibia shows similar although lesser changes.

out any note of dwarfism or odd proportions. The mandibular hypoplasia, initially ascribed to infection, was later felt to reflect *in utero* pressure of the fetal sternum on the developing chin. More recent speculation has centered on the mandibular hypoplasia as being primary, with secondary displacement of the tongue against the developing palate, leading to a U shaped palatal defect.[1,5] Major bone anomalies have been noted with shortening[8] to virtual absence of the femurs;[2] hand deformities have been seen as well.[2] However, more generalized "chondrodysplasia" was not noted except for the report of Weissenbacher and Zweymuller in 1964, who described a case combining chondrodysplasia and micrognathia. The appearance of their patient also suggested rhizomelic proportions with roentgenograms showing bulbous enlargement of the proximal and distal root bones. Their patient, a male, had marked stridor in the supine position. He resisted attempts to stretch his knee, hip, or elbow joints and favored the prone position; tracheal intubation was required as were gavage feedings. His length of 48.5 centimeters was slightly below normal. They assumed their patient combined "fetal chondrodysplasia" and micrognathia.

However, communication with Dr. Weissenbacher[10] in 1974 reveals that the patient, now ten years old, is at 97.5 percent of normal growth and there are no longer any signs of Pierre Robin syndrome.

Review of the infantile roentgenograms of their case fails to show any sign of achondroplasia (which they initially suspected in 1964) and their notation of undergrown base of the skull probably relates to the normal cranium and tiny mandible. Maroteaux *et al.*[4] felt their cases of "micrognathic dwarfism" were additional examples of the same entity as found in Weissenbacher and Zweymuller's case; they were unaware of the normal growth of that patient. Their three cases of "micrognathic dwarfism" included a first case whose long bones failed to show a dumbbell configuration. The second and third cases do show

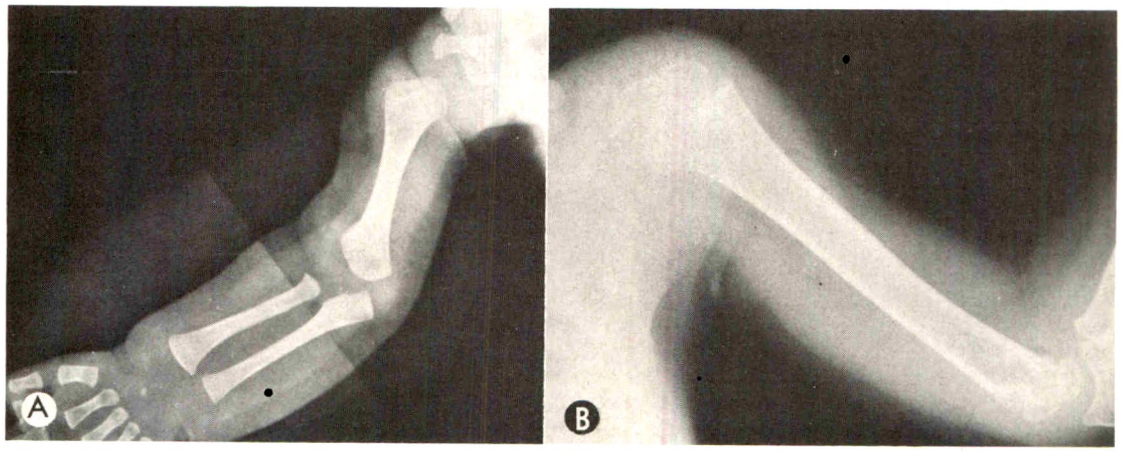

Fig. 4. Case I. (*A*) The humerus shows the same proximal and distal bulbous contour. Case II was identical. Case I. (*B*) At three and one-half years the humerus appears normal.

this bulbous deformity but the patients were small, with the third case below the third percentile.

We do not feel that our two cases and the original case of Weissenbacher and Zweymuller should be termed "micrognathic dwarfs." Further, micrognathia can accompany other types of dwarfism, such as diastrophic dwarfism[1] making the term "micrognathic dwarf" confusing to us.

The proper diagnosis of a patient with micrognathia and seeming rhizomelic deformity requires careful measurements, skeletal roentgenographic study and serial observations. Confusion can obviously exist between several genetically different types of dwarfism, including diastrophic and metatropic dwarfism and the Kniest syndrome.[7] Achondroplasia can usually be promptly excluded since its neonatal roentgenographic manifestations are typical.[7]

Diastrophic dwarfs usually have club feet, joint contractures, and hypermobile "hitch-hiker thumbs," and the long bones are not bulbous in configuration.[7] Metatropic dwarfism shows a changing pattern from infancy with the initial long trunk, short limb appearance being altered by progressive spinal flattening. The patients do show the trochanter enlargement with "dumbbell" contour of the root bones noted in our cases but are dwarfed at birth and become progressively deformed; the chest is narrow.[7]

The Kniest syndrome (clinically confused with both metatropic dwarfism and achondroplasia) is a rare condition[3,7] characterized by a peculiar flattened facies, short trunk, broad chest, shortened root bones with dumbbell configuration, and moderate neonatal spinal changes that are similar to our cases but go on to develop progressive flattening with growth. Should a patient with Kniest syndrome also have micrognathia at birth, it could easily be confused with both the Weissenbacher and Zweymuller syndrome and the "micrognathic dwarf" of Maroteaux *et al.*[4] Such occurred in the following case.

Case III. (Fig. 5, *A–E*) This white male was noted to have micrognathia, posteriorly placed tongue, and cleft palate at birth. A clinical disproportionate shortness of the extremities was noted with the root bones primarily involved. The first diagnosis was of Pierre Robin syndrome with achondroplasia. His length of 46 centimeters at eight months of age placed his growth below the third percentile.

Initial roentgenographic findings of dumbbell femurs and humeri were noted. The thorax was broad with horizontal ribs. The lumbar spine showed bulbous changes similar to our Case I and Case II and the patient of Weissenbacher and Zweymuller. He was then diagnosed as a "micrognathic dwarf."

As his dwarfism became more obvious, serial

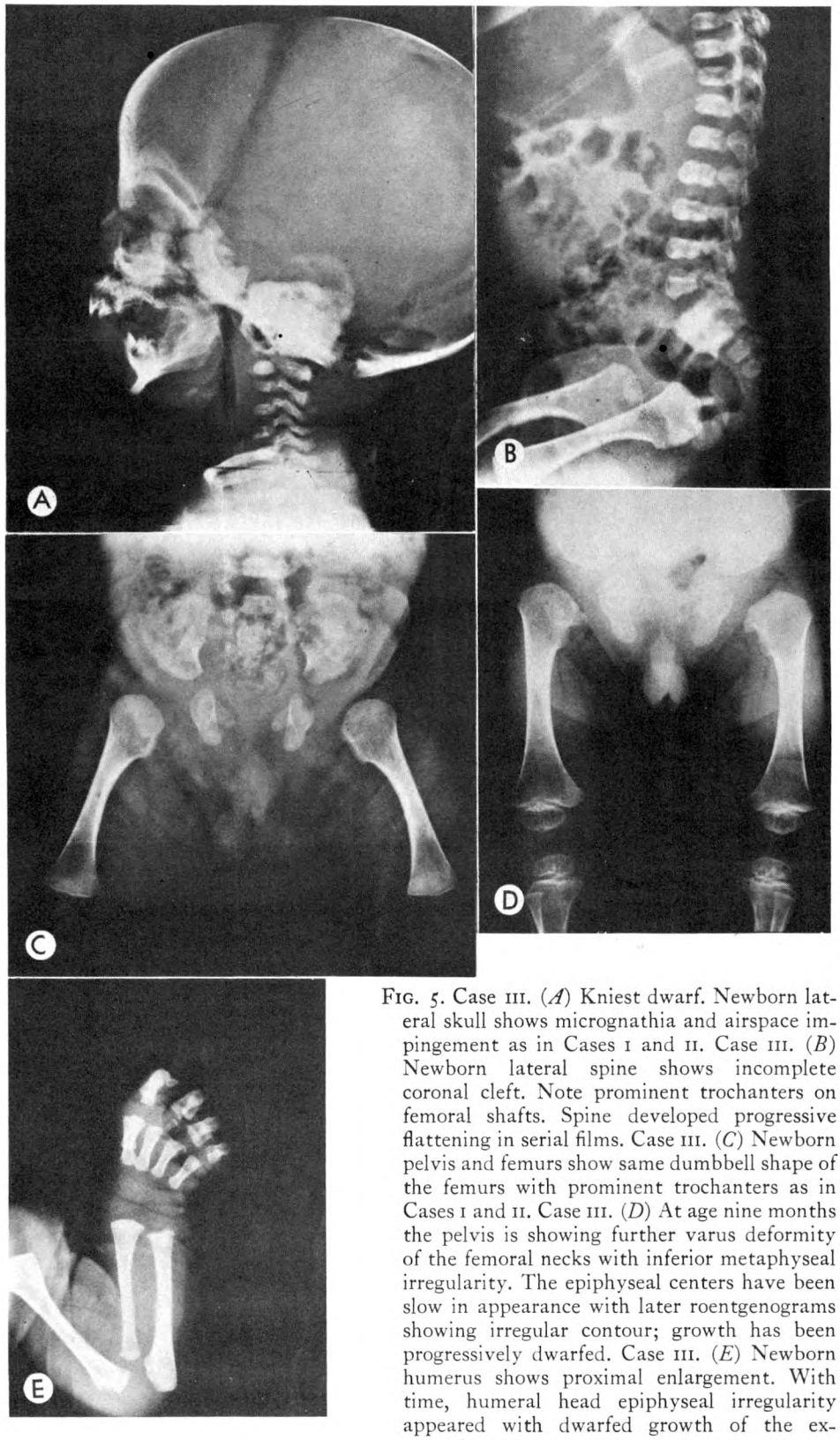

FIG. 5. Case III. (*A*) Kniest dwarf. Newborn lateral skull shows micrognathia and airspace impingement as in Cases I and II. Case III. (*B*) Newborn lateral spine shows incomplete coronal cleft. Note prominent trochanters on femoral shafts. Spine developed progressive flattening in serial films. Case III. (*C*) Newborn pelvis and femurs show same dumbbell shape of the femurs with prominent trochanters as in Cases I and II. Case III. (*D*) At age nine months the pelvis is showing further varus deformity of the femoral necks with inferior metaphyseal irregularity. The epiphyseal centers have been slow in appearance with later roentgenograms showing irregular contour; growth has been progressively dwarfed. Case III. (*E*) Newborn humerus shows proximal enlargement. With time, humeral head epiphyseal irregularity appeared with dwarfed growth of the extremities.

roentgenographic studies showed progressive flattening of the spine with marked coxa vara of the femoral necks. Femoral epiphyseal ossification was retarded. The femoral necks began to show inferior irregularity as noted in the Kniest syndrome. Consultation with Dr. Frederick Silverman led to a suggestion that the patient actually had the Kniest syndrome and time appears to have proved him correct.

Comment. The patient obviously combined dwarfism and micrognathia. It required time and serial roentgenographic studies to show the changes of the Kniest syndrome. Micrognathia as such is not usually part of the Kniest syndrome although the patients are described as having a peculiar flattened facies.

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REFERENCES

1. Hanson, J. W., and Smith, D. W. U-shaped palatal defect in Pierre Robin anomaly: developmental and clinical relevance. *J. Pediat.*, 1975, *87*, 30–33.
2. Holthusen, W. Pierre Robin syndrome: unusual associated developmental defects. *Ann. radiol.*, 1971, *15*, 253–262.
3. Lachman, R. S., Rimoin, D. L., Hollister, D. W., Dorst, J. P., Siggers, D. C., McAlister, W., Kaufman, R. L., and Langer, L. O. Kniest syndrome. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1975, *123*, 805–814.
4. Maroteaux, P., Roux, C., and Fruchter, Z. Micrognathic dwarfism. *Presse méd.*, 1970, *78*, 2371–2374.
5. Randall, P., Krogman, W. M., and Jahina, S. Pierre Robin and syndrome that bears his name. *Cleft Palate J.*, 1955, *2*, 237–245.
6. Roein, P. Glossoptosis due to atresia and hypotrophy of mandible. *Am. J. Dis. Childhood*, 1934, *48*, 541–547.
7. Spranger, J. W., Langer, L. O., and Widermann, H. R. Bone Dysplasia. W. B. Saunders Company, Philadelphia, 1974, pp. 108–119.
8. Walden, R. H., Logosso, R. L., and Brennan, L. Pierre Robin syndrome in association with combined congenital lengthening and shortening of long bones. *Plast. & Reconstruct. Surg.*, 1971, *48*, 80–82.
9. Weissenbacher, G., and Zweymuller, E. Coincidental occurrence of Pierre Robin and fetal chondrodysplasia. *Monatsschr. Kinderh.*, 1964, *112*, 315–317.
10. Weissenbacher, G. Personal communication, 1974.

THE ASSOCIATION OF CLEIDOCRANIAL DYSOSTOSIS WITH HEARING LOSS*

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ABSTRACT:

Three new cases of cleidocranial dysostosis with hearing loss are reported in this paper. The significant points concerning this association are: (1) the hearing deficit is predominantly a middle ear conduction problem secondary to structural abnormalities of the ossicles; (2) there is sometimes a small bone conduction deficit indicating either a cochlear or an eighth nerve problem; (3) the middle ear hearing loss was corrected surgically in one reported case; (4) there is dense sclerosis of the temporal bone which makes a middle ear operation technically difficult; and (5) hearing loss with cleidocranial dysostosis may be more common than the number of cases in the literature suggests.

CLEIDOCRANIAL dysostosis is a rare, inherited abnormality which affects the development of membranous and cartilaginous bone.[5,6,9] In spite of the striking roentgenologic findings, there are relatively few clinical problems associated with this disease, *i.e.*, cephalopelvic disproportion, dental dysplasia, and hearing loss. Cephalopelvic disproportion and dental dysplasia have been amply considered. However, the relationship of cleidocranial dysostosis to hearing loss has been cited in only eight cases, all in the otologic literature. Because this association has not been described in radiologic journals, this paper will discuss the roentgen findings in the temporal bone and includes three new cases with hearing loss.

PERTINENT LITERATURE

Nager and De Reynier[7] in 1948 first reported three cases (in a single family) of cleidocranial dysostosis with a mixed hearing loss. All of these patients had narrowing of the external auditory canals and mastoid hypoplasia. In 1954, Davis[1] described a single case in a 24 year old man whose hearing was slightly improved by a bone conduction hearing aid. In 1958, Gay[3] reported the fifth case in an eleven year old girl with moderate mixed deafness. This patient also had no pneumatization of the mastoids and narrowing of the external auditory canals. Jaffee[4] reported a single case with no previous history of ear infection in a five year old male with normal ear canals; however, the tympanic membranes were dull and retracted. Testing by audiometry revealed a bilateral conductive hearing loss. Roentgenography showed sclerotic mastoids with only a few air cells. Adenoidectomy and bilateral myringotomy resulted in a marked hearing improvement. The seventh example of cleidocranial dysostosis with hearing loss was reported by Fønss.[2] The patient, a 13 year old girl, had a deformed right external auricle, narrowed external canals, and a conductive hearing loss. Audiometry showed 50 and 25 decibels of conductive hearing deficit on the right and left sides, respectively. Plain roentgenograms demonstrated dense mastoids with absent pneumatization. Temporal bone laminagraphy revealed a narrow right external canal with an abnormally small middle ear. In addition, the ossicles were clumped together in the attic; neither the handle of the malleus nor the long process of the incus was identified. Laminagrams of the left temporal bone

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TABLE I

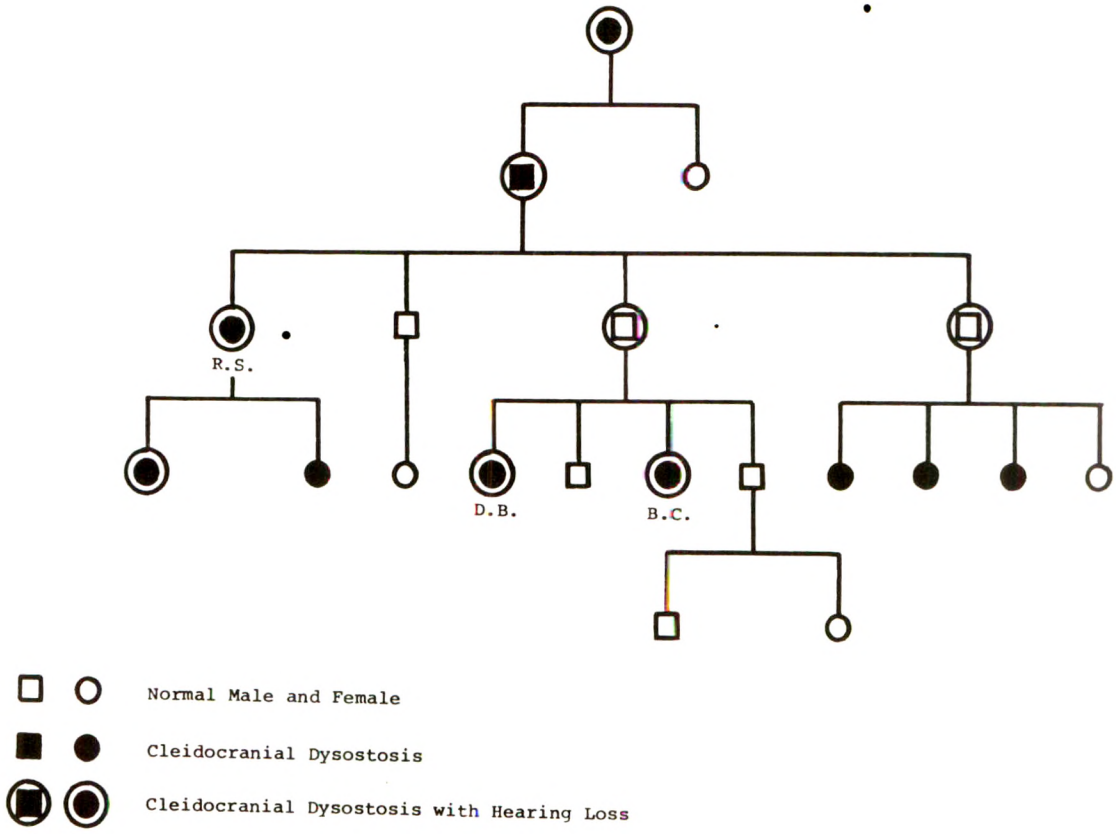

FIG. 1. Five additional members of family have documented cleidocranial dysostosis with associated hearing loss.

showed slight narrowing of the external canal and an abnormally small middle ear and ossicles. However, the long process of the incus and the handle of the malleus could be identified. The inner ear and porus acousticus were normal bilaterally. The last case, recorded by Pou[8] in 1971, occurred in a 23 year old girl who developed progressive loss of hearing over an eight month period. On physical examination, she had narrow external auditory canals. Audiometry showed a 40 decibel mixed deficit on the left and an 80 decibel loss on the right, primarily conductive. She underwent a right middle ear exploration through an external auditory approach. The procedure was difficult because of severe narrowing of the external canal. At operation, the long process of the incus was short, causing the stapes to be fixed against the facial nerve canal. In addition, the footplate of the stapes was thickened and fixed at the oval window. Both the incus and the malleus were also considerably thickened. The footplate of the stapes was removed and a wire-vein prosthesis was introduced. Following operation, the conductive hearing loss on the right was improved.

REPORT OF CASES

Our three patients (R.S., D.B., and B.C.), all from the same family, have roentgenographically documented cleidocranial dysostosis and audiometrically documented hearing loss. There are thirteen individuals with cleidocranial dysostosis in this family over a four generation span. Eight of the thirteen have an associated

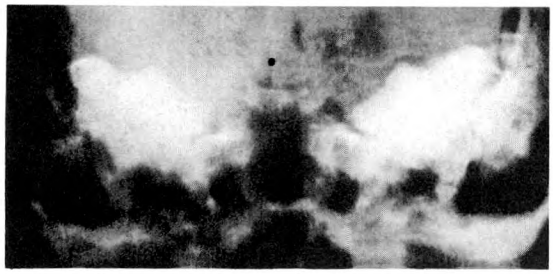

FIG. 2. Towne projection showing marked sclerosis of both petrous bones.

hearing loss. R.S., D.B., and B.C. are the only three who have been fully documented.

CASE I. R.S. is a 38 year old woman with a 25 year history of bilateral progressive hearing loss, more marked in the left ear. Audiometry showed a combined loss in the left ear averaging 82 decibels, with a 35–45 decibel air-bone gap. There was no prior history of ear infection. Past history included three cesarean sections for cephalopelvic disproportion. Roentgenography of the chest showed the clavicles to be hypoplastic, the right clavicle being only a poorly defined, thin bone. The left clavicle, also thin, consisted of at least two segments with a pseudoarthrosis. Skull roentgenograms showed many Wormian bones, a persistent metopic suture, and poor pneumatization of the mastoids. A left stapedectomy was attempted through the external auditory canal. However, the canal was so narrow that it was impossible to obtain adequate exposure. Two months later, the patient underwent a second operation. Through a posterior auricular approach, the sclerotic, thickened bony auditory canal was thinned with an electric drill to permit the entry of an adequate ear speculum. The skin was then elevated down to the annulus and the eardrum reflected forward. This revealed marked thickening of the posterior auditory canal with an extensive overhang, obscuring the oval window and the stapes. Considerable bleeding ensued and it was not possible to expose the oval window and stapes adequately. Therefore, the operation was terminated.

CASES II and III. D.B. and B.C. are nieces of R.S. Roentgenography of the chest and skull demonstrated the characteristic findings of cleidocranial dysostosis in both patients. Audiometry showed both of them to have mild to moderate mixed hearing loss. Neither patient had a history of previous ear infection. Five additional members of this family have documented cleidocranial dysostosis and are known by history to have an associated hearing loss (Fig. 1).

ROENTGENOLOGIC FINDINGS

This discussion will be limited to a description of the temporal bone because the latter has received scant attention in the literature. Jarvis and Keats[5] mention "thickening" of the temporal squamosa in passing, but no other changes. All of the patients in our series had pronounced sclerosis of the petrous and mastoid bones due to cortical thickening (Fig. 2). The squamosa was similarly involved, although the degree of squamosal thickening varied somewhat from patient to patient (Fig. 3). However, in all patients, the thin, normally radiolucent squamosa was radiopaque. The petrosal sclerosis was so striking that the radiolucent semicircular canals and cochlea stood out in bold relief, reminiscent of the newborn skull (Fig. 4).

DISCUSSION

Dental dysplasia, cephalopelvic dispro-

FIG. 3. Lateral view of skull demonstrating increased density of the temporal squamosa. Normally, this is one of the thinnest areas in the calvaria.

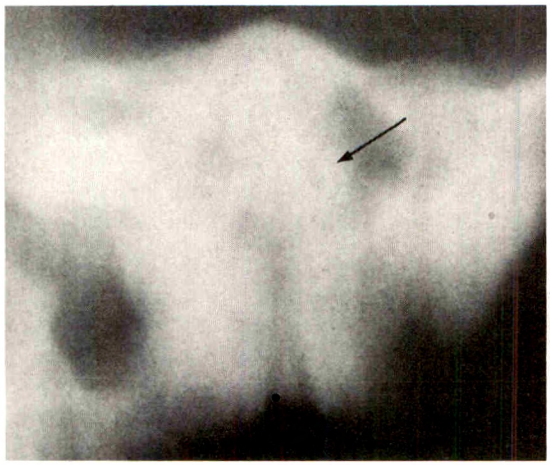

Fig. 4. Coned-down view of left petrous bone showing the marked bony thickening around the semicircular canals (arrow).

portion, and hearing loss are the only significant clinical problems associated with cleidocranial dysostosis. There are three possibilities which may explain the paucity of reports of hearing loss in this disease: (1) since the hearing loss is partial rather than total, patients may not be inclined to seek out medical advice; (2) only a few physicians are aware of the association of cleidocranial dysostosis with hearing loss and look for it; and (3) the association of cleidocranial dysostosis with hearing loss, indeed, may be rare.

The etiology of the hearing deficit is not entirely clear although there are at least two factors involved. One component is a small bone conduction loss which indicates either a neural or a cochlear problem. The relationship of the sclerosis of the temporal bone to a neural or cochlear abnormality is unknown. It is conceivable that sclerotic bone may encroach on the eighth nerve. However, a normal facial nerve argues against this. The predominant component in the hearing loss in all cases tested by audiometry is a middle ear conduction problem. The one case in which laminagraphy was done showed dense abnormal ossicles clumped together in the attic. Operative visualization in another patient showed bulky, thick ossicles with fixation of the footplate of the stapes to the oval window. It seems likely that abnormalities of the ossicles will be found in other cases of cleidocranial dysostosis if specifically looked for.

Appreciation of the nature of the hearing loss in cleidocranial dysostosis is important because of the possibility of surgical correction. Also important is knowledge of the dense sclerosis of the temporal bone which may necessitate a modification of the surgical approach.

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REFERENCES

1. Davis, P. L. Deafness and cleidocranial dysostosis. *A.M.A. Arch. Otolaryng.*, 1954, *59*, 602–603.
2. Føns, M. Ear malformations in cleidocranial dysostosis. *Acta oto-laryng.*, 1969, *67*, 483–489.
3. Gay, I. Case of dysostosis cleidocranialis with mixed deafness. *J. Laryng. & Otol.*, 1958, *72*, 915.
4. Jaffee S. Congenital shoulder-neck auditory anomalies. *Laryngoscope*, 1968, *78*, 2119–2139.
5. Jarvis, J. L., and Keats, T. E. Cleidocranial dysostosis: review of 40 new cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *121*, 5–16.
6. Lasker, G. W. Inheritance of cleidocranial dysostosis. *Human Biol.*, 1946, *18*, 103–126.
7. Nager, F. R., and De Reynier, J. P. Das Gehororgan bei den Angeborenen Kopfmissbildungen. *Pract. oto-rhino-laryng.*, 1948, *10*, Suppl. 2, 1–125.
8. Pou, J. W. Congenital anomalies of middle ear: presentation of two cases. *Laryngoscope*, 1971, *81*, 831–839.
9. Soule, A. D. Mutational dysostosis (cleido-cranial dysostosis). *J. Bone & Joint Surg.*, 1946, *28*, 81–102.

GASTROINTESTINAL MANIFESTATIONS OF THE MUSCULAR DYSTROPHIES*

A REVIEW OF ROENTGEN FINDINGS

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ABSTRACT:

Five patients with acute megacolon with varied types of progressive muscular dystrophy are presented. Dysfunction of smooth muscle among patients with muscular dystrophy is reviewed. The extra gastrointestinal roentgen features are summarized. Recognition of the diffuse smooth muscular involvement among patients with muscular dystrophy is stressed for proper diagnosis and patient management.

THE muscular dystrophies are generally considered to be disorders of striated or voluntary muscle. However, it is also recognized that involvement of smooth muscle frequently occurs.[8,12,22,25] Emphasis on involvement of the gastrointestinal tract has largely been concerned with esophageal dysfunction[5,9,21] and gallbladder disease,[5,6] particularly in patients with myotonic dystrophy. Five patients with varied types of muscular dystrophy seen at the Mount Sinai Hospital presented with recurrent complaints of abdominal pain, distention and obstruction resulting from acute megacolon (non-obstructing). In addition, three of these patients had associated sigmoid volvulus. These unusual findings prompted this report.

REPORT OF CASES

CASE I. A 35 year old male was diagnosed eight years prior to admission as having myotonic dystrophy. Two years prior to admission he underwent an exploratory laparotomy at another hospital because of symptoms of recurrent intestinal obstruction. No organic cause of obstruction was found at surgery. The patient was well until the current admission, when he presented with abdominal distention, crampy abdominal pains, nausea, and vomiting. He was managed medically with a long tube and parenteral fluids. Physical findings, typical of myotonia dystrophica, included cataracts, frontal baldness, and atrophy and weakness of the muscles of the distal extremities and face. A barium enema study revealed marked segmental dilatation of the colon from the splenic flexure to the proximal sigmoid. The sigmoid appeared relatively narrow and showed normal mucosa and normal contractility on postevacuation roentgenograms. The rectum was normal. The length of dilated colon was less than seen on a preliminary abdominal roentgenogram obtained on admission two days earlier (Fig. 1, *A–D*). An upper gastrointestinal series demonstrated irregular and poor motility in the subaortic segment of the esophagus; the primary wave disappeared at the level of the aortic knob. The caliber of the esophagus was normal. The stomach was transverse with partial, nonobstructive, organoaxial rotation. The small bowel study demonstrated dilated proximal jejunal loops, and the transit time was normal (Fig. 1, *E* and *F*). A skull series was normal.

CASE II. A 22 year old male was diagnosed during childhood as having myotonia dystrophica. He had a four year history prior to admission of severe recurrent crampy abdominal pains associated with either diarrhea or black stools occuring one to three times per week and occasionally lasting one to two days. One month prior to admission, a barium enema examination revealed a markedly redundant and dilated sigmoid colon with a volvulus

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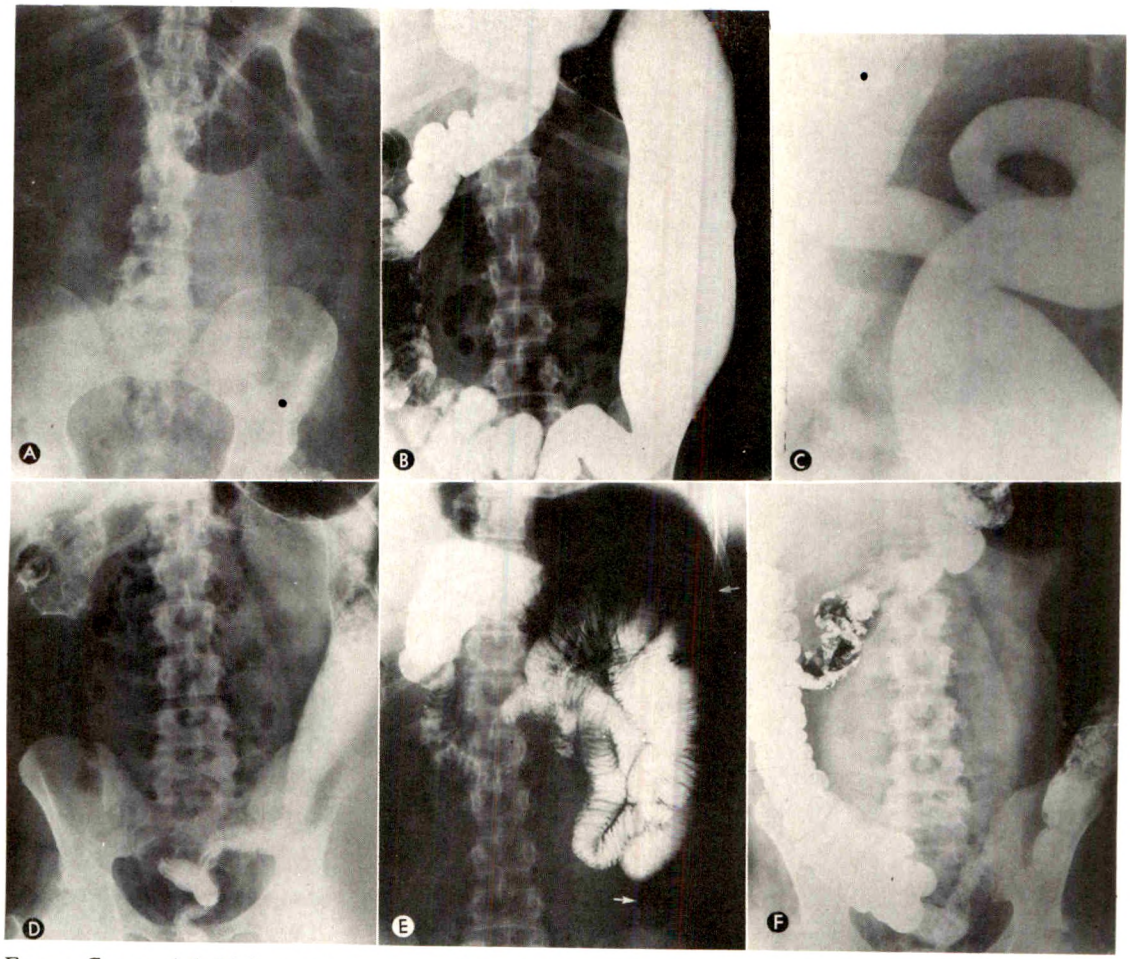

Fig. 1. Case 1. (*A*) Plain roentgenogram of the abdomen shows marked dilatation of the colon from the hepatic flexure to the sigmoid. The left colon is relatively ahaustral. The ascending colon is normal in caliber. There is no small bowel distention. (*B*) Barium enema examination performed two days later demonstrates a dilated, ahaustral colon from the splenic flexure to the sigmoid with the transverse colon normal in caliber on this roentgenogram. The right colon is normal and contains fecal matter. (*C*) Spot view of the sigmoid demonstrates smooth narrowing of the sigmoid colon which merges smoothly and imperceptibly with the normal rectum and dilated descending colon. (*D*) Post evacuation roentgenogram shows normal contractility of the sigmoid with normal mucosa. The descending colon is less dilated. (*E*) Upper gastrointestinal series shows a transverse stomach with partial organoaxial rotation. The jejunal loops are dilated. The left colon (arrows) is still seen. (*F*) Examination six hours after the barium meal study shows all the barium now in the colon. The distended left colon does not retain barium.

which could be reduced with sigmoid intubation. The patient entered Mount Sinai Hospital for elective resection of the megasigmoid.

Pulmonary function studies revealed a slight decrease in ventilatory capacity with a marked decrease in maximal voluntary ventilation. The electrocardiogram was normal. Biopsies of the thenar eminence and gastrocnemius muscles, and an electromyogram were positive for muscular dystrophy. Pertinent physical findings included frontal baldness, testicular atrophy, ptosis, and atrophy of the muscles of the distal extremities and myotonia.

A barium enema examination performed prior to surgery revealed a dilated colon from the hepatic flexure to the rectum. The sigmoid was markedly redundant and partially rotated on itself. A narrowed segment in the sigmoid represented one limb of the volvulus. The retrograde filling of the colon was slow and incomplete (Fig. 2*A*). An upper gastrointestinal series performed one year earlier revealed a

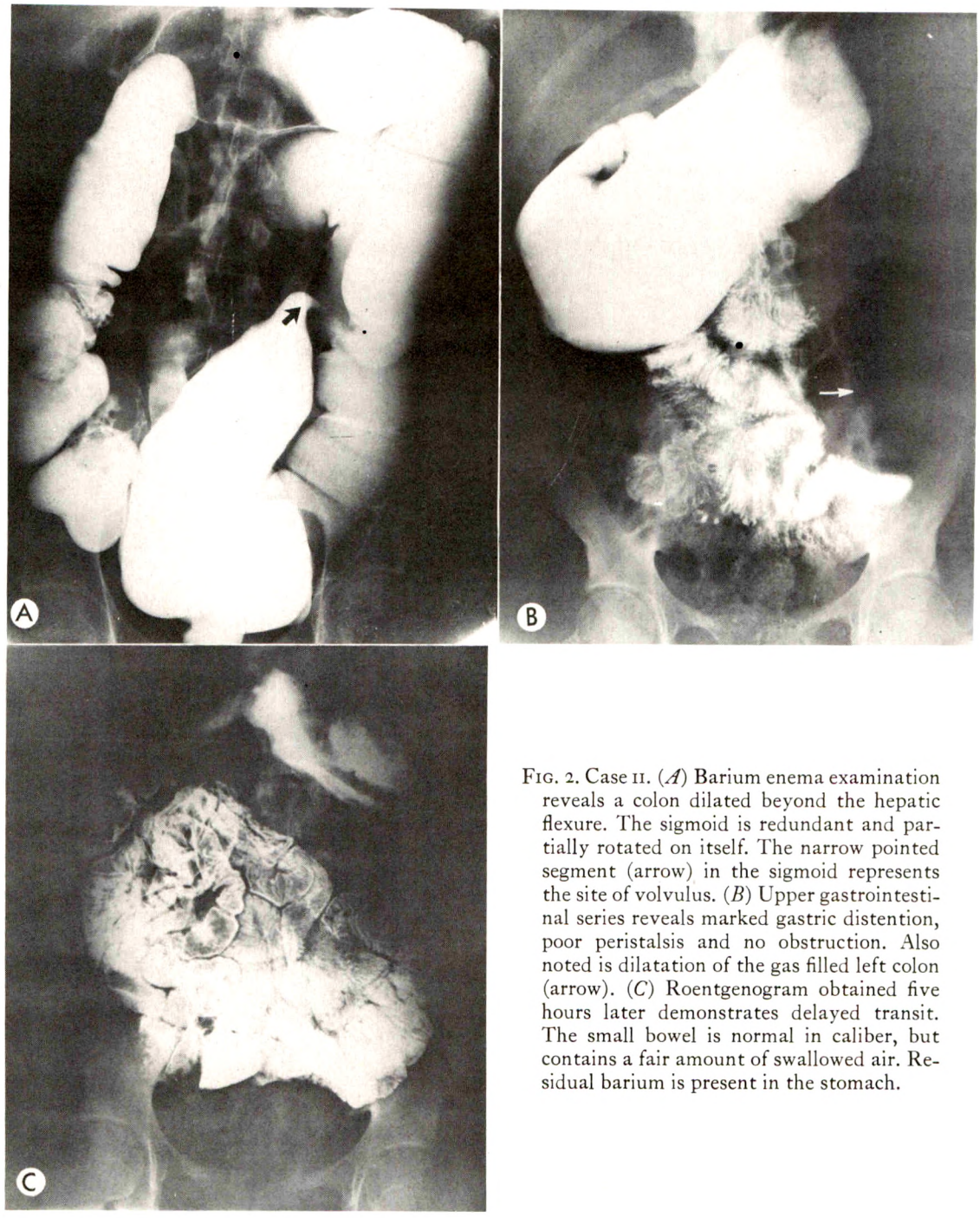

FIG. 2. Case II. (*A*) Barium enema examination reveals a colon dilated beyond the hepatic flexure. The sigmoid is redundant and partially rotated on itself. The narrow pointed segment (arrow) in the sigmoid represents the site of volvulus. (*B*) Upper gastrointestinal series reveals marked gastric distention, poor peristalsis and no obstruction. Also noted is dilatation of the gas filled left colon (arrow). (*C*) Roentgenogram obtained five hours later demonstrates delayed transit. The small bowel is normal in caliber, but contains a fair amount of swallowed air. Residual barium is present in the stomach.

markedly dilated stomach without obstruction. Examination obtained five hours after the barium meal examination showed most of the barium within the jejunum and proximal ileum indicating a slow transit (Fig. 2, *B* and *C*).

CASE III. A 54 year old female was diagnosed as having a progressive muscular dystrophy at age 40. In addition to complaints referrable to weakness of the muscles in her extremities, she also had a six year history of recurrent episodes of acute abdominal pain associated with bloating and constipation. Three weeks prior to admission, she developed a left lower

lobe pneumonia. Pertinent physical findings included atrophy of the proximal skeletal muscles including the chest, resulting in slight difficulty in breathing which required the use of accessory respiratory muscles.

Barium enema examination performed one week prior to admission during an acute episode of abdominal pain and distention disclosed a sigmoid volvulus. This was reduced by rectal intubation. The patient was admitted for elective resection because of recurrent volvulus. A plain roentgenogram of the abdomen prior to surgery showed distention of the colon with retention of barium in the descending colon from the barium enema examination performed one week before. The retained barium was above the site of obstruction even though the tip of the rectal tube was beyond the site of the volvulus. A skull roentgenogram disclosed hyperostosis frontalis interna.

The resected sigmoid was dilated. Histologic study did not disclose a significant amount of muscle atrophy or fibrous replacement even though clinical and electromyographic studies were consistent with muscular dystrophy. One month following resection of the sigmoid, the patient was readmitted for a partial small bowel obstruction and was treated successfully with a Cantor tube (Fig. 3).

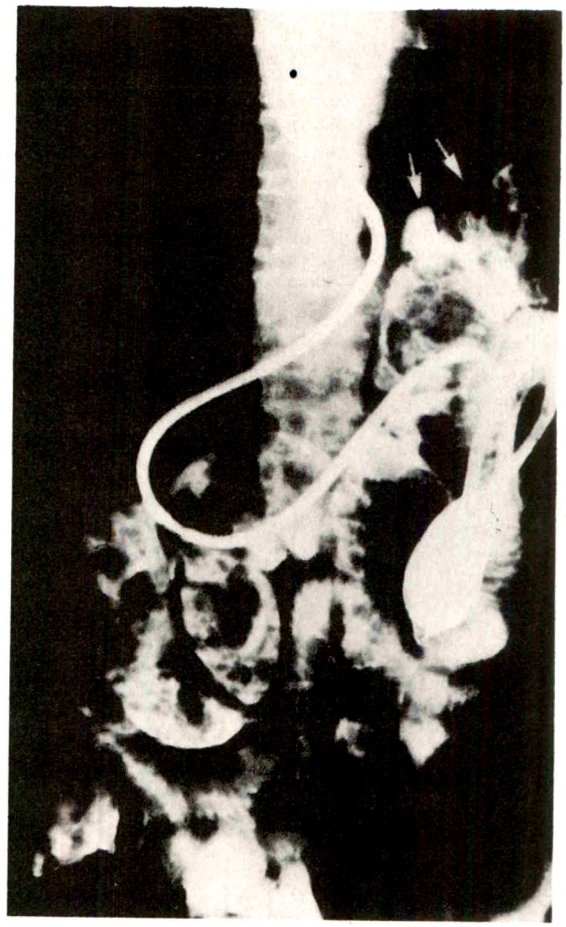

Fig. 3. Case III. A small bowel study performed through a Cantor tube one month postsigmoid resection for recurrent volvulus reveals slight widening of the small bowel. There is no obstruction at this time. Typical findings of obstruction were seen on a flat plate of the abdomen one week prior to this examination. The left colon outlined by contrast medium is dilated and contains air and a considerable amount of fecal matter (arrows). The right colon is less wide. Pseudosacculation is seen at the splenic flexure.

Case IV. A 44 year old female developed facioscapulohumeral type of muscular dystrophy at age ten. Her abdominal complaints began five years before admission with cholelithiasis and cholecystititis. She underwent a cholecystectomy. A barium enema examination performed at the time of this surgery revealed marked dilatation of the right colon. Two months after the gallbladder surgery, the patient was treated for intestinal obstruction conservatively by a long tube. Other gastrointestinal complaints included left lower quadrant crampy pain relieved by defecation and constipation alternating with diarrhea and passage of mucus. Because of complaints of severe abdominal pain the patient was admitted to Mount Sinai Hospital. Positive physical findings included atrophy and weakness of the proximal muscles of the upper extremities, face, and pelvis. The deep tendon reflexes were absent. The patient's abdominal complaints subsided following antispasmodic treatment. A barium enema examination at this time revealed an ahaustral megacolon which showed poor evacuation (Fig. 4*A*). A chest roentgenogram demonstrated recurrent subluxation of both shoulders (Fig. 4*B*).

Case V.* A 27 year old male was diagnosed as having myotonia dystrophica six years prior to the current admission. Gastrointestinal complaints began five years earlier and consisted of recurrent episodes of colicky periumbilical pain associated with black stools and diarrhea. Because of the increasing severity of the abdominal symptoms, the patient was

* This case has been published recently as a single case.[7]

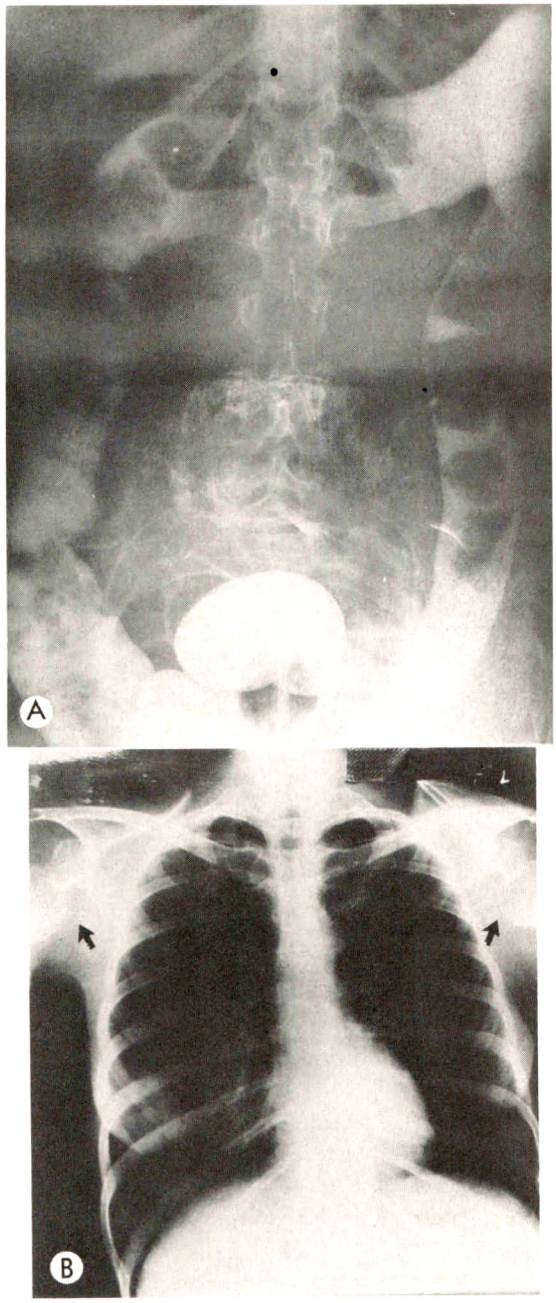

FIG. 4. Case IV. (*A*) A barium enema examination in a patient with facioscapulohumeral type of muscular dystrophy reveals an ahaustral dilated colon. The dilatation is more in the transverse colon and cecum. (*B*) A chest roentgenogram reveals bilateral subluxation of the shoulders (arrows).

admitted. Other major complaints included mild dysphagia and urinary incontinence. A past history of recurrent upper respiratory infection was also obtained. Major physical findings included frontal baldness, weakness and atrophy of the muscles of the forearm, face, and neck. Cranial nerves eight to eleven were also involved. Myotonia was evident clinically and was confirmed by electromyographic study. Pulmonary function studies indicated a disturbance in ventilation with a ventilatory capacity of 73 percent and a maximal voluntary ventilation of 61 percent. A small bowel series showed dilated loops with a delayed transit. Barium enema examination demonstrated a markedly redundant and dilated sigmoid colon associated with an incompletely obstructing sigmoid volvulus (Fig. 5). The patient underwent elective resection. The postoperative course was uneventful.

DISCUSSION

The muscular dystrophies are considered to be a group of clinically well defined genetic and biochemical syndromes characterized by abiotrophy of skeletal muscle *i.e.*, degeneration and atrophy after a latent period of apparently normal development and function.[26] The classification of dystrophic muscular disease (Table 1) is at present based upon genetic and clinical patterns,[23] since the primary pathogenesis of the subdivisions is unknown.[1]

Excluding pharyngeal and upper esophageal impairment which is a disorder of striated muscle, only myotonia dystrophica (Steinert's disease) has been implicated with smooth muscle dysfunction of the gastrointestinal tract.[5,6,8,9,12,21,22,25] A disturbance in the swallowing mechanism is related to reduced amplitude of the pharynx and upper esophagus as well as prolonged relaxation shown by manometric and cineradiographic studies. This results in failure to clear barium adequately from the valleculae and pyriform sinuses with occasional tracheal aspiration and nasal regurgitation.[8,21,24]

Symptoms of dysphagia, regurgitation, and heartburn reported frequently in myotonia dystrophica are related to weak and ineffectual lower esophageal peristalsis in association with the dysfunction of the lower esophageal sphincter and of the skeletal musculature of the pharynx, cricopharyngeus and upper esophagus.[8,21] The esophagus may be dilated, may demon-

strate poor stripping of the barium meal, or may show delay in passage of barium through the esophagogastric junction.

The stomach is affected by muscular dysfunction as shown by reports of acute gastric dilatation and atony. The peristaltic activity is less during the acute phase and may be absent, diminished, or altered in quality along with some degree of dilatation.[14,18]

The higher incidence of gallbladder disease and cholelithiasis in myotonia dystrophica is considered to be the result of impaired contraction and emptying of the gallbladder.[6,8] However, this increased incidence is not resolved, since similar dilatation and poor contractility seen following vagotomy are not associated with a high incidence of cholelithiasis.

Malabsorption and chronic small bowel dilatation and stasis have also implicated smooth muscle dysfunction in myotonia dystrophica with symptoms similar to a blind loop syndrome or to those seen in collagen diseases.[5,10,17]

Fig. 5. Case v. Barium enema examination demonstrates a markedly dilated and redundant left colon with a partial sigmoid volvulus.

Table I

Classification of Types of Progressive Muscular Dystrophy[23]

I. "Pure" Muscular Dystrophy
A. X-linked—severe (Duchenne type)
benign (Becker type)
B. Autosomal recessive
limb-girdle
childhood muscular dystrophy (except Duchenne)
congenital muscular dystrophy
C. Facioscapulohumeral muscular dystrophy
D. Distal muscular dystrophy
E. Ocular muscular dystrophy
F. Oculapharyngeal muscular dystrophy
II. Cases with Myotonia
A. Myotonia congenita (Thomson's disease)
B. Myotonia dystrophica (Steinert's disease)
C. Paramyotonia congenita (Eulenberg's disease)

Recurrent episodes of crampy abdominal pain, diarrhea, obstipation, or a combination of these, associated with the presence of megacolon are also well recognized.[2,7,12,20]

Megacolon with or without complications such as obstruction, perforation, or volvulus may also be seen with Hirschsprung's disease, Chagas' disease, scleroderma, dermatomyositis, diabetes, amyloidosis, myxedema, in schizophrenia and following the use of drugs such as anticholinergics, artane and ganglionic blockers. Idiopathic intestinal pseudo-obstruction is also recognized.[19]

Volvulus of the left colon in the absence of muscular dystrophy usually occurs in later life, and is often related to a redundant sigmoid with a narrow mesocolon. Volvulus was present in three of our patients who were 22, 27 and 54 years old. This complication should not be surprising since decreased motor function may be noted throughout the rest of the gastrointestinal tract. The dilated and atonic segment, like a fluid containing sac, is apt to twist and compromise its own blood supply. This further increases the degree of obstruction, and if unrelieved results in gangrene and perforation.

TABLE II

GASTROINTESTINAL ROENTGEN FEATURES OF THE MUSCULAR DYSTROPHIES

Pharynx: weak or absent cricopharyngeus sphincter
positive vallecular sign
aspiration
Esophagus: decreased, uncoordinated, or non-peristaltic stripping
dilated esophagus
delay at the esophagogastric junction
Stomach: distention, atony, altered peristalsis
Small bowel: obstruction, malabsorption, ileus
Colon: dilatation, pseudo-obstruction, volvulus
Gallbladder: decreased contractability, increased incidence of cholelithiasis

A more recently reported roentgen feature of myotonic dystrophy stressed segmental narrowing rather than dilatation of the sigmoid colon. This was present in all of our patients. The etiology of this feature is not known.[18] It is not quite clear if this segmental narrowing is real or is only apparent, relative to the dilated colon above. Functionally, this narrowed or transitional segment shows normal contractility and smooth distention indicating intact smooth muscle and neuro-muscular function. It is unlike the behavior pattern of a dilated abnormal segment. The involved segment may show irregular, disorganized and ineffectual contraction. If segmental narrowing is a true hypercontractile phase, then one must conclude that another form of smooth muscle involvement may be present, consisting of a hyperactive state or a delayed relaxation. A similar state has not been seen nor reported in any other viscus. Analysis of additional material may clarify this point.

Pathologic examination of the smooth muscle in the dilated part of the gastrointestinal tract in patients with muscular dystrophy has been limited. An often cited reference of a histologic study in four patients with progressive muscular dystrophies revealed atrophy of the muscle cells, edema, fibrosis and normal neural elements; these are not pathognomonic of muscular dystrophy.[3]

The onset of gastrointestinal dysfunction, particularly small or large bowel distention, is often initiated by an acute stress such as trauma or illness.[4] This was present in two of our patients who had pneumonia and cholecystitis shortly before the onset of their acute phase of colon distention. Except for volvulus, the mechanism of acute symptoms has not been explained. Additional factors may be the onset of acute dilatation of the viscus in association with a pre-existing, chronic distention. Alternately, it may be the resistance offered by the transitional segment to a hypo- or aperistaltic segment located proximally.

The fact that smooth muscle dysfunction occurred among muscular dystrophy patients without myotonia dystrophica further broadens the pathologic manifestations of progressive muscular dystrophy, and therefore the recognition that muscular dystrophy is a disorder of both skeletal and smooth muscle.

A summary of the above gastrointestinal roentgen features of the muscular dystrophies is presented in Table II; and the extra-gastrointestinal roentgen features, which are more commonly recognized, are summarized in Table III.

TABLE III

EXTRA-GASTROINTESTINAL ROENTGEN FEATURES OF THE MUSCULAR DYSTROPHIES[4]

Skull:[16] cranial hyperostosis, even in males; small sella turcica, increase in size of sinuses, small basal angle; hypotelorism; small nasal angle
Heart: size usually normal (may develop features of failure secondary to conduction disturbances)
Lungs: increased incidence of pneumonia (infectious, aspiration) secondary to pharyngo-esophageal dysfunction, alveolar hypoventilation (related to decrease diaphragm excursion, weakness of chest musculature, and altered central nervous system respiratory mechanism), or both
Genitourinary: atonic bladder with urinary retention
Musculo-skeletal:[16] replacement of muscle by fat with loss of volume. Secondary involvement of bone with atrophy, demineralization, and contractures

Recognition by the radiologist of all the systemic and roentgen features of patients with muscular dystrophy is important not only to provide proper diagnosis for better patient management in known dystrophy patients, but even to suggest the correct diagnosis if the obvious skeletal and neuromuscular pathology is not fully apparent.

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REFERENCES

1. Adams, R. D. Disorders of Voluntary Muscle. Edited by J. N. Walton. J. & A. Churchill, Ltd., London, 1969, pp. 186–189.
2. Bertrand, L. Le megacolon dans la maladie de Steinert. *Rev. neurol.*, 1949, *81*, 480–486.
3. Bevans, M. Changes in musculature of gastrointestinal tract and in myocardium in progressive muscular dystrophy. *Arch. Path.*, 1945, *40*, 225–238.
4. Caughey, J. E., and Myrianthopoulos, N. C. Dystrophia Myotonia and Related Disorders. Charles C Thomas, Publisher, Springfield, Ill., 1963.
5. Chiu, Vincent, S. W., and Englert, E., Jr. Gastrointestinal disturbances in myotonia dystrophica. *Gastroenterology*, 1962, *42*, 745–746.
6. Englert, E., Jr., Chiu, and Vincent, S. W. Smooth muscle dysfunction in etiology of gallbladder disease in myotonia dystrophies. *Gastroenterology*, 1962, *42*, 752. (Abst.).
7. Greenstein, A. J., and Kark, A. E. Sigmoid volvulus in muscular dystrophy. *Am. J. Gastroenterol.*, 1972, *57*, 571–577.
8. Harvey, J. C., Sherbourne, D. H., and Siegel, C. I. Smooth muscle involvement in myotonic dystrophy. *Am. J. Med.*, 1965, *39*, 81–90.
9. Hughes, D. T. D., Swann, J. C., Gleason, J. A., and Lee, F. T. Abnormalities in swallowing associated with dystrophia myotonia. *Brain*, 1965, *88*, 1037–1042.
10. Kaufman, K. K., and Heckert, E. W. Dystrophia myotonia with associated sprue-like symptoms. *Am. J. Med.*, 1954, *16*, 614–616.
11. Kerry, R. L., Lee, F., and Ransom, H. K. Roentgenologic examination in diagnosis and treatment of colon volvulus. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 343–348.
12. Kohn, N. N., Faires, J. S., and Rodman, T. Unusual manifestations due to involvement of involuntary muscle in dystrophia myotonica. *New England J. Med.*, 1964, *271*, 1179–1183.
13. Krain, S., and Rabinowitz, J. D. Radiologic features of myotonic dystrophy with presentation of new finding. *Clin. Radiol.*, 1971, *22*, 462–465.
14. Kuiper, D. H. Gastric bezoar in patient with myotonia dystrophy. *Am. J. Digest. Dis.*, 1971, *16*, 529–534.
15. Lee, K. F., Lin, S. R., and Hodes, P. J. New roentgenologic findings in myotonia dystrophy: analysis of 18 patients. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 179–185.
16. Lewitan, A., and Nathanson, L. Roentgen features of muscular dystrophy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1955, *73*, 226–234.
17. Lufs, S. Dystrophia myotonica mit steatorrhea. *Acta med. scandinav.*, 1941, *106*, 557–578.
18. Mascucci, E. F., Canter, H. D., and Katz, S. Involuntary muscle involvement (cardiac and esophageal) in myotonia dystrophica. *M. Ann. District of Columbia*, 1962, *31*, 630–637.
19. Moss, A. A., Goldberg, H. I., and Brotman, M. Idiopathic intestinal pseudo-obstruction. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 312–317.
20. Patterson, M., and Rios, G. Gastrointestinal tract and muscular dystrophy. *Texas Rep. Biol. & Med.*, 1959, *17*, 502–511.
21. Pierce, J. W., Creamer, B., and Mac Dermot, R. Pharynx and oesophagus in dystrophic myotonia. *Gut*, 1965, *6*, 392–395.
22. Schuman, B. M., Rinaldo, J. A., and Darnley, J. D. Visceral changes in myotonia dystrophy. *Ann. Int. Med.*, 1965, *63*, 793–799.
23. Walton, J. N., and Gardner, M. Disorders of Voluntary Muscle. Edited by J. N. Walton. J. & A. Churchill, Ltd., London, 1969, p. 461.
24. Weitzner, S. Changes in pharyngeal and esophageal musculature in oculopharyngeal muscular dystrophy. *Am. J. Digest. Dis.*, 1969, *14*, 805–810.
25. Welsh, J. D., Haase, G. R., and Bynum, T. E. Myotonic muscular dystrophy. *A.M.A. Arch. Int. Med.*, 1964, *114*, 669–679.
26. Zundel, W. S., and Tyler, F. H. Muscular dystrophies. *New England J. Med.*, 1965, *273*, 537–543, 596–60.

TRANSIENT POST-VAGOTOMY DYSPHAGIA: A DISTINCT CLINICAL AND ROENTGENOGRAPHIC ENTITY*

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ABSTRACT:

Dysphagia is a relatively infrequent complication of vagotomy in the postoperative period. The most common form is a transient post-vagotomy dysphagia which requires no treatment other than the temporary exclusion of solid foods. Accurate diagnosis is possible on the basis of clinical history and typical roentgenographic findings. The onset of dysphagia characteristically occurs with the first ingestion of solid foods on the seventh to fourteenth postoperative days. A barium swallow examination reveals persistent tapered narrowing of the terminal three to four centimeters of the esophagus. Most cases are relieved in two to six weeks without clinical or roentgenographic residua. Five cases of transient postvagotomy dysphagia are presented.

VAGOTOMY is a surgical procedure attended by a low morbidity. Dysphagia is a complication of vagotomy in the early postoperative period. Many physicians are unaware of this possibility. When it does occur, therefore, it may be particularly vexing to the surgeon and a source of considerable anxiety for the patient. The most common cause of dysphagia in the early postoperative period is of a transient nature requiring no treatment other than temporary exclusion of solid foods. An accurate diagnosis is therefore desirable since it precludes the necessity of more aggressive forms of intervention. The diagnosis can be suggested on the basis of typical roentgenographic findings.

The occurrence of abnormal esophageal motility following bilateral vagotomy was noted by Cannon[3] in 1906. The first case report of this complication was by Moses[10] in 1947. The reported incidence[1,5] of dysphagia after transabdominal vagotomy has ranged from less than one percent to as high as 14 percent, most series reporting an incidence of three percent or less.[2,11,12]

The dysphagia is first noted between seven and 21 days following surgery;[1] usually at the time solid foods are first introduced into the diet.[5] The dysphagia is not accompanied by pain. The obstruction of the esophagus is rarely complete. Most patients have noted spontaneous recovery within four to eight weeks.

MATERIAL

This series consists of five cases, four males and one female ranging in age from 32 to 71 years. All patients underwent a transabdominal vagotomy for ulcer disease. In two a Bilroth 1 partial gastrectomy was performed and in three a pyloroplasty. Two patients also underwent simultaneous cholecystectomy. The time of onset of dysphagia varied from the seventh to the fifteenth postoperative day. In each case it was painless and associated with the initial ingestion of solid foods. Treatment consisted of temporary dietary exclusion of solid foods in each case and single passage of a number 18 Levin tube in one case (Case 1) and a single bougienage to a number 48 French bougie in another (Case III). The symptoms were completely relieved in all patients in ten to 30 days. The clinical findings are summarized in Table 1.

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TABLE I

CLINICAL SUMMARY

Case	Age	Sex	Operation Performed	Onset of Dysphagia Post-operative	Treatment	Duration of Dysphagia
I	38	F	Cholecystectomy Vagotomy Pyloroplasty	15 days	Passage single Levin tube No. 18 and diet	20 days
II	39	M	Vagotomy Pyloroplasty	10 days	Diet only	30 days
III	36	M	Vagotomy Bilroth I	10 days	Single bougienage to 48 French and diet	14 days
IV	32	M	Vagotomy Bilroth I	12 days	Diet only	10 days
V	71	M	Cholecystectomy Vagotomy Pyloroplasty	7 days	Diet only	13 days

ROENTGENOGRAPHIC FINDINGS

The typical roentgenographic finding is a smooth, tapered, persistent, narrowing of the distal three to four centimeters of the esophagus. The lumen in the narrowed segment measures two to four millimeters in its transverse diameter (Fig. 1, *A–D*; 2, *A* and *B*; and 3, *A* and *B*). There may be slight dilatation of the esophagus proximal to the narrowed segment. The motility proximal to the narrowed segment is normal. The offending bolus of solid food may be identified within the barium column at the site of tapering (Fig. 1*A*; 2*A*; and 3*A*).

ETIOLOGY

This condition is the result of denervation of the lower esophageal segment. Previous investigations have revealed that a high sectioning of the vagus nerves in dogs and other experimental animals results in a disorder of the esophagus which mimics achalasia.[3,4,8] When the vagus nerves are sectioned, the denervated lower esophageal segment fails to relax in response to swallowing. This has been described as post-vagotomy achalasia[11] or cardiospasm.[5,8,14] Although these terms suggest that the lower esophageal segment is denervated and fails to open, they erroneously imply that the motility of the proximal esophagus is also disturbed. This is not the case. Only the motility of the lower esophageal segment is affected. These terms are, therefore, misleading and inappropriate.

Esophageal manometric studies in affected patients[1] substantiate the failure of the lower esophageal segment to relax in response to swallowing. Instead of relaxing, the basal resting pressure of the lower esophageal segment is maintained. Serial manometric examinations reveal that this is a transient phenomenon. When the patient is asymptomatic the manometric examination returns to normal, demonstrating a reduction in the pressure of the lower esophageal segment in response to swallowing.

It is of interest to note that Postlethwait *et al.*[12] performed esophagograms on 15 asymptomatic patients within ten days after vagotomy. Five showed persistent narrowing of the lower esophagus similar to

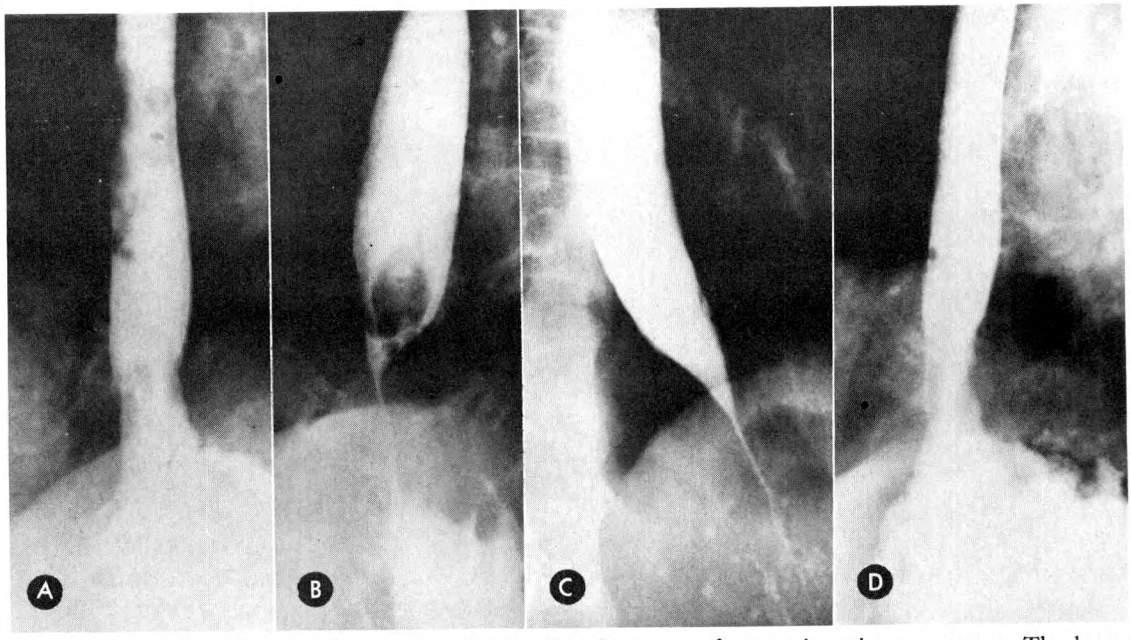

FIG. 1. Case I. (*A*) Thirty-eight year old female. Esophagogram four weeks prior to surgery. The lower esophageal segment is of normal caliber. (*B*) On the 15th postoperative day she experienced dysphagia following ingestion of meat. The bolus is identified as a filling defect. The lower esophageal segment is narrowed with tapered proximal margins. (*C*) The bolus has been dislodged and the narrowed segment is shown to better advantage. (*D*) The dysphagia was relieved in 20 days. This esophagogram was obtained eight months following surgery. The lower esophageal segment functions normally.

that described in patients with dysphagia.

The incidence of dysphagia following transthoracic vagotomy, as reported by Grimson *et al.*,[6] is a rather high 36.8 percent. In the transthoracic approach the vagus nerves are sectioned in a more proximal location when compared to the transabdominal approach. This results in a longer denervated segment and may account for the higher incidence of dysphagia. The available experimental studies of vagus resection, esophageal motility studies and similar transient effects of vagotomy on the stomach and small bowel seem to be sufficient proof that this condition is secondary to vagal denervation alone.

TREATMENT

In most cases no treatment other than the dietary exclusion of solid foods is necessary. A temporary reversion to a liquid and soft diet is all that is required. Within ten to 20 days after the onset of dysphagia the patient can be returned to a full solid food diet in most cases. Although bougienage has been utilized in the treatment by others and was utilized in Case III in this series, most authors concur that such treatment should rarely be necessary. It is reserved for only those patients who fail to respond to the more conservative, simple expedient of exclusion of solid foods. When dilatation is deemed necessary, it is likely that only a single bougienage, utilizing a series of mercury weighted bougies, will be required. This was the treatment given in Case III.

DIFFERENTIAL DIAGNOSIS

Should the symptoms fail to respond to those measures outlined above one should consider the possibility of other, less frequent, disorders that may affect the lower esophageal segment following vagotomy.[2,9,12] These conditions are: periesophageal fibrosis, postoperative esophagitis, and intramural hematoma of the lower esophageal segment.

Periesophageal fibrosis is a rare complication following vagotomy.[2,9] The onset of dysphagia in these cases occurs later than that due to vagal denervation. The dysphagia is accompanied by pain referred to the lower esophageal region. These symptoms are first noted with the ingestion of solid food but may be progressive. Subsequently, the ingestion of soft foods and even liquids may also be associated with dysphagia. The esophagogram in periesophageal fibrosis reveals a narrowing of the distal esophagus with delayed emptying of the esophagus, quite similar to that seen in vagal denervation. At esophagoscopy or bougienage there is a marked increase in resistance to passage of the instrument through the narrowed zone. Such resistance is usually not encountered in vagal denervation.[5] At esophagoscopy the mucosa in the involved segment is normal, as it is in the vagal denervation. Esophageal motility studies are able to distinguish the dysphagia of periesophageal fibrosis from that associated with vagal denervation.[1,7] These studies demonstrate a relatively normal relaxation of the lower esophageal segment in response to swallowing. This is in contradistinction to vagal denervation wherein the resting pressure is maintained in response to swallowing. Repeated dilatations are necessary in order to overcome the periesophageal fibrosis and restore normal swallowing. On occasion the fibrosis has been so extensive that an operation is required to move the fibrotic collar about the lower esophageal segment.[9]

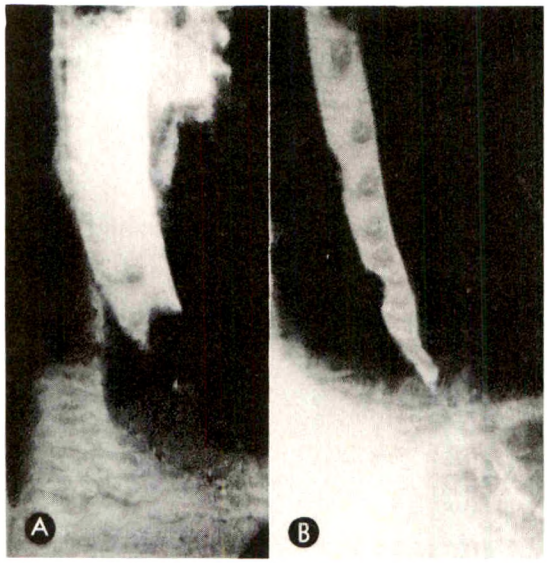

Fig. 2. Case II. (*A*) Thirty-nine year old male first noted dysphagia on the tenth postoperative day following the ingestion of solid food. The esophagogram was obtained on the 35th postoperative day when symptoms increased following the ingestion of meat. Note the bolus just proximal to the narrowed distal segment. A thin trickle of barium passes beyond the bolus. (*B*) Case III. A 36 year old male who first noted dysphagia on the tenth postoperative day. The esophagogram reveals a narrowed distal segment with tapered proximal margins.

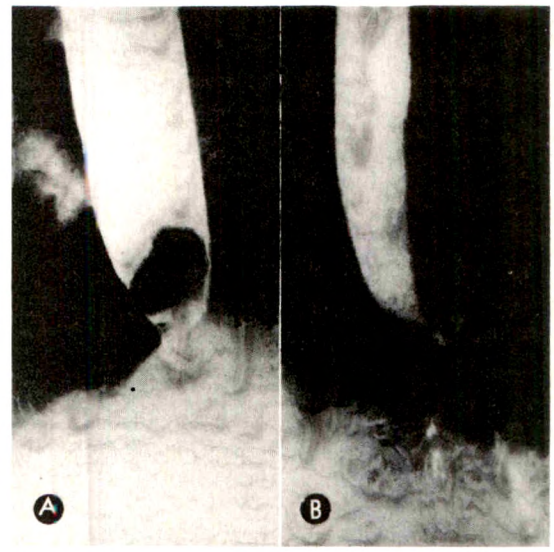

Fig. 3. Case IV. (*A*) A 32 year old male noted dysphagia following the ingestion of meat on the twelfth postoperative day. A lateral view of the barium filled esophagus reveals the offending bolus just proximal to the tapered narrowing of the distal segment. (*B*) Case V. A 71 year old male noted dysphagia seven days following a transabdominal bilateral vagotomy, pyloroplasty, and cholecystectomy. The distal segment of the esophagus is narrowed with tapered proximal margins.

Dysphagia due to postoperative esophagitis is even more infrequent.[2,7] This condition probably results from a combination of operative manipulation and postoperative nasogastric intubation. The symptoms are said to arise early in the postoperative

course prior to the initiation of solid food diet, and dysphagia is accompanied by pain. Esophagograms demonstrate narrowing of the distal esophageal segment. On endoscopy the esophageal mucosa is inflamed, thus distinguishing this condition from other causes of post-vagotomy dysphagia. The treatment is similar to other forms of esophagitis.

An intramural hematoma of the lower esophageal segment has been described as a cause of dysphagia following transabdominal vagotomy.[13] Dysphagia was first noted on the seventh postoperative day with solid foods but progressed very rapidly to a total inability to swallow even liquids. Esophagoscopy and studies with barium demonstrated dilatation of the proximal esophagus and a tapered narrowing of the distal segment. At operation, a mass was found in the wall of the esophagus which proved to be an organizing hematoma. The progressive nature of the dysphagia and the rigid narrowing of the lower esophageal segment is identified by endoscopy, differentiate an intramural hematoma from transient vagal denervation. It is unlikely that one could distinguish between an intramural hematoma and periesophageal fibrosis short of surgical intervention.

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REFERENCES

1. ANDERSON, H. A., SCHLEGEL, J. F., and OLSEN, A. M. Post-vagotomy dysphagia. *Gastrointest. Endosc.*, 1966, *12*, 13–18.
2. BRUCE, M., and SMALL, W. P. Dysphagia following vagotomy. *Roy. Coll. Surg. Edinburgh J.*, 1958–59, *4*, 170–178.
3. CANNON. W. B. Motor activities of stomach and small intestine after splanchnic and vagus section. *Am. J. Physiol.*, 1906–07, *17*, 429–442.
4. CARVETH, S. W., SCHLEGEL, J. F., CODE, C. F., and ELLIS, F. H., JR. Esophageal motility after vagotomy, phrenicotomy, myotomy and myomectomy in dogs. *Surg., Gynec. & Obst.*, 1962, *114*, 31–42.
5. DAGRADI, A. E., STEMPIEN, S. J., SEIFER, H. W., and WEINBERG, J. A. Terminal esophageal (vestibular) spasm after vagotomy. *A.M.A. Arch. Surg.*, 1962, *85*, 955–968.
6. GRIMSON, K. S., BAYLIN, G. J., TAYLOR, H. M., HESSER, F. H., and RUNDLES, R. W. Transthoracic vagotomy: effects in 57 patients with peptic ulcer and clinical limitations. *J.A.M.A.*, 1947, *134*, 925–932.
7. GUILLORY, J. R., JR., and CLAGETT, O. T. Post-vagotomy dysphagia. *Surg. Clin. North America*, 1967, *47*, 833–839.
8. HARRIS, J., and MILLER, C. M. Cardiospasm following vagotomy. *Surgery*, 1960, *47*, 568–570.
9. HOYUMPA, A. M., JR., MORRISON, J. T., and MOSKOWITZ, M. Post-vagotomy dysphagia: report of case with periesophageal fibrosis. *Am. J. Gastroenterol.*, 1972, *57*, 133–138.
10. MOSES, W. R. Critique on vagotomy. *New England J. Med.*, 1947, *237*, 603–610.
11. PIERANDOZZI, J. S., and RITTER, J. H. Transient achalasia: complication of vagotomy. *Am. J. Surg.*, 1966, *111*, 356–358.
12. POSTLETHWAIT, R. W., KIM, S. K., and DILLON, M. L. Esophageal complications of vagotomy. *Surg., Gynec. & Obst.*, 1969, *128*, 481–488.
13. RABIAH, F. A., and ELLIOTT, H. B. Intramural hematoma of esophagus: unusual complication of vagotomy. *Am. J. Digest. Dis.*, 1968, *13*, 925–928.
14. WILCOX, R. S. Cardiospasm following vagotomy, *Am. J. Surg.*, 1950, *79*, 843–847.

VOL. 125, NO. 4

LOCALIZATION WITH THE EMI SCANNER*

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ABSTRACT:

Accurate localization of lesions seen on computerized tomographic scans obtained with the EMI unit is often difficult due to a paucity of reliable landmarks and to varying head angulation. A simple and accurate system of transposing the location of any particular lesion to a roentgenogram obtained with the scanner tube is described.

PRECISE anatomic localization of cerebral masses on transverse sections derived from computerized tomographic brain scans (EMI) is often difficult. We have devised a method to localize the plane and locus of such lesions.

The anatomic localization of lesions on the computerized tomographic (CT) scan depends on their relation to anatomic landmarks within a given section. The most readily available points of reference are the ventricular system and the cisterns. In our experience, this approach has been unsatisfactory, especially in patients with lesions measuring less than 2 cm in diameter.

One of the difficulties in precise localization on CT scans is the absence of calvarial landmarks that can serve as reference points on any single tomographic section. Structures at the base of the skull are poorly defined because the computer algorithm cannot handle major tissue density transitions. With CT brain scanning, transverse sections of the brain are obtained in relation to Reid's baseline (infraorbital-meatal line). The head is angled at 0° to this line when the areas of interest are the orbits or supratentorial structures. If the posterior fossa is to be examined, the head is angled at +15 to +20°. The true angle of inclination of the skull is often uncertain because anatomic landmarks are obscured by the head box. In addition, the angulation may vary from that originally intended or measured because of motion by the patient during the examination. Such variation may alter the apparent site of the lesion on the scan and result in misleading information (Fig. 1).

The problem of localization of small lesions is resolved by obtaining lateral or frontal and lateral skull roentgenograms while the patient is in the scanning position. The roentgenogram is obtained in the following manner. A vacuum cassette,* loaded with Par-speed film placed between two 8×10 high-speed screens is taped to the side of the plexiglass water box opposite the tube (Fig. 2). The gantry is rotated 90° from its starting position, thus placing the tube perpendicular to the sagittal plane of the skull (Fig. 3). The index switch at the rear of the motor control is placed in the off position. Bypass test switches in the computer rack labelled "computer ready" and "scanner at start" are set in the down or bypass position. The scanner is started at the 33 mA and 120 KV settings used for routine scanning. The tube is permitted two linear passes and the scan is then aborted. During these two passes the gantry does not rotate. The chair or table is then shifted 3.5 cm and the scanner is started again and aborted after two passes. This process is repeated three more times to obtain a complete lateral exposure of the skull (Fig. 4*A*). The same procedure may be repeated in the frontal plane if desired. For adequate exposure in this projection, three passes of the tube are required at

* Cronex vacuum bags for low-dose mammography (Dupont).

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† Academic Trainee in Diagnostic Neuroradiology. Supported by grant NSC5603 from the National Institutes of Health, United States Public Health Service.

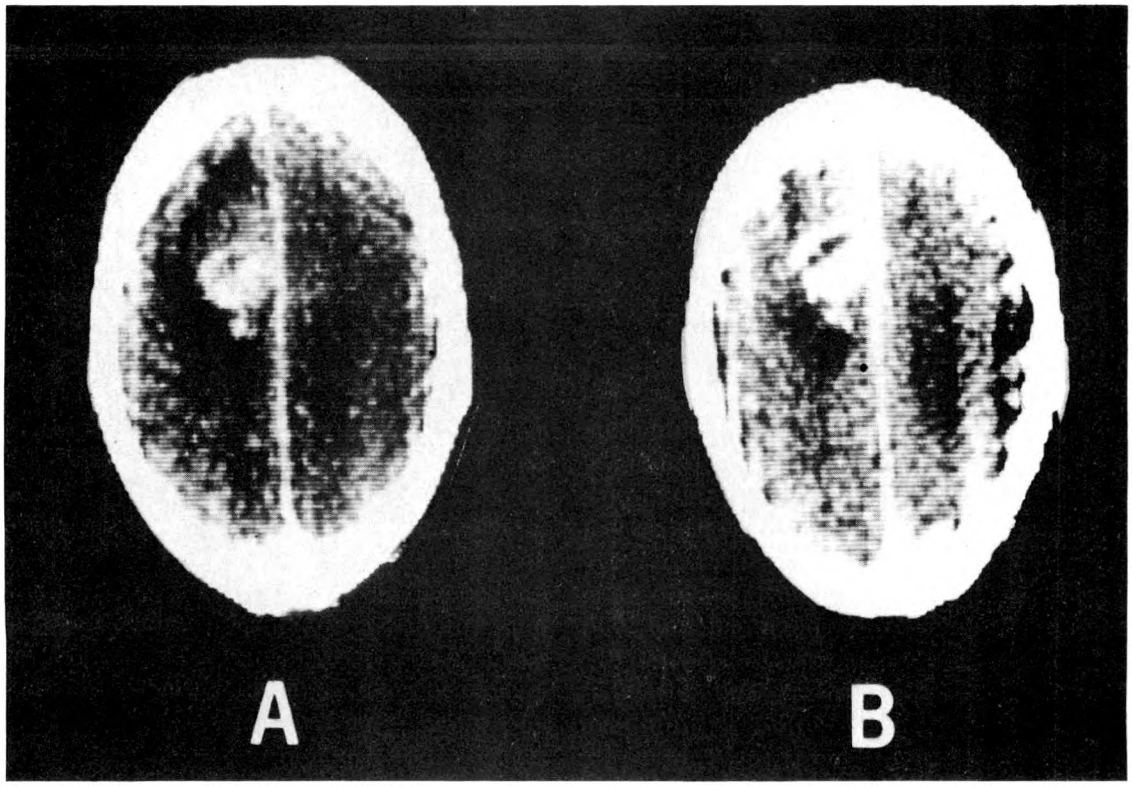

FIG. 1. A 51 year old woman with known left frontal glioma. (*A*) Scan performed with the head angled at 0° to Reid's baseline. (*B*) Scan performed with head angled at 20° to Reid's baseline. Note the apparent anterior shift of the lesion when angulation of the head is increased. Both scans are performed with contrast enhancement.

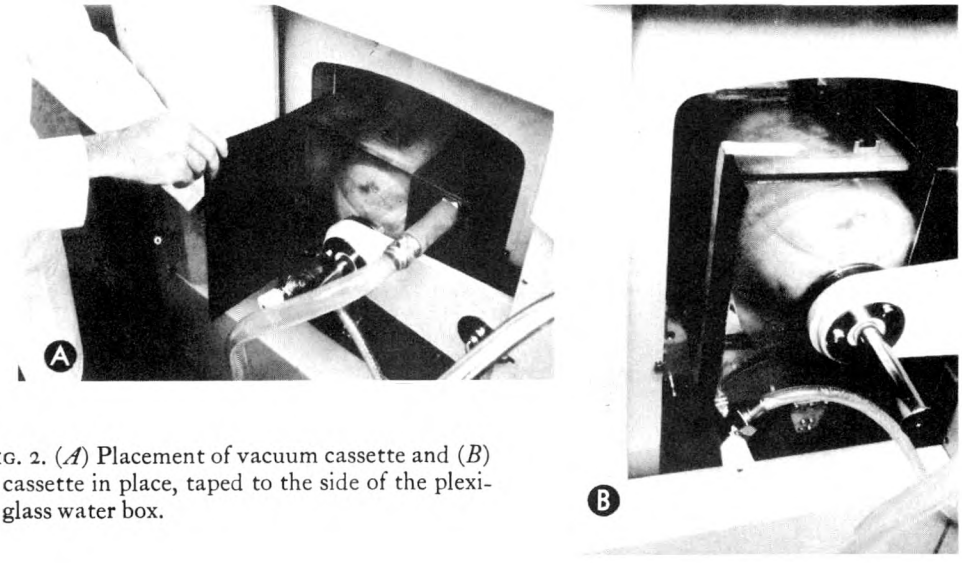

FIG. 2. (*A*) Placement of vacuum cassette and (*B*) cassette in place, taped to the side of the plexiglass water box.

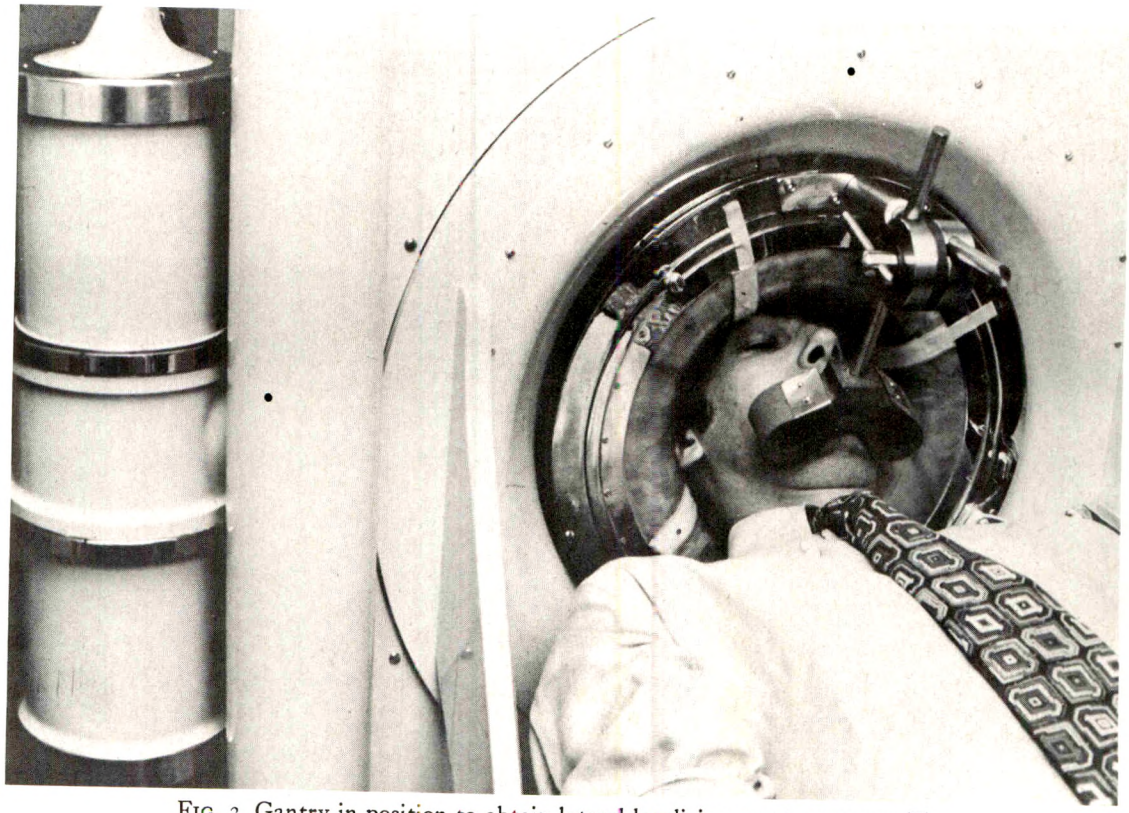

Fig. 3. Gantry in position to obtain lateral localizing roentgenogram. The roentgen-ray tube is at 90° to the sagittal plane of the skull.

each 3.5 cm interval (Fig. 4*B*).

The resultant roentgenogram of the skull is not of sharp detail because of the linear motion of the tube and the large focal spot. The vacuum cassette permits film abutment to the edge of the scanning-box so that calvarial landmarks corresponding to the plane of the first section are included. Ordinary film cassettes are unsatisfactory because there is a distance of approximately 1 cm between the edge of the film and the edge of the cassette.

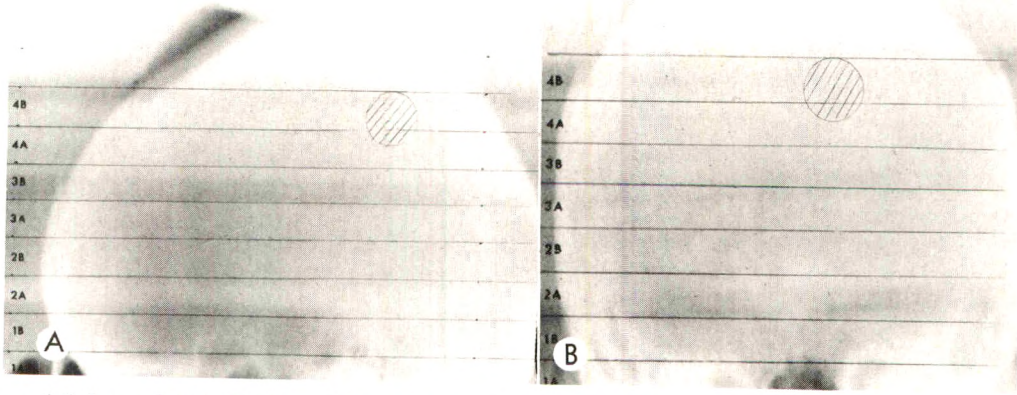

Fig. 4. (*A*) Lateral and (*B*) frontal localizing roentgenograms with parallel lines drawn to represent scan intervals. Cross-hatched area represents a localized lesion described in Figure 5, *A* and *B*.

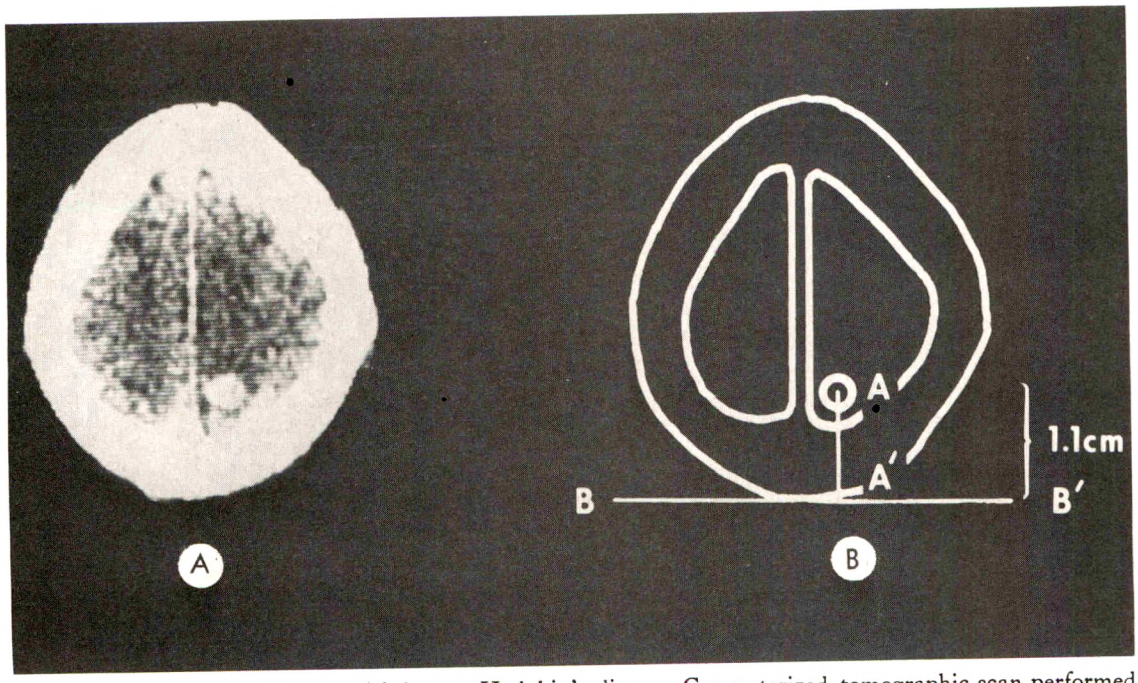

FIG. 5. (*A*) A 28 year old man with known Hodgkin's disease. Computerized tomographic scan performed with contrast enhancement demonstrated on section 4B a 1.5 cm area of increased uptake in the medial aspect of the right occipital lobe. At surgery this lesion proved to be a toruloma. (*B*) Diagram of scan 4B. A line (A-A′) is drawn from the center of the lesion perpendicular to the line (B-B′) drawn tangential to the posterior outer table of the calvarium. The distance A-A′ measures 1.1 cm. This distance is multiplied by the 3.3 Polaroid minification factor and is transposed to the localization film on plane 4B. On the frontal localizing film the parasagittal location of the lesion is transposed from the scan to the film in a similar manner.

The film magnification factor in the cephalocaudad direction is 1.08. Parallel lines drawn from the base of the roentgenogram to represent each 1.3 cm section are therefore drawn at 1.4 cm intervals (1.08 × 1.3). When the 0.8 cm cone is employed, the resultant distance between constructed parallel lines is 0.87 cm (1.08 × 0.8) (Fig. 4). Clear plastic overlays with the lines drawn at appropriate intervals will facilitate this process. An alternative is to place a thin sheet of plexiglass with tantalum wires embedded at appropriate spacing between the cassette and the water box so that the lines are imposed upon the roentgenogram at the time of exposure. On our unit the minification of the Polaroid image in relation to the normal anteroposterior dimension of the head is 3.3. This factor is used in transposing measurements from the Polaroid image to the localization film (Fig. 5, *A* and *B*).

The described technique has been performed on a series of patients who subsequently underwent surgical intervention for lesions measuring as little as 1 cm. The roentgenograms have proved to be accurate and invaluable in enabling the surgeon to localize the lesion.

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BONE TUMOR IMAGING BY SCINTIGRAPHY OF THE SKELETON, MARROW RETICULOENDOTHELIAL SYSTEM, AND THE PROLIFERATIVE TISSUE*

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ABSTRACT:

Paralleled clinical studies of radioisotopic imaging of tumor metastases in the bone were undertaken with Tc^{99m} pyrophosphate, Ga^{67} citrate, and Tc^{99m} sulfur colloid. All three were capable of anticipating the bone roentgenograms. A critical analysis suggested that positive delineation with Ga^{67} citrate would sometimes be superior to the others. This deserves further investigation. Clinically the combined use of Tc^{99m} pyrophosphate with Ga^{67} for the chest areas and extremities or with Tc^{99m} colloid for the pelvic and abdominal areas would be more fruitful in early detection and exact localization of bone metastases.

SCINTIGRAPHIC delineation of bone metastases of cancer may be achieved in different ways, each involving a different mechanism of radioisotopic deposition: (a) skeletal imaging with increased deposition in areas of active bone reaction and increased blood supply or absence of deposition in areas of bone defect;[10] (b) reticuloendothelial (RE) imaging of the bone marrow in which the RE cells are displaced by the expanding tumor cells resulting in a reduced to absent radioisotopic concentration, indicative of tumor growth;[8] and (c) the so-called tumor imaging with various tumor affinitive radiopharmaceuticals with which a tumor can be delineated with or without accompanying skeletal imaging.[13]

Employing parallel studies with different radiopharmaceuticals, the present communication is intended to obtain insights into the prospects and possible limitations of these bone tumor imagings. At this time, bone tumor imaging with Ga^{67} citrate appears to be the easiest and most prospective because of its tumor affinity as well as its affinity for bone.[14,15] Such a technique would be of use in precisely delimiting fields of teletherapy or planned surgery.[13]

MATERIAL AND METHOD

Eighteen patients with proved or suspected secondary bone neoplasms were studied. All were examined at the Hospital of the Research Institute for Tuberculosis and Cancer, Tohoku University, Sendai, Japan. In most cases, the histopathological diagnoses were established from surgical biopsy or sputum cytology. In five cases, the diagnosis of malignancy was demonstrated by roentgenological signs or other clinical and laboratory findings.

Tc^{99m} pyrophosphate was prepared according to the method of Cohen *et al.*[4] The preparation of vial kits containing sodium pyrophosphate and tin chloride was employed (Tc^{99m} PRP‡). One to two hours after intravenous administration of 3–5 mCi of this agent, the lesions were scanned or scintigraphed.

Ga^{67} citrate‡ was intravenously injected, and the bone tumor imaging was carried

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out two days later. The dosage used was 1.5–2.0 mCi.

Tc^{99m} bleomycin. Radioisotopic labeling of bleomycin was performed as described by Mori *et al.*[11,12] Briefly, 15 mg of bleomycin§ were dissolved in the mixture of 5 ml Tc^{99m} pertechnetate and 0.05 ml dilute tin chloride (5 mg/ml), and it was allowed to react for five minutes at room temperature. Excessive tin was removed by the addition of ascorbic acid. The final pH was adjusted to 7.0 with bicarbonate solution. Millipore filtration with a sheet of filter membrane of 0.22 μm pore size removed the precipitated tin and contaminating microorganisms. This labeling was proved to be 96 percent efficient. The dosage used was 3–10 mCi. The amount of bleomycin was 5 mg or less. Tumor imaging was started one hour following intravenous administration.

Tc^{99m} sulfur colloid‡ was prepared with $Na_2S_2O_3$ and HCl and was stabilized with mannitol in vial kits. Bone marrow reticuloendothelial (RE) imaging was commenced 15 minutes after the administration of 10 mCi Tc^{99m} colloid.

Scintigraphy and scintiscanning. Radioisotopic imaging was carried out with a 5×2 inch NaI crystal rectilinear scintiscanner with a color adapter‖ and a scinticamera.¶

RESULTS

The primary diseases, skeletal lesions, roentgenographic findings, and the results of radioisotopic bone lesion imagings are summarized in Table 1. The skeletal, marrow RE, and proliferative tissue imagings were all found capable of detecting lesions before skeletal roentgenograms. Ga^{67} scanning has already been shown to possess this ability.[7] Several cases with comparable parallel studies confirmed our concept of proliferative tissue imaging[15] and will be illustrated.

§ Nihon Kayaku Pharmaceuticals, Tokyo, Japan.
‖ Toshiba Electric, Tokyo, Japan.
¶ Pho/Gamma HP, Nuclear-Chicago, Chicago.

Case 10, T.O. This patient had a three year history of lymphoepithelioma malignum treated by three courses of radiation therapy for recurrent neck metastases. When seen by us, there was no roentgenographic abnormality except for a slight sclerotic change in the right iliac crest (Fig. 1*A*). Marrow RE imaging with Tc^{99m} colloid demonstrated cold regions in the third and fourth lumbar vertebrae and a suspected cold area in the right iliac crest (Fig. 1*B*). A Tc^{99m} PRP scintigram was positive in these regions (Fig. 1*C*). A similar positive scintigram was attained with Ga^{67} citrate (Fig. 1*D*). In this case, however, tumor outgrowths extended beyond the vertebral column. Thus, all three different scintigraphs were seen capable of antedating roentgenograms. A second marrow RE imaging was performed two months later, and it clearly demonstrated further growth of the metastatic tumors (Fig. 1*F*). This was then confirmed by roentgenography (Fig. 1*E*) and by autopsy.

Case 18, Y.K. This patient had skeletal dissemination of leiomyosarcoma originating from the stomach. The bony metastases to the pelvic region were unusual. There was a massive osteolytic lesion involving the right ilium near the sacroiliac joint, as well as osteoblastic change in the sacrum (Fig. 2*A*). These regions were positive with Ga^{67} scintiscanning (Fig. 2*B*), although the right iliac lesion was not entirely demonstrated by skeletal imaging (Fig. 2*C*). The tumor mass in this region became palpable as it grew beyond the skeleton. As the patient was very debilitated, a marrow RE imaging was not performed. These results seemed to agree with our previous results with experimental bone tumors, in which we demonstrated a clear-cut discrimination in tumor imaging by bone-seeking agents.[15]

DISCUSSION

The ultimate purposes of bone tumor imaging are (1) diagnosis of exact localization and extent of the tumor, (2) early diag-

TABLE I

SUMMARY OF PATIENTS STUDIED: PRIMARY CANCER, ROENTGENOGRAPHIC DATA, AND RESULTS OF RADIOISTOPIC INVESTIGATIONS

Case No.	Name	Age and Sex	Primary Cancer	Bone Lesions Studied	Roentgenograms	Tc^{99m} Pyrophosphate	Tc^{99m} Colloid	Ga^{67} Citrate	Comments
1.	F.S.	48 F	Breast cancer	Thoracic and lumbar vertebrae	Yes,* blastic	Yes*			Autopsied
2.	M.S.	43 F	Lung cancer (adenocarcinoma)	Left humerus	Yes, lytic	Yes			
3.	S.A.	44 F	Stomach cancer (scirrhous)	Thoracic and lumbar vertebrae	No*	Yes			Gastrectomized
4.	K.Y.	70 M	Lung cancer	Right metatarsus	Yes, lytic	Yes	Yes		Died 6 months later
5.	S.C.	62 M	Lung cancer	Lumbar vertebrae	No	Yes	Yes		Died 1 month later
6.	T.I.	76 M	Lung cancer	Lumbar vertebrae	Yes, blastic	Yes	Yes	Yes	
7.	K.H.	60 F	Lung cancer (adenocarcinoma)	Mandible	Yes, lytic	Yes	Yes	Yes	Died 1 month later
8.	I.M.	65 F	Lung cancer	Left ilium	No	Yes	Yes		Died 2 months later
				Ribs	Yes, lytic	Yes	No		
9.	B.M.	71 M	Lung cancer	Lumbar vertebrae	No	Yes	Yes		Died 2 months later; autopsied
10.	T.O.	38 M	Lymphoepithelioma malignum	Lumbar vertebrae	Yes	Yes	Yes	Yes	Died 2 months later, autopsied (Fig. 1)
11.	K.O.	60 M	Lung cancer (adenocarcinoma)	Left femur	Yes, lytic	Yes	Yes		Died 2 months later
				Lumbar vertebrae	No	Yes	Yes		
12.	S.K.	78 M	Lung cancer (squamous cell carcinoma)	Lumbar vertebrae	No	Yes	Yes		Died 3 months later
13.	S.T.	58 M	Pancreas cancer	Left ilium	No	No	Yes		Died and autopsied
14.	A.O.	53 F	Breast cancer (adenocarcinoma tubulare)	Pelvis	No	No	No	No	Radical mastectomy and irradiation
									Alive 1½ years after initial study
15.	T.S.	73 M	Esophageal cancer	Left shoulder	Yes, lytic	Yes		Yes	Died; autopsied
16.	T.H.	80 M	Lung cancer	Ribs	Yes, lytic	Yes		Yes	Died 2 months later. Tc^{99m} bleomycin in place of Ga^{67}
17.	K.G.	45 F	Breast cancer	Pelvis		No			Mastectomy and irradiation 3 years before. Recurrence to skin and mediastinum, irradiated
				femur, tibia	No	No			
18.	Y.K.	60 F	Leiomyosarcoma of stomach	Pelvis	Yes, lytic	Yes and no		Yes	Skin metastasis. Biopsied (Fig. 2)
				Femora		Yes		Yes	Autopsied 7 months later

* Abnormalities: (+) Yes (−) No.

nosis of distant metastases, and (3) early detection or screening of bone tumor in a patient.

Tumor imaging depends on two areas of radiopharmaceutical deposition: in a tumor *per se*, and in its surroundings. The tumor itself consists of (a) proliferative tissue, (b) necrotic tissue, and (c) supporting stroma. With classical radiostrontium bone tumor imaging, however, the principal mechanism for deposition is (d) the dynamic metabolism of bone calcium and phosphate reacting vigorously to the presence of the invading tumor.

Incorporation into the osteogenesis of osteogenic sarcoma[10] and the tumor affinity for Sr^{87m} in selected extraosseous tumors[3,17] are classified in category a. Skeletal imaging with Tc^{99m} pyrophosphate undoubtedly falls in category d. Marrow reticuloendothelial (RE) imaging combines a, b, and c as it depends on the dispersion of the RE cells by the tumor mass. Ga^{67} imaging is different because it possesses so-called tumor affinity as well as an affinity for the reactive bone formation, *i.e.*, categories a and d.[1,5] This feature of Ga^{67} can be contrasted to the pure tumor-affinitive imaging with I^{131}-iododeoxyuridine in experimental bone tumors, in which the tumor *per se* was imaged but not bone in reaction to tumor invasion.[16]

As demonstrated previously, all three imagings of the skeletal system, marrow RE system, and proliferative tissue are capable of anticipating roentgen changes in

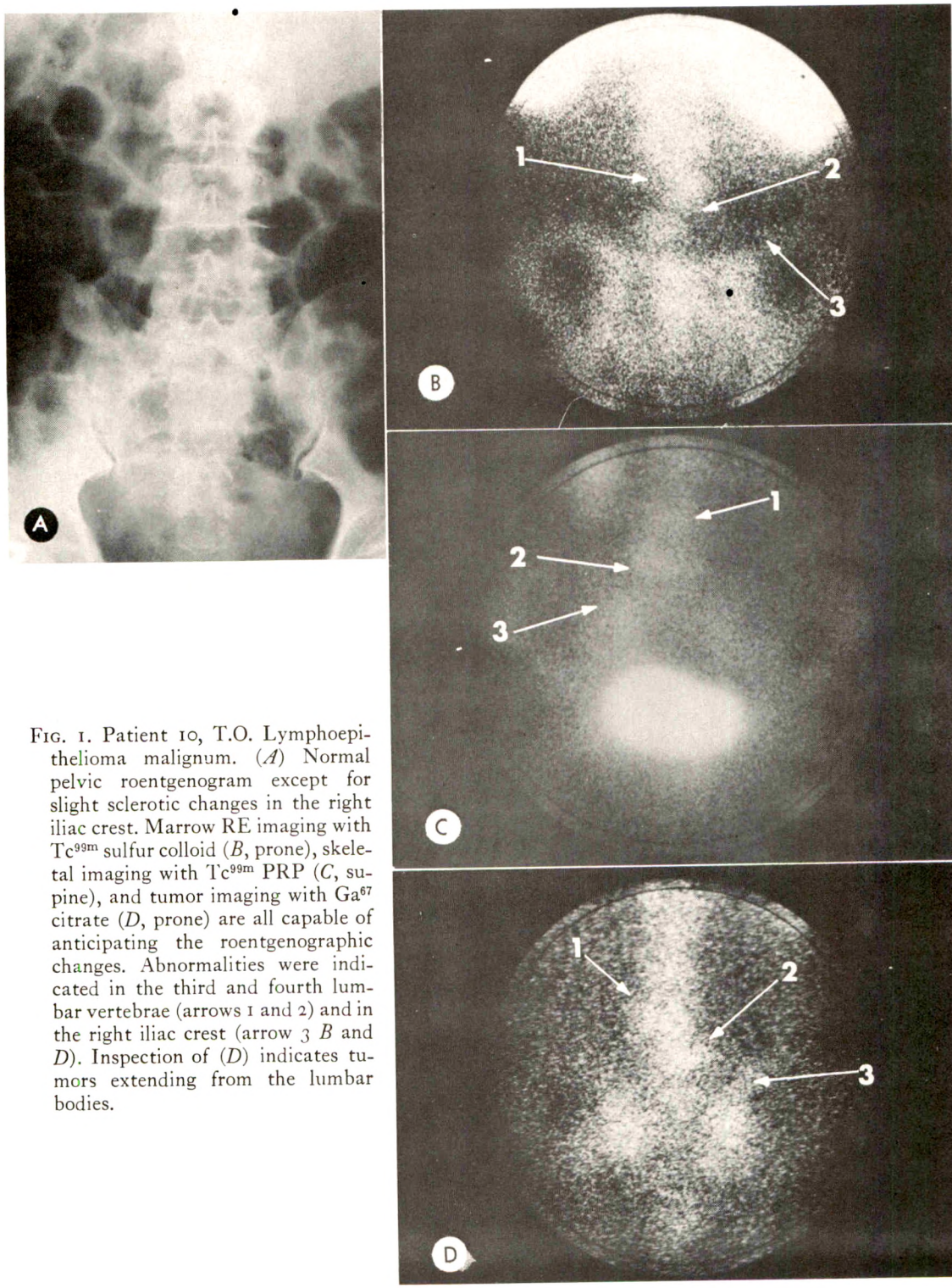


FIG. 1. Patient 10, T.O. Lymphoepithelioma malignum. (*A*) Normal pelvic roentgenogram except for slight sclerotic changes in the right iliac crest. Marrow RE imaging with Tc^{99m} sulfur colloid (*B*, prone), skeletal imaging with Tc^{99m} PRP (*C*, supine), and tumor imaging with Ga^{67} citrate (*D*, prone) are all capable of anticipating the roentgenographic changes. Abnormalities were indicated in the third and fourth lumbar vertebrae (arrows 1 and 2) and in the right iliac crest (arrow 3 *B* and *D*). Inspection of (*D*) indicates tumors extending from the lumbar bodies.

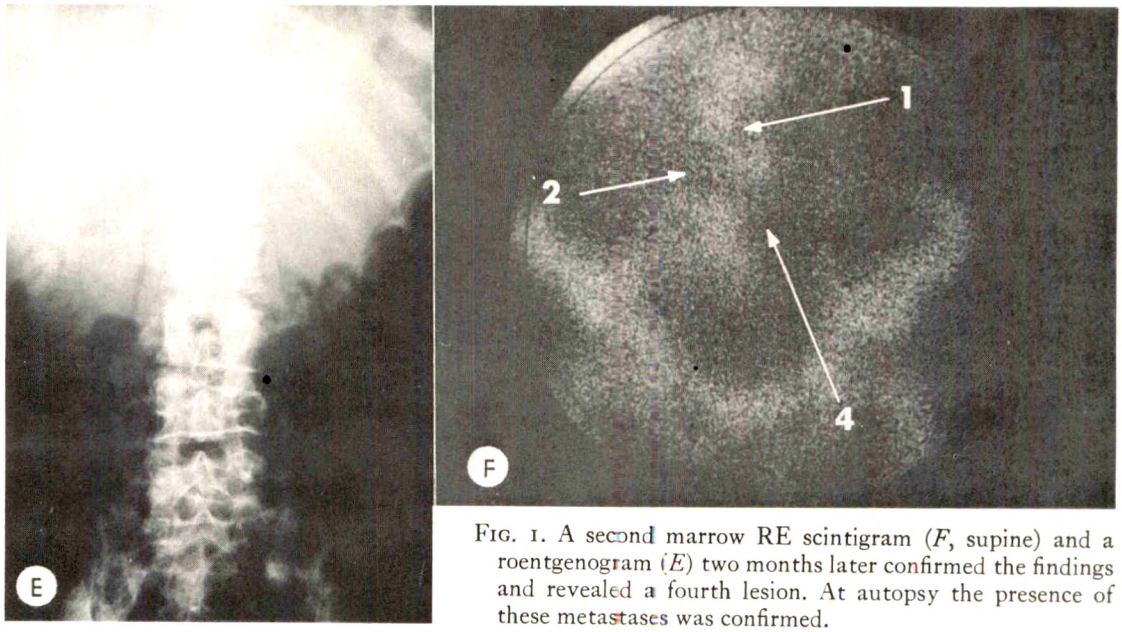
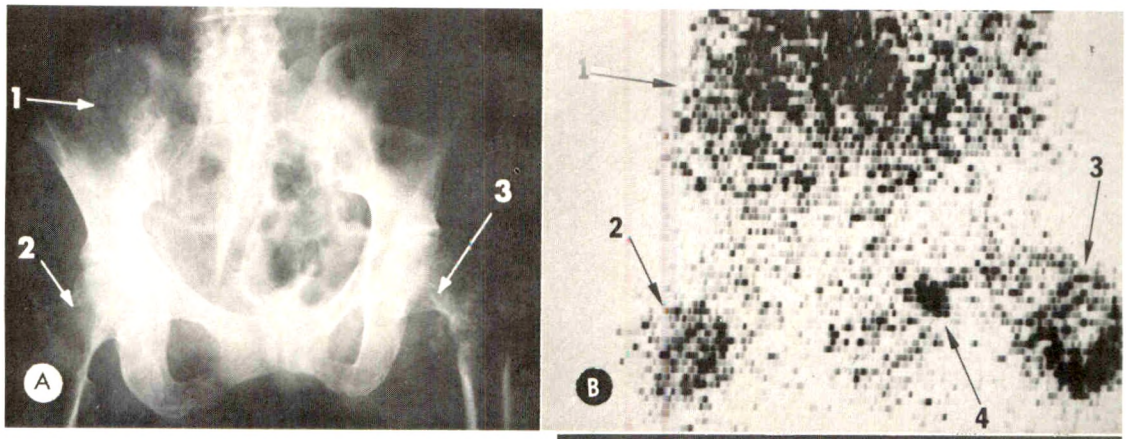


Fig. 1. A second marrow RE scintigram (*F*, supine) and a roentgenogram (*E*) two months later confirmed the findings and revealed a fourth lesion. At autopsy the presence of these metastases was confirmed.



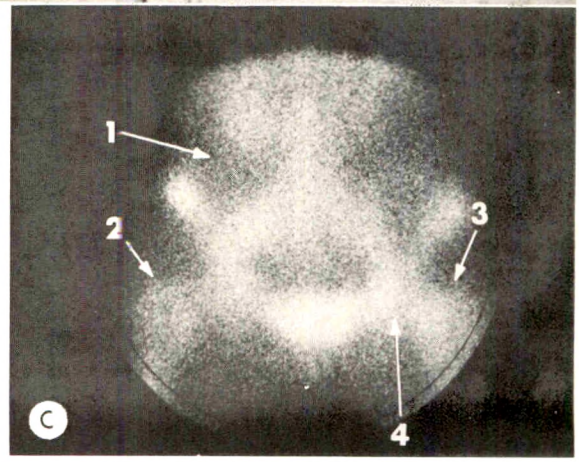


Fig. 2. Patient 18, Y.K. Leiomyosarcoma. (*A*) Osteolysis is seen in the right ilium (arrow 1), and in both proximal femurs (arrows 2 and 3). The right sacroiliac joint and the lower lumbar vertebrae are sclerotic. (*B*) Ga^{67} scan was positive in these regions, including the right iliac mass. It preceded the roentgenographic changes in showing the left pubic lesion (arrow 4). (*C*) The skeletal imaging agreed with results of the Ga^{67} scan except for the failure in demonstrating the tumor mass in the right ilium.

bone.[7] Thus, an earlier diagnosis of distant metastases to the skeletal system can be achieved by any of these three techniques. Probably the earliest is clearly demonstrated by marrow RE imaging in the diagnosis of an experimental hematogeneous metastatic bone carcinoma,[8] as well as with Ga^{67} tumor imaging.[15] It avoids waiting for development of reactive bone formation when the metastasis arises hematogeneously, and trabecular or cortical bone is involved somewhat later.

The osseous reaction to tumor invasion consists of osteoblastic as well as osteolytic changes,[9] and this feature is responsible for the increased deposition of radiostronium[10] and most likely Tc^{99m} pyrophosphate. These techniques are sensitive once the reactive metabolism of calcium and phosphate attains certain levels. The necropsy radioassay data on giant cell tumor by Andrews *et al.*[1] verified that Ga^{72} deposition was about seven times greater in the portion of bone adjacent to the tumor than in the tumor *per se*. In our experience, the use of Tc^{99m} pyrophosphate makes it feasible to obtain an entire skeletal view within a short period of time, with increased accuracy. Improved resolution of the gamma camera has also contributed to the accuracy.

Skeletal imaging has been reported to show false negativity as high as 30 percent in a representative series by Scheer *et al.*[19] When the tumor growth is too rapid, such as in reticulum cell sarcoma, there may not be appreciable bone reaction or dynamic changes in calcium metabolism,[2] and the lesion will remain cold with Tc^{99m} pyrophosphate or radiostrontium scintigrams. Enlarging skeletal tumors may also present negative scans unless they contain enough osteoblastic components. Recently, Sr^{87m} has been noted to have a tumor affinity for several soft tissue tumors.[3,17,18] Although the mechanism is uncertain, this agent cannot but be surpassed by Ga^{67}, because the latter is, so to speak, a "universal" tumor imaging agent.[6,7] Ga^{67} is easily obtained from commercial sources, easy to administer, has a convenient half-life of 78 hours, etc. Radiation therapists are encouraged that Ga^{67} is capable of fully delineating an entire tumor mass except for necrotic portions. Fields of teletherapy can be planned in the absence of roentgenographic confirmation. It could be of great help in planning surgery. This agent, however, has the drawback of being excreted into the gastrointestinal tract, and it may give rise to false positivity; this may be eliminated by cleansing enemas.[7] Tc^{99m} pyrophosphate is excreted through the kidneys and accumulates within the urinary bladder. This frequently interferes with pelvic imaging. With these merits and limits in mind, we recommend the following imaging schemes for the diagnosis of bone metastases: (1) Tc^{99m} pyrophosphate and Ga^{67} for imaging of the chest and the extremities; and (2) Tc^{99m} pyrophosphate and Tc^{99m} colloid for imaging of the pelvis and abdomen. For the purpose of surveying possible bone metastases, skeletal imaging with Tc^{99m} pyrophosphate would be the easiest, most time-saving and fruitful study.

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REFERENCES

1. Andrews, G. A., Root, S. W., and Kerman, H. D. Study of gallium^{72}. VI. Clinical studies with gallium^{72}. *Radiology*, 1953, *61*, 570–588.
2. Charkes, N. D., Young, I., and Sklaroff, D. M. Pathologic basis of strontium bone scan. *J.A.M.A.*, 1968, *206*, 2482–2488.
3. Chaudhuri, T. K., Chaudhuri, T. K., Go, R. T., Taube, R. R., and Christie, J. H. Uptake of ^{87m}Sr by liver metastases from carcinoma of colon. *J. Nuclear Med.*, 1973, *14*, 293–294.
4. Cohen, Y., Perez, R., Henry, R., Bardy, A., and Pannecière, C. Utilisation du pyrophosphate de sodium marque par le technetium 99m dans la scintigraphie du squelette. *Compt. Rend. Acad. Sc.*, Paris, 1972, *275*, 1719–1721.
5. Dudley, H. C., Imirie, G. W., Jr., and Istock, J. T. Deposition of radiogallium (Ga^{72}) in proliferative tissues. *Radiology*, 1950, *55*, 571–575.

6. Edwards, C. L., and Hayes, R. L. Tumor scanning with ^{67}Ga citrate. *J. Nuclear Med.*, 1969, *10*, 103–105.
7. Ito, Y., Okuyama, S., Awano, T., Takahashi, K., Sato, T., and Kanno, I. Diagnostic evaluation of ^{67}Ga scanning of lung cancer and other diseases. *Radiology*, 1971, *101*, 355–362.
8. Ito, Y., Okuyama, S., Suzuki, M., Sakurai, M., Sato, T., and Takagi, H. Bone marrow scintigraphy in early diagnosis of experimental metastatic bone carcinoma. *Cancer*, 1973, *31*, 1222–1230.
9. Milch, R. A., and Changus, G. W. Response of bone to tumor invasion. *Cancer*, 1956, *9*, 340–351.
10. Moon, N. F. Skeleton. In: Principles of Nuclear Medicine. Edited by H. N. Wagner, Jr. W. B. Saunders Company, Philadelphia, 1968, pp. 703–721.
11. Mori, T., Hamamoto, K., and Torizuka, K. ^{99m}Tc labeled bleomycin for tumor diagnosis: experimental and clinical application. *Jap. J. Clin. Radiol.*, 1973, *18*, 201–205.
12. Mori, T., Hamamoto, K., Onoyama, Y., and Torizuka, K. Tumor imaging after administration of ^{99m}Tc labeled bleomycin. To be published.
13. Okuyama, S., Ito, Y., Awano, T., and Sato, T. Prospects of ^{67}Ga scanning in bone neoplasms. *Radiology*, 1973, *107*, 123–128.
14. Okuyama, S., Ito, Y., Suzuki, M., and Sato, T. Discriminative accretion of radioactive gallium and strontium: tumor affinity of gallium. *Sc. Rep. Res. Inst. Tohoku Univ.* (Ser. c.), 1973, *20*, 1–4.
15. Okuyama, S., Ito, Y., Sato, T., Takahashi, K., and Matsuzawa, T. Tumor and skeletal imaging in bone carcinoma: experimental demonstration. *Radiology*, 1974, *113*, 681–685.
16. Okuyama, S., Sato, T., Ito, Y., and Matsuzawa, T. Proliferative tissue imaging in experimental tumor: studies with ^{67}Ga and ^{131}I-iododeoxyuridine. To be published.
17. Papavasiliou, O., Kostamis, P., Angelekis, P., and Constantinides, C. Localization of Sr-87m in extraosseous tumors. *J. Nuclear Med.*, 1971, *12*, 265–268.
18. Samuels, L. D. Detection and localization of extraskeletal malignant neoplasms of children with strontium 87m. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 777–782.
19. Scheer, K. E., Harbst, H., Kampmann, H., zum Winkel, K., Marier-Borst, W., Lorenz, W. T., and Blaniuk, L. Bone scintigraphy with ^{18}F and ^{87}Sr. In: Medical Radioisotope Scintigraphy. Vienna International Atomic Agency, 1968, pp. 325–339.

ACCURACY OF ^{99m}TC-DIPHOSPHONATE BONE SCANS AND ROENTGENOGRAMS IN THE DETECTION OF PROSTATE, BREAST AND LUNG CARCINOMA METASTASES*

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ABSTRACT:

A technetium 99m diphosphonate scan is a sensitive detector of bony metastases of breast, prostate, and lung cancer. For these particular neoplasms, a negative bone scan in an asymptomatic patient is adequate evidence for absence of bony metastases and a correlative roentgenographic examination may not be necessary. Positive studies demonstrating multiple characteristic discrete areas of increased activity should be considered strong evidence for metastases. Single equivocal lesions require roentgenographic and occasionally biopsy correlation. If roentgenography fails to reveal the source of increased uptake, (*e.g.*, degenerative disease) the scan lesion should remain suspicious for metastases.

THE management of patients with cancer is influenced by the extent of tumor spread, making accurate staging of malignant neoplasms important information for determination of proper medical or surgical care. The specific therapeutic regimen for patients with proved or suspected malignant disease may depend upon the presence or absence of skeletal metastasis. This report is an analysis of the relative accuracy of technetium 99m disphosphonate bone scan and the roentgenographic skeletal survey in the detection of metastatic bone disease in a selected group of patients with primary breast, prostate, and lung carcinoma.

MATERIAL AND METHOD

The diphosphonate used for these studies was chemically synthesized according to the method of Castronovo[1] and was labeled with technetium 99m as described by Castronovo and Callahan.[2] Routine bone scans were obtained following injection of 10–15 mCi of technetium 99m diphosphonate. Either a Nuclear-Chicago gamma camera or Ohio-Nuclear dual probe rectilinear scanner was used for imaging. Images on the rectilinear scanner were either 2:1 or 5:1 reductions. All scans had views of the head, trunk, and lower extremities to just below the knees. The roentgenographic metastatic survey included roentgenograms of the skull, spine, chest, pelvis, and proximal upper and lower extremities.

The case material consisted of 259 patient studies drawn from the first 800 technetium 99m diphosphonate scans. The patients included in this study met the following criteria: (1) histologic proof of breast, prostate, or lung carcinoma; and (2) roentgenographic and radionuclide studies on the same patient carried out within a two week period. Available for analysis were 118 patients with breast carcinoma, 87 with prostate carcinoma and 54 with carcinoma of the lung.

Based on initial scan and the roentgenographic interpretations, the patients were placed into one of seven categories.

Group 1: Patients with positive evidence of

* Presented at the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, Quebec, Canada, September 25–28, 1973.
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TABLE I

ACCURACY OF RADIONUCLIDE SCANS AND ROENTGENOGRAMS

(259 Cases)

	Breast	Prostate	Lung	Total	Percent Total
(I)* +Scan +X-ray	17	17	8	42	16
(II)† +Scan +X-ray	18	11	6	35	14
(III) +Scan −X-ray	15	20	13	48	19
(IV) −Scan +X-ray	3	3	0	6	2
(V) −Scan −X-ray	52	28	22	78	30
(VI) Equivocal Scans	13	8	5	26	10
(VII) Equivocal X-rays	18	4	2	24	9

* Equivalent number of lesions on radionuclide and x-ray studies.
† More lesions on radionuclide scans than on roentgenograms.

metastases on the roentgenogram and the bone scan. These patients demonstrated essentially the same areas of metastases on both studies.

Group II: Cases having positive scans and positive roentgenograms with more metastases demonstrated on the scans.

Group III: Cases with positive scans and negative roentgenograms.

Group IV: Cases demonstrating negative scans and positive roentgenograms.

Group V: Cases with scans and roentgenograms both negative.

Group VI: Cases of equivocal scans. These patients demonstrated single and sometimes diffuse regions of abnormal uptake that could not definitely be characterized as diagnostic of metastases.

Group VII: Cases of equivocal roentgenograms. These patients demonstrated apparent sclerotic or lytic areas in the roentgenograms that could not definitely be characterized as indicative of metastases.

RESULTS

Data were obtained for each type of tumor as well as for the entire group of patients as expressed in Table I.

As presented in Table I, positive scans and positive roentgenograms with the same number of metastases visualized on the two studies were demonstrated in 42 cases. In 35 cases, roentgenograms and scans were positive but additional metastases were visualized on the scan. In 48 cases (20 percent of all cases), patients had positive scans and negative roentgenograms.

In our series, 137 patients demonstrated evidence of bony spread of tumor (Table II). Forty-eight of these patients, or 36 percent of the number of patients with metastases, had roentgenographic studies that were interpreted as normal (false normal roentgenograms). From this group of 137 patients demonstrating metastases, 83 patients in Groups II and III demonstrated increased information by radionuclide study. The bone scan represents a signifi-

TABLE II

NUMBER OF PATIENTS DEMONSTRATING EVIDENCE OF METASTASES

(Total 137)

Group	Total
(I) +Scan +X-ray	42
(II) +Scan +X-ray	35
(III) +Scan −X-ray	48
(IV) −Scan +X-ray	6
(V) Equivocal scans (+X-ray)	2
(VI) Equivocal X-rays (+Scans)	4

cant improvement in sensitivity in the detection of metastatic disease.

Of the 26 patients in the equivocal bone scan category, additional data including detailed roentgenograms of suspicious areas showed that eight patients had degenerative joint disease, two had Paget's disease, five had old or more recent non-pathological rib fracture, one had a non-pathological compression of a vertebral body, and one had marked hyperostosis frontalis interna of the skull. There was one equivocal scan due to osteonecrosis following radiation therapy. Two patients demonstrated roentgenograms that were consistent with metastases and, therefore, are included as demonstrating metastases in Table II. Six of the 26 patients remained equivocal for metastases, and a one year follow-up proved four of these patients to have increased uptake in the scan due to a spread of the primary tumor to bone.

Twenty-four of the 259 patients had roentgenograms that originally were interpreted as equivocal. Correlation of roentgenograms with bone scans in these patients demonstrated no areas of increased uptake in 18 instances and follow-up roentgenograms were negative. These equivocal areas were interpreted as representing bone islands, infarctions, degenerative joint disease, and artifacts. Four patients with equivocal roentgenograms demonstrated increased uptake in the same areas on the bone scan and were interpreted as being secondary to metastases. Two patients remained equivocal for metastases based on the roentgen findings.

There were six patients whose roentgenograms indicated metastases, while the scans were interpreted as not showing tumor involvement (false negative scans). Three of these cases had received radiation therapy to the areas in question and based on the negative radionuclide bone scan, the neoplastic disease in the treated area was considered inactive. In these cases, correlative roentgenograms continued to show sclerotic changes that could easily be interpreted as metastases. A fourth patient had difficulty voiding and had a sacral lesion which was obscured by a large bladder containing the radionuclide. The remaining two cases were misinterpreted and were prostatic carcinoma patients with metastases so extensive that focal areas were not appreciated. These last two cases led to a useful sign that can reduce such misinterpretations, as discussed later.

DISCUSSION

Histological data indicate that prostate, breast, and lung carcinoma metastases to bone stimulate new bone formation and neovascularity.[6] Such changes are consistently demonstrated and probably account for most of the increased activity noted on the bone scan. This osteoid response results in increased metabolic turnover of bone mineral which is detected earlier by radionuclide scan than by roentgenographic means, which often requires 40 to 50 percent destruction of bone (*e.g.*, vertebral body) before metastasis can be appreciated.

Our statistics demonstrated that technetium 99m diphosphonate images are a

sensitive detector of metastatic disease to the bone. When compared to the standard roentgenographic metastatic survey the bone scan was more sensitive in about 30 percent of the total number of this group of cases and in about 60 percent of the cases demonstrating metastases. Our experience confirms previous reports in which strontium 85, fluorine 18 and technetium 99m as polyphosphate and diphosphonate were shown to be more sensitive in the detection of metastases than roentgen ray techniques.[4,7–9]

Apparent exceptions to the osteoid response have been noted with thyroid carcinoma, reticulum cell sarcoma, and multiple myeloma of bone. In such cases, primarily lytic lesions can be demonstrated by roentgenography quite well, whereas the bone scan may be normal or relatively normal. Therefore, the type of primary neoplastic disease should be taken into account when the bone scan is being considered in the evaluation of patients for metastases to bone.

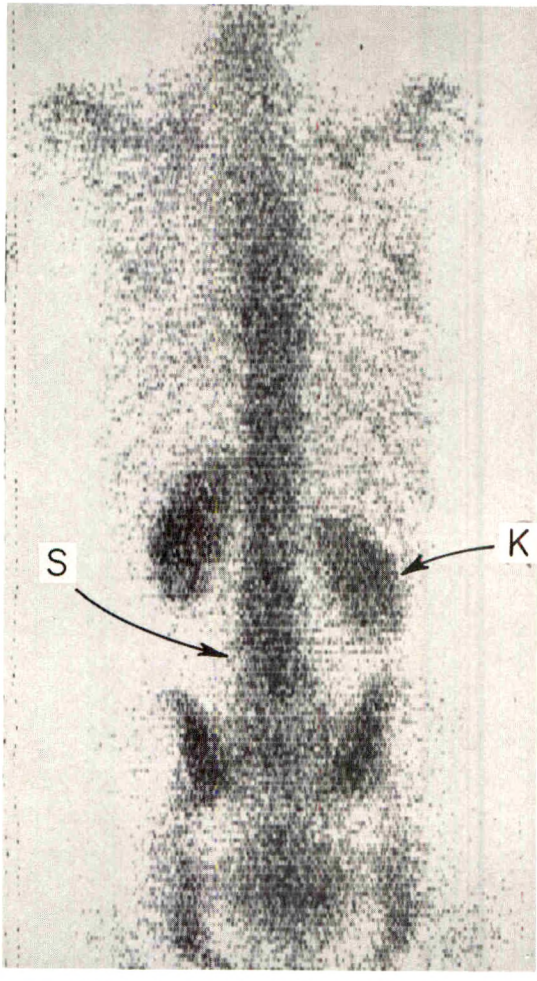

Fig. 1. Normal scan (posterior view): kidney (K) activity equal to adjacent lumbar spine activity (S).

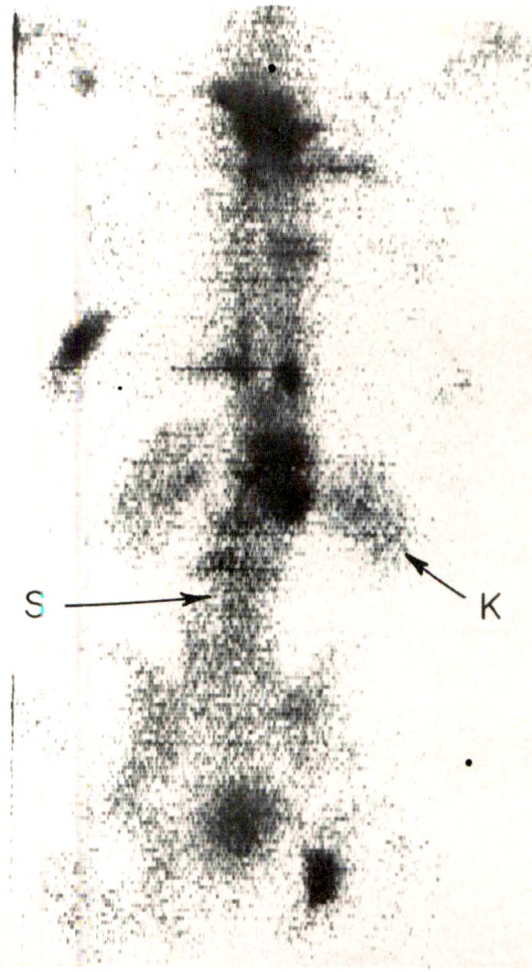

Fig. 2. Patient with multiple metastases from breast carcinoma showing kidney (K) activity equal to adjacent activity in uninvolved spine (S).

The percentage of scans and roentgenograms that were originally interpreted as equivocal was about the same with nine percent of the roentgenograms and ten percent of the bone scans in that category. The low number of false negative bone scans in this series demonstrates the increased sensi-

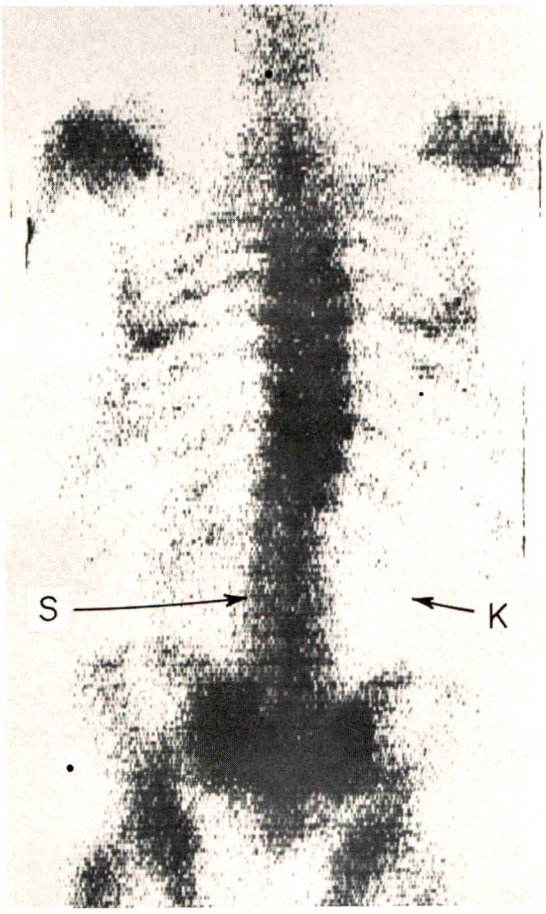

FIG. 3. "Absent kidney" sign in an elderly male with diffuse prostate carcinoma metastases producing a so-called "super scan" with very low activity in the kidney (K) and very high activity in the spine (S).

tivity of the scan as a screening procedure. Because of the higher contrast inherent in the bone scan, it can be searched and interpreted more rapidly than is possible with a roentgen metastatic survey.

Twenty-six bone scans were originally interpreted as equivocal for metastases, emphasizing that the bone scan is nonspecific. These scans required close roentgenographic correlation for a more meaningful interpretation and in a majority of instances, the cause of increased uptake (*e.g.*, degenerative disease) became evident. Our experience indicates that if the etiology remains obscure following roentgenographic correlation, the scan should be considered suspicious for metastases. We have come to view the request for a bone scan as a consultation, and make every effort to correlate all available information including detailed history, physical examination, and laboratory data as well as available roentgenograms in order that the most definitive answer can be given to the attending physician.

Initial misinterpretations of extensive prostate carcinoma metastases led to our appreciation of the "reduced kidney activity sign" and the appreciation of a so-called "super bone scan," in which the skeleton stands out in bold relief. We soon recognized that patients in whom the skeleton is heavily involved by a pathological process (*e.g.*, extensive metastases) produce a marked increase in bone radionuclide concentration with less activity available for excretion by the kidneys, thereby making the urinary tract less visible. Comparing the amount of activity noted in the kidneys to that seen in the lumbar spine can be a useful sign. At four hours after the administration of the technetium 99m diphosphonate, the kidneys and the lumbar spine usually show equivalent amounts of radioactivity. If the kidneys contain significantly less activity or are not visualized at all, and there is evidence of normal renal function, one should be suspicious of an extensive process markedly affecting bone mineral accretion. These observations suggest that a measurement of the ratio of activity in the kidneys to that of the lumbar spine may be effective (Fig. 1–3). The diagnosis of systemic disorders affecting bone mineral turnover (*e.g.*, hyperparathyroidism) may be possible. The value of the renal-spine ratio (RSR) is under investigation.

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REFERENCES

1. CASTRONOVO, F. P., JR. Method for synthesis of 1-hydroxy-ethylidene-1, 1-disodium phosphonate (HEDSPA): skeletal seeking radiopharmaceutical after labeling with ^{99m}Tc. *J. Nuclear Med.*, 1974, *15*, 127–130.

2. Castronovo, F. P., Jr., and Callahan, R. J. New bone scanning agent ^{99m}Tc labeled 1-hydroxy-ethylidene-1, 1-disodium phosphonate. *J. Nuclear Med.*, 1972, *13*, 823–827.
3. DeNardo, G. L., and Volpe, J. A. 85-Sr scintiscan in bone disease. *Ann. Int. Med.*, 1966, *65*, 44–53.
4. Harmer, C. L., Burns, J. E., Sams, A., *et al.* Value of fluorine-18 for scanning bone tumors. *Clin. Radiol.*, 1969, *20*, 204–212.
5. Legge, D. A., Tauxe, W. N., Pugh, D. G., *et al.* Radioisotope scanning of metastatic lesions of bone. *Mayo Clin. Proc. Staff Meet.*, 1970, *45*, 755–761.
6. Milch, R. A., and Changus, G. W. Response of bone to tumor invasion. *Cancer*, 1956, *9*, 340.
7. Milner, T. H., Maynard, C. D., and Cowan, R. J. Evaluation of strontium-85 bone scans and roentgenograms in 100 patients. *A.M.A. Arch. Surg.*, 1971, *103*, 371–372.
8. Silberstein, E. D., *et al.* Imaging of bone metastases with ^{99m}Tc-Sn-EHDP (diphosphonate), ^{18}F, and skeletal radiography. *Radiology*, 1973, *107*, 551–555.
9. Spencer, R., Herbert, R., Rish, M. W., *et al.* Bone scanning with ^{85}Sr, ^{87m}Sr and ^{18}F. *Brit. J. Radiol.*, 1967, *40*, 641–654.

WHOLE BODY TRANSMISSION-EMISSION SCANNING WITH WHOLE BODY CAMERA*

By YUTAKA SUZUKI, M.D.,† HIROFUMI MORI, M.D., TAKATOSHI MICHIGISHI, M.D., KINICHI HISADA, M.D., *and* MASAMICHI MATSUDAIRA, R.T.
KANAZAWA, JAPAN

ABSTRACT:

Whole body transmission-emission scanning was carried out using a whole body camera. A disk source 30 cm in diameter filled with ^{99m}Tc was used for transmission scanning. Correct interpretation of emission scanning requires accurate anatomical orientation of the images.

The value of whole body transmission-emission scanning is emphasized.

ACCURATE evaluation of radionuclide distribution in the body always requires that the spatial relationship of emission scan data be oriented to the anatomy of the patient. Although reference markers or standard roentgenograms have been used for this purpose, there are limitations in all of these methods. Transmission scanning has largely overcome these problems.[1,3] However, this procedure is used mainly for outlining the organs bordered by air. We have tried whole body transmission scanning with a whole body camera as a means of improving the orientation of the whole body emission scan. The purpose of this paper is to describe its efficacy and point out ways in which this technique has improved our diagnostic acumen.

MATERIAL AND METHOD

Both whole body emission and transmission images were obtained simultaneously by using the Picker Omniview system, Picker Dyna Camera II-C.‡ A disk source 30 cm in diameter filled with 3 to 5 mCi of ^{99m}Tc-pertechnetate was used for the transmission image. The dose of ^{99m}Tc-pertechnetate was adjusted to get approximately the same number of counts as emission activity. One spectrometer was set to the photopeak of the emission photon and the other was set to 140 KeV. The whole body images were taken on roentgen-ray film with 7:1 or 5:1 minification and information density of 200 counts/cm^2. When the radioactivity in the body was small, the emission image and transmission image were taken separately with different exposure factors, *i.e.*, the source intensity was adjusted high for the emission image and low for the transmission image, so that the transmission-emission image was clearer than in the conventional method. The whole body emission-transmission scanning was done for radionuclide myelography with ^{169}Yb DTPA, survey of the metastatic thyroid cancer with ^{131}I, and radionuclide lymphography with ^{198}Au colloid.

‡ Picker Corporation, 595 Miner Road, Cleveland, Ohio 44143.

REPORT OF CASES

CASE I. M.K. was a 62 year old female with thyroid carcinoma. She had had a total thyroidectomy two months ago in another hospital and the histological diagnosis was follicular carcinoma. The patient was referred to our department for evaluation of bone metastasis. Whole body emission-transmission scanning was done 48 hours after oral administration of 2 mCi of ^{131}I and confirmed the fact that metastatic lesions were present in many parts of the body. The whole body transmission image made the anatomical orientation of the lesions much easier (Fig. 1).

CASE II. M.T. was a 65 year old male with bronchogenic cancer. On physical examination, muscle weakness and loss of sensation were

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found in the lower extremities. Radionuclide myelography was requested to rule out spinal cord metastasis. Whole body transmission scanning and radionuclide myelography with 1 mCi of ^{169}Yb DTPA were performed simultaneously. The emission-transmission scanning 24 hours after intrathecal injection revealed complete obstruction of the spinal subarachnoid space at the level of the diaphragm (Fig. 2*A*). Contrast myelography showed the obstruction at the same level found by radionuclide myelography (Fig. 2*B*). Whole body transmission scanning was very useful for the determination of the level of obstruction.

DISCUSSION

Anatomical orientation of emission scan data is quite important in scan interpretation. Usually, this is obtained by using reference marks to present external features or by superimposing a roentgenogram of the part. These methods may introduce inaccuracy due to geometric distortions. Transmission scanning has been proved to reduce these inaccuracies and to be useful

Fig. 1. Whole body transmission-emission 48 hours after oral administration of 2 mCi of ^{131}I (posterior view). Areas of increased activity reveal the metastatic foci of thyroid cancer.

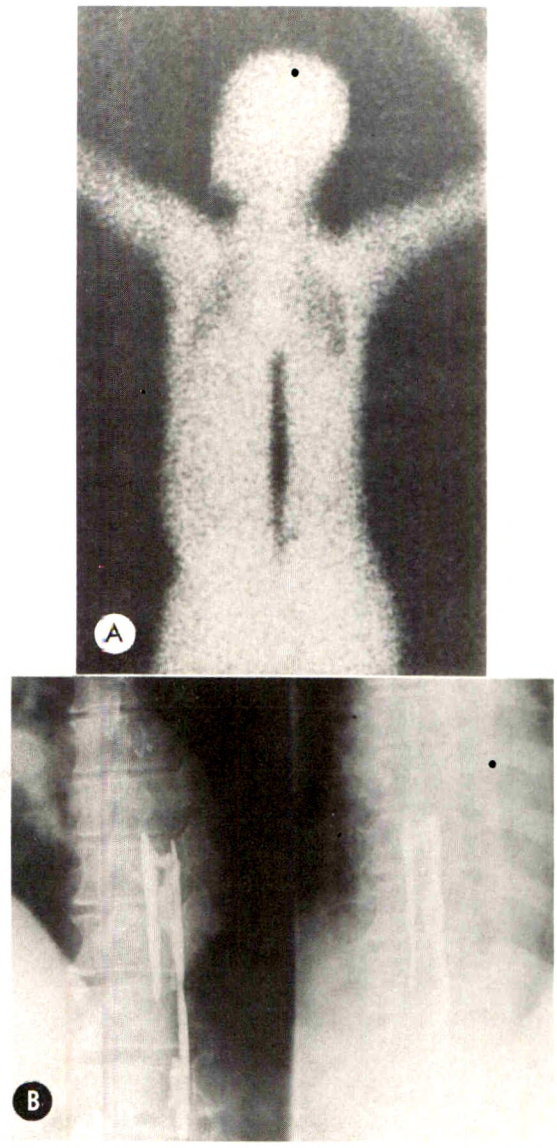

Fig. 2. (*A*) Whole body transmission-emission scanning of radionuclide myelography with 1 mCi of ^{169}Yb DTPA 24 hours after intrathecal injection (posterior view). The complete obstruction of the spinal subarachnoid space is clearly visualized. Anatomical orientation is readily obtained here. (*B*) Right posterior oblique and anteroposterior views of contrast myelogram show cupping of the upper margin of the subarachnoid contrast column.

in anatomic orientation, aiding in interpretation of emission scans.[1,3] This procedure has become widely used in nuclear medicine,[6,7] but a disadvantage of the conventional transmission scanning has been the limited field of view; therefore transmission scanning has been applied mainly to localized areas of the body.

In 1972 Cooke and Kaplan[2] first described the principles and application of the whole body camera system. This system has the advantage of high sensitivity, better image linearity and uniformity, and good resolution.[4] Use of the whole body camera system is easily expanded to transmission scanning. The time needed to perform whole body transmission scanning is less than 30 minutes, and emission scanning can be obtained simultaneously. Thus there is a significant saving of time and patient comfort.

In interpretation of radionuclide myelography and lymphography, the biggest problem is to get accurate anatomic orientation of the images. Use of whole body transmission technique can readily resolve this problem. In our experience, the whole body transmission image is also useful in survey of metastasis of thyroid cancer with ^{131}I. We are now using this technique routinely in follow-up study of patients with thyroid cancer. Application of this technique to tumor scanning may improve the accuracy of diagnosis.

Various radionuclide sources have been used in transmission imaging procedures, including ^{125}I, ^{241}Am, and ^{99m}Tc. Of these, only ^{99m}Tc has proved satisfactory for studies with the scintillation camera because of loss of spatial resolution with this device at lower photon energies.[5] Moreover, ^{99m}Tc is readily available in any nuclear medicine laboratory and quite economical.

In conclusion, whole body emission-transmission scanning with the whole body camera is quite useful in anatomical orientation of lesions. This procedure could be applied to various kinds of imaging studies in nuclear medicine without extra cost and time.

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REFERENCES

1. ANGER, H. O., and MCRAE, J. Transmission scintiphotography. *J. Nuclear Med.*, 1968, *9*, 267–269.
2. COOKE, M. B. D., and KAPLAN, E. Whole-body imaging and count profiling with modified Anger camera. I. Principles and application. *J. Nuclear Med.*, 1972, *13*, 899–902.
3. KUHL, D. E., HALE, J., and EATON, W. L. Transmission scanning: useful adjunct to conventional emission scanning for accurately keying isotope deposition to radiographic anatomy. *Radiology*, 1966, *87*, 278–284.
4. LEE, V. W., SANO, R., and FREEDMAN, G. Whole-body gamma camera imaging using moving table accessory. *J. Nuclear Med.*, 1973, *14*, 830–833.
5. SORENSEN, J. F. Evaluation of radionuclide sources for transmission scanning. *Invest. Radiol.*, 1970, *5*, 92–102.
6. VOLPE, J. A., MCRAE, J., and JOHNSTON, G. S. Transmission scintigraphy in evaluation of subphrenic abscess. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 733–734.
7. YEH, E., RUETZ, P. P., and MEADE, R. C. Separation of lung-liver scintiphotos due to ascites—false positive test for subdiaphragmatic abscess. *J. Nuclear Med.*, 1972, *13*, 249–251.

Vol. 125, No. 4

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Published by: Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Illinois 62717

Issued monthly, Annual subscription: United States, U.S. Possessions, U.S. Trusteeships, $30.00; Pan American Union and Canada, $35.00; other countries, $35.00. Current single numbers, $3.00. Advertising rates submitted on application. Editorial office, 401 Professional Building, Detroit, Michigan 48201. Office of publication, 301–327 East Lawrence Avenue, Springfield, Illinois 62717. Information of interest to authors and readers will be found on page ii.

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The Fifty-eighth Annual Meeting: Bayshore Inn, Vancouver, British Columbia, Canada, May 9–14, 1976.

EDITORIAL

THE SEVENTY-SIXTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Seventy-sixth Annual Meeting of the American Roentgen Ray Society beautifully fulfilled its preparation and promise. Held in Atlanta, Georgia, between September 30 and October 3, 1975, in a lovely time of the year there, it amply supplied the intellectual, recreational, and social requirements of the members and guests who attended.

The meeting opened Tuesday morning with the call to order by the President, Dr. Colin B. Holman, and he, with Dr. Melvin M. Figley, Chairman of the Executive Council, installed the new President, Dr. Ted F. Leigh. Dr. Leigh's inaugural address prepared us for the meeting to come—a feast of fine radiology, well spiced with new ideas.

Dr. Leigh had prepared a stimulating, new, and thought-provoking program. The 197 essayists, who presented a total of 69 papers, required simultaneous sessions on Wednesday and Friday mornings, while single sessions were presented on Tuesday and Thursday mornings. The papers were thoughtfully grouped according to subject. As in the past, award papers were contributed from The Society for Pediatric Radiology, The American Society of Neuroradiology, and The Society of Gastrointestinal Radiologists. In addition to expository and innovative papers on the traditional subjects, Dr. Leigh's program presented material covering ultrasound, thermography, computerized axial tomography of both the head and body, nuclear medicine, and radiation therapy.

The program culminated in the Caldwell Lecture, "Iatrogenic Lesions of the Skeleton." Dr. Harold G. Jacobson introduced the Caldwell Lecturer, Dr. Ronald O. Murray, of London, England, who spoke with authority, eloquence, warmth, and fascination on a subject which has interested him for years, as judged by his mastery of it. Following the Lecture, Dr. Leigh presented the society's Caldwell Medal to Dr. Murray.

The program of Instruction Courses was again this year expanded by the director, Dr. Harold O. Peterson. Fifty-nine courses consisting of one or two 90 minute sessions were presented by a faculty of 78 instructors, from Monday through Friday, and these courses covered in detail small fields in diagnostic and therapeutic radiology as well as subjects in ultrasound, xeroradiography, nuclear medicine, and computerized axial tomography. An innovation this year was a Comprehensive Course on the Chest, presented in six daily meetings, including three hour sessions on Sunday and Monday, September 28 and 29. The faculty of 14 physicians for this intensive course covered many facets, both elementary and abstruse, of diseases of the lungs from a roentgen point of view.

Attendance at the Scientific Program and the Instruction Courses was recognized by credit in postgraduate medical education by the American College of Radiology.

The exhibition halls of the hotel lent themselves well to the presentation of the 104 scientific and informational exhibits and this was carried out under the direction and planning of the Chairman of the Scientific Exhibits Committee, Dr. Byron G. Brogdon. The excellence of this vital part of the learning experience of the meeting is attested by the fact that gold, silver, and bronze awards, as well as 20 certificates of merit were deserved and given. These are

described elsewhere in this issue. The exhibits were grouped according to subject, the exhibitors showed great interest in explaining their material, and the sufficient space in the hall permitted the inevitable and welcome discussion of the exhibits. The daily unknowns, the Adult and Pediatric Cases of the Day, afforded competition, titillation, and frustration.

The Committee on Local Arrangements, chaired by Dr. James V. Rogers, and the Ladies' Committee, co-chaired by Mrs. James V. Rogers and Mrs. B. Donald Minor made things pleasant, easy, and attractive for members and guests at the meeting. There were the traditional golf outing, and the about-to-become-traditional tennis tournament, together with a fine program for the fortunate consorts of those who attended the meeting. The Ladies' Program (and this may some day, *mutatis mutandis*, become the Consorts' Program) included tours of local antebellum homes and a monastery, a visit to the remarkable Stone Mountain Park, and an exhibit of arts and crafts, among other pursuits. The committee established a Hospitality Room in the hotel which was womanned by a group which functioned beautifully (in every sense of the word) as guides, explicators, and confidantes, making it an information center, meeting place, and haven for the uncomfortable.

The active press coverage was in the experienced and able hands of Mr. Charles Honaker and his associates. The technical exhibits added to the information available at the meeting, and they are listed elsewhere. The space for these exhibits adjoined that for the scientific exhibits, was spacious, and housed the 60 exhibitors conveniently.

Dr. Ted Leigh, as President, planner, executive, and orchestrator deserves and has earned the thanks and praise of the society for this memorable meeting.

E. Frederick Lang, M.D.

THE SCIENTIFIC EXHIBITS

THE SCIENTIFIC EXHIBITS of the Seventy-sixth Annual Meeting of The American Roentgen Ray Society, held at the Marriott Motor Hotel in Atlanta, Georgia, September 30–October 3, 1975, were truly impressive, both quantitatively and qualitatively. One hundred and four exhibits displayed a spectrum of scientific and educational material unsurpassed in the annals of the Society. Many of these exhibits were complimented by companion presentations during the Scientific Sessions, and expanded viewing hours allowed the dedicated attendee to avail himself of both oral and visual educational opportunities.

The anonymous Scientific Awards Committee fairly accomplished the difficult task of evaluating all exhibits competitively and after much study and deliberation awarded 20 Certificates of Merit and selected three exhibits for the coveted *Gold*, *Silver* and *Bronze* Medals of The American Roentgen Ray Society.

GOLD MEDAL

Density Perception. Edward J. Lane, M.D., Anthony V. Proto, M.D., and Thomas W. Phillips, M.D., State University of New York, Upstate Medical Center, Syracuse, New York.

The perception of the roentgen image is influenced greatly by retinal neural networks which produce Mach bands. Part A of this exhibit illustrated the mechanism of production of Mach bands and showed how differences in contour, contrast and density of radiographic images influence their visualization. Mach bands often facilitate the perception of roentgen densities; however, misinterpretation of their significance may lead to errors in diagnosis. The vital role played by Mach bands in clinical radiological interpretation was illustrated by example.

In Part B, measurement of object density (Archimedes principle) and film density (photodensitometry) demonstrated with an experimental model a distinctly visualized roentgen density difference with an object density difference of 1 percent. Clinical material clearly demonstrated visualization of a soft tissue density difference of 5 percent. Variations in object contour density, film contrast and density that aid or detract in density perception were demonstrated both with an experimental model and with clinical material.

SILVER MEDAL

Pathogenesis of Bleeding Colonic Diverticulosis: New Observations. Morton A. Meyers, M.D., Daniel R. Alonzo, M.D., George F. Gray, M.D., and Jeanne W. Baer, M.D., The New York Hospital—Cornell University Medical College and St. Luke's Hospital, New York, New York.

The most common cause of acute severe rectal hemorrhage is colonic diverticulosis. However, the precise site of bleeding, etiology, and pathogenesis have not been previously identified.

Arteriographic, micro-angiographic, and detailed histologic observations in 14 proved cases disclosed strikingly consistent changes related to the characteristic angioarchitecture of colonic diverticula which the authors have established. This represents the largest reported series. The changes include: (1) asymmetric rupture of the vasa recta precisely at the dome or the antimesenteric margin of the diverticulum; (2) general absence of diverticulitis; (3) conspicuous eccentric intimal thickening of the vasa recta, often with medial thinning, at and near the bleeding point.

Comparison with colonic diverticula in non-bleeding cases indicates that traumatic factors arising within the diverticular lumen induce asymmetric intimal proliferation and scarring of the associated vasa recta predisposing to rupture and massive bleeding.

Bronze Medal

Radiology in Primary Hyperaldosteronism. Heun Y. Yune, M.D., Eugene C. Klatte, M.D., Clarence E. Grim, M.D., and Harry N. Wellman, M.D., Indiana University School of Medicine, Indianapolis, Indiana.

Radiology plays a significant role in the diagnostic work-up of the group of hypertensive patients whose laboratory data are characterized by hypokalemia and low renin. Selective adrenal venous blood sampling with simultaneous venography is the single most effective way to lateralize and often localize the lesion responsible for the primary hyperaldosteronism. The authors' clinical experience in the past two years in dealing with approximately 40 such patients was summarized and analyzed. Preoperative differential aldosterone data were correlated with observations on the adrenal venography and the pathology of the surgical specimen.

Certificates of Merit

Computerized Tomography of the Human Body. Ralph J. Alfidi, M.D., Leopold Gonzales, M.D., John Haaga, M.D., Thomas F. Meaney, M.D., A. R. Antunez, M.D., P. M. Duchesneau, M.D., J. MacIntyre, M.D., and E. L. Jelden, M.D., Cleveland Clinic, Cleveland, Ohio.

The present stage of development of computerized radiographic tomography offers promise of becoming a new diagnostic field in the evaluation of the body.

The differentiation between tissue densities obtained with the prototype unit known as the Delta Scanner (Ohio Nuclear, Inc., Solon, Ohio) allows visualization of body structures not possible before with the known imaging methods.

The pancreas, in normal and pathological states, has been demonstrated.

Primary and metastatic tumors of the liver have been identified, as well as the gallbladder and biliary ducts. Tumors and cystic lesions of the kidneys, adrenals, pelvic masses, retroperitoneal tumors, periaortic lymph nodes and aneurysms of the abdominal aorta have been identified.

Pericardial effusion and cardiac valvular calcifications have also been shown, as well as mediastinal masses and lymphadenopathy.

Brain lesions have been visualized with resolution comparable to the EMI Scanner. We have demonstrated cases of diastematomyelia and a chordoma of the sacrum.

This exhibit showed a representative example of the conditions previously mentioned.

Correlating ultrasound scans and angiograms were presented in some of the cases.

Imaging Considerations for Improved Thin Section Tomography. Gary T. Barnes, Ph.D., David M. Witten, M.D., and John G. Kahler, M.D., University of Alabama in Birmingham, Birmingham, Alabama.

The physical factors underlying detail perception in thin section tomography were examined. Clinical comparisons were made between film/screen combinations which take advantage of these factors and more routinely used combinations.

High K.V. Magnification Technique in Diagnosis of Upper Airway Obstruction in Infants and Children. Walter E. Berdon, M.D., David H. Baker, M.D., Thomas L. Slovis, M.D., Jack O. Haller, M.D., and Peter Joseph, Ph.D., Babies Hospital, Columbia Presbyterian Medical Center, New York, New York.

Anteroposterior and lateral high K.V. magnification technique has proved most valuable in assessing supra and subglottic obstructive disease in infants and children. Inflammatory disease (epiglottitis, croup) as well as intraluminal obstruction from hemangioma and foreign bodies have been diagnosed with increasing frequency since the technique was instituted. It is considered, along with barium esophagography, as the screening technique in such patients.

Amebiasis. Jose Manuel Cardoso, M.D., Kenji Kimura, M.D., Miguel Stoopen, M.D., Lizandro Elizando, M.D., Robert Churchill, M.D., and Rogelio Moncada, M.D., Loyola University Medical Center,

Maywood, Illinois and Hospital General Centro Medico National, I.M.S.S., Mexico City, Mexico.

Over 2,000 cases of intestinal and hepatic amebiasis have been seen during the past 10 years at the institution in Mexico City. Intestinal involvement and detection of liver abscesses through radionuclide imaging, ultrasonography, total body opacification, gastrointestinal series and angiography were displayed as well as results of selective transarterial hepatic infusion of Dehydroemetine for liver abscesses.

Computerized Axial Tomography and Brain Scintigraphy in Stroke Syndrome. Lee C. Chiu, M.D., Steve H. Cornell, M.D., Laszlo B. Fodor, M.D., and James H. Christie, M.D., University of Iowa Hospitals and Clinics, Iowa City, Iowa.

There are certain characteristic signs that appear with stroke syndrome both on computerized axial tomography and Tc^{99m} pertechnetate brain scintigraphy. When these signs are correlated jointly, they offer more accuracy in diagnosis without further invasive study, particularly in the ischemic middle cerebral artery syndrome. Cases demonstrating these findings were discussed and illustrated.

The Twelve Significant Indirect Signs of Cervical Spine Trauma. William M. Clark, M.D., John A. Gehweiler, Jr., M.D., and Richard H. Laib, M.D., Duke University Medical Center, Durham, North Carolina.

Twelve practical, indirect radiographic signs of significant cervical spine trauma are illustrated. These were divided into: soft tissue signs, vertebral alignment and changes in their joint spaces.

Hodgkin's Disease of the Lung and Mediastinum: Radiologic-Pathologic Correlations. E. R. Heitzman, M.D., Anthony V. Proto, M.D., and B. Markarian, M.D., State University of New York, Upstate Medical Center, Syracuse, New York.

Hodgkin's disease is the most common neoplasm occurring in young adults and is the most common lymphoma in any age group. This exhibit presented a radiologic-pathologic correlative study of Hodgkin's disease involving the lung and mediastinum. It discussed: (1) the varied radiological manifestations of thoracic Hodgkin's disease in relation to the anatomy of the lymphatic system of the lung and mediastinum; (2) the appearance of mediastinal Hodgkin's related to histopathologic classification; (3) the problem of opportunistic infection and its radiological differentiation from parenchymal Hodgkin's disease; and (4) radiological features which influence treatment planning in radiotherapy.

Avian Respiration: How Birds Breathe. A. Everette James, Jr., M.D., Grover Hutchins, M.D., Frederick Stitik, M.D., T. K. Natarajan, M.E.E., Phillip Steig, M.D., Richard M. Heller, M.D., Barry Burns, Ph.D., Mitchell Bush, D.V.M., and Mark C. Horning, Johns Hopkins Hospital, Baltimore, Maryland.

Avian respiration shows facilitated gas exchange when compared with humans. Using dynamic imaging techniques (radiography, fluoroscopy, bronchography, angiography, as well as perfusion and inhalation lung scans) the avian respiratory system was accurately characterized *in vivo* and compared with postmortem anatomical, injection, and histological studies. The distribution of inspired air was documented using regional analysis by computer of dynamic images of all phases of respiration.

These studies showed that the reason for the facilitated gas exchange in birds is their great surface area of blood-terminal airway interface.

Mycoplasma Pneumonia: Two Distinct Syndromes. Pamela S. Jensen, M.D., and Charles E. Putman, M.D., Yale University School of Medicine, New Haven, Connecticut.

Pulmonary infections secondary to *Mycoplasma Pneumoniae* are commonly manifest by the acute onset of cough, chills, and fever associated with the radiographic findings of airspace (alveolar) consolidation in a lobar or subsegmental distribution with minimal volume loss. Recent investigation of over 200 documented mycoplasma

infections indicates that there is another frequent but markedly dissimilar presentation consisting of chronic dyspnea associated with a diffuse interstitial pulmonary process compatible with chronic interstitial pneumonitis. Analysis of the clinical data and representative radiographs of these two forms of disease were presented. In addition, the relationship between mycoplasma infections and immunologic disorders, specifically sarcoidosis and systemic lupus erythematosus, was stressed.

Intracranial Hemangiopericytomas: Angiography, Pathology and Differential Diagnosis. Joseph A. Marc, M.D., Y. Takei, M.D., M. M. Schechter, M.D., and J. C. Hoffman, M.D., Emory University, Atlanta, Georgia.

Intracranial hemangiopericytomas are tumors which angiographically and histologically can mimic other vascular intracranial masses. Several terms have been applied to this tumor including angioblastic meningioma, meningeal sarcoma, vascular meningioma and angiosarcoma. As these tumors are prone to recurrence after surgery as well as resistant to radiation therapy, it is important to recognize them angiographically and histologically. This exhibit illustrated: (1) the angiographic findings common to 5 cases of histologically proven hemangiopericytoma; and (2) the angiographic differential points between these tumors and other vascular tumors with which they are confused, notably glioblastoma multiforme, vascular meningothelial meningioma and supratentorial hemangioblastoma. The important histologic characteristics of hemangiopericytoma also were emphasized.

The Small Bowel in Immunoglobulin Deficiency Syndromes. Richard H. Marshak, M.D., Rubem Pochaczevsky, M.D., Daniel Maklansky, M.D., Arthur E. Lindner, M.D., and Jerold D. Kurzban, M.D., Mt. Sinai School of Medicine, the City University of New York, New York, New York.

After the first reported cases of congenital agammaglobulinemia by Burton in 1952, it was soon realized that the disease was more frequent in adults and the term was changed to late onset acquired hypogammaglobulinemia, but malabsorption and diarrhea soon became a constant feature of this entity. The immunoglobulins have been studied extensively. It was the purpose of this exhibit to present a variety of immunodeficiency disorders and their roentgen characteristics. Of the immunoglobulins, IgA, Igg, and Igm are predominantly involved. Some of the disorders discussed were: sprue, immunoglobulin variable disease, Iga deficiency disease, intestinal lymphangiectasia, amyloidosis and heavy-chain disease.

Optimal Visualization of Esophageal Varices. Roscoe E. Miller, M.D., Edward M. Cockerill, M.D., Stanley M. Chernish, M.D., Gordon C. McLaughlin, III, M.D., and Bruce E. Rodda, Ph.D., Indiana University School of Medicine, Indianapolis, Indiana.

Following the parenteral administration of an anticholinergic drug, enlargement of varices during endoscopy has recently been reported. Can the radiographic appearance of distal esophageal varices be enhanced either by drugs, position, respiration, time or any combination thereof? This question prompted a double blind crossover evaluation of placebo, propantheline bromide, and glucagon combined with various maneuvers.

Twelve patients with endoscopic proved varices volunteered for three examinations each. The results demonstrated: (1) that an anticholinergic agent enhanced the appearance of varices in the distal esophagus; (2) that the effects of propantheline bromide are more diagnostic, but have greater side effects than glucagon or placebo; and (3) that contrary to previous reports, positioning, time and respiratory cycle does influence visualization and identification of varices.

Opportunistic Pulmonary Infections: Clinical Correlations and Radiographic Findings. Christine H. Murphy, M.D., Stewart R. Roberts, Jr., M.D., Stephen F. Schwarz-

mann, M.D., and Ted F. Leigh, M.D., Emory University Affiliated Hospitals, Atlanta, Georgia.

Due to prolongation of life through organ transplantation, chemotherapy of malignant disease, surgery and instrumentation, and antibiotic therapy, organisms which normally inhabit the host in a commensal relationship have increasingly become themselves pathogens. Opportunistic pulmonary infections were categorized and correlated with the underlying primary disease. Typical findings of illustrative opportunistic pulmonary infections were presented.

The Radiology of Pulmonary Tuberculosis in Childhood. Martin H. Reed, M.D., and Reynaldo D. Pagtakhan, M.D., Health Sciences Children's Centre, Winnipeg, Manitoba.

Despite the declining prevalence of tuberculosis in North America, it remains a significant health problem in certain groups and areas. Radiologists must, therefore, be aware of the condition. This exhibit was based on the authors' experience of approximately 40 new cases a year. It showed the important radiological features of pulmonary tuberculosis in childhood which include:

1. The primary complex.
   This comprises the initial focus of infection in the lung and in the enlarged draining lymph nodes.
2. Secondary effects of lymph node enlargement:
   (a) Overinflation
   (b) Atelectasis
   (c) Bronchiectasis
3. Pleural effusion
4. Miliary disease
5. Other uncommon abnormalities.

Diffuse Idiopathic Skeletal Hyperostosis. Donald Resnick, M.D., and Gen Niwayama, M.D., Veterans Administration Hospital, San Diego, California.

In this beautiful exhibit newer concepts in the pathogenesis and roentgenographic appearance of spinal osteophytes were outlined in a radiographic and pathologic evaluation of over 200 autopsies. The entire anterior thoracic and lumbar vertebral column was removed intact and coronal or sagittal sections of the frozen specimens were radiographed and photographed. Tissue maceration and histologic evaluation utilizing paraffin embedding were employed.

The spinal osteophytes of diffuse idiopathic skeletal hyperostosis begin as periosteal new bone formation along the anterior and right lateral aspects of the vertebra. Initial or concomitant calcification and ossification in the anterior longitudinal ligament eventually merge with the underlying vertebra and produce large excrescences which appear as "flowing" osteophytes along portions of the thoracic spine, frequently with an underlying radiolucency, or irregular spurs at the vertebral body-intervertebral disk junction in the lumbar region. The outgrowths are usually associated with maintenance of disk height.

Evidence presented suggests this disease is distinct from "degenerative" disk disease (intervertebral osteochondrosis) and part of a generalized "ossifying diathesis" of the skeleton. The differential diagnosis of spinal outgrowths (*e.g.*, ankylosing spondylitis, acromegaly) was discussed again utilizing radiographic and pathologic correlation.

The Sella Turcica (Some Lesser Known Dynamic Features). Mohammad Sarwar, M.D., and Leonard E. Swischuk, M.D., University of Texas Medical Branch, Galveston, Texas.

This exhibit emphasized some lesser known dynamic aspects of the sella turcica. Basically these include:

1. Infrequently recognized changing configurations in endocrinopathies and metabolic disease (*i.e.*, the rebound "cherry" sella of hypothyroidism, etc.)
2. Progressive sellar changes in certain chondrodystrophies and bony dysplasias (*i.e.*, the progressive growing sella of Hurler's syndrome and the dysplastic sella of neurofibromatosis).

3. The etiology of the elongated sella in conditions such as achondroplasia, arrested hydrocephalus, etc.
4. Progressive sellar changes (demineralization, dorsum and posterior clinoid erosion, enlargement) reflecting chronically increased intracranial pressure (from the infant to the adult) and
5. The problem of the J-shaped or Omega-shaped sella.

Complications of Abdominal Aortic Reconstructive Surgery: Roentgenologic Evaluation. William M. Thompson, M.D., Irwin S. Johnsrude, M.D., Donald C. Jackson, M.D., and Robert A. Older, M.D., Duke University Medical Center, Durham, North Carolina.

Complications following abdominal aortic surgery are infrequent, but sometimes fatal. These include vessel occlusion, infection and/or erosion of the graft with abscess formation, false aneurysm, fistula formation, and aortic rupture. Conventional abdominal radiography, barium studies, and angiography may determine the nature and extent of the complication and predict future therapy.

From 1969 through 1973, 35 of over 1,000 patients at Duke University Medical Center had complications following abdominal aortic surgery. Five of these had aortoenteric or paraprosthetic enteric fistulas. The problems associated with accurate diagnosis and the proper sequence of radiographic evaluation were illustrated.

Evaluation of Orbital Lesions. Alfred L. Weber, M.D., Richard L. Dallow, M.D., and K. Jack Momose, M.D., Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

This exhibit illustrated lesions of the orbit and eye seen at the Massachusetts General Hospital and Massachusetts Eye and Ear Infirmary in the last 2½ years. The material was drawn from 300 ultrasound studies of the orbit and 150 CAT scans, done during this period. These examinations were compared with conventional radiographic techniques, such as plain films, tomography and angiography. The main emphasis was placed on orbital lesions. Entities shown included tumors, inflammatory conditions, dysthyroid exophthalmos and orbital hemorrhage.

Breast Parenchymal Patterns as Index of Risk for Developing Breast Cancer. John N. Wolfe, M.D., Hutzel Hospital, Detroit, Michigan.

The exhibit concerned itself with the identification of patterns of breast parenchyma which in themselves indicate a risk for the development of breast cancer.

In a relatively asymptomatic population one can isolate 95 percent of breast cancers; for example, in approximately 50 percent of the population between the ages of 30 and 45 and approximately 60 percent of the population thereafter.

The criteria can be more selective, 78 percent of the carcinomas can be isolated in 30 percent of the population between the ages of 30 and 49 and 22 percent of the population thereafter.

Intrarenal Arterial Collaterals. Heun Y. Yune, M.D., and Eugene C. Klatte, M.D., Indiana University School of Medicine, Indianapolis, Indiana.

The old contention by some anatomists that the intrarenal branches of the renal artery are incapable of developing collateral channels because they are end arteries is proved to be wrong. Angiographic demonstration of intrarenal arterial collaterals was recently reported. In the past three years, the authors have encountered five patients who had significant stenosis of either the main renal artery or renal artery branches which was associated with primarily intrarenal collateral circulation. The angiographic anatomy, and the incidence and clinical significance of these collaterals were evaluated in this exhibit.

Other Excellent Exhibits Were:

Pelvic Masses in Children. Ehsan Afshani, M.D., and Jerald P. Kuhn, M.D., Children's Hospital of Buffalo, Buffalo, New York.

This exhibit was designed to demon-

strate the classification and types of common pelvic masses that can occur in children and the roentgenographic demonstration of masses by virtue of other adjacent structure abnormalities. The use of ultrasound also facilitates the exact location and type of masses. The importance of the use of all roentgenographic modalities as well as ultrasound was recommended to help the surgeon for proper management.

The Arterial Blood Supply to the Liver in Cirrhotic Patients. Jeanne W. Baer, M.D., St. Luke's Hospital Center, New York, New York.

The manner in which the liver derives its arterial supply was studied in 25 noncirrhotic patients and 20 cirrhotic patients. Although some arterial supply was derived from the superior mesenteric artery through the gastroduodenal arcade in 2/25 noncirrhotic patients, this was a much more common finding in cirrhosis, 8/20. The degree of collateral flow to the liver from the superior mesenteric artery was greater in cirrhosis as demonstrated by the failure to fill the gastroduodenal artery during a celiac angiogram in 4/20 cirrhotics. This phenomenon is thought to be related to the number and size of intrahepatic arterioportal shunts.

Diagnostic Radiological Health Sciences Learning Laboratory. Reynold F. Brown, M.D., The American College of Radiology, San Francisco, California.

The Diagnostic Radiological Health Sciences Learning Laboratory is an educational system which teaches the basic skills of the three components of a radiographic examination: (1) the selection of the patient for the examination; (2) the conduct of the examination; and (3) interpretation of the results. Materials are provided for the specific educational needs of each of these components.

The lectures in the "Selection of Patients for X-ray Examinations" syllabus present the information needed by physicians to make informed patient selection decisions. The "Correlated Physics-Lecture Laboratory Series" provides radiology residents and others involved in the performance of radiographic examinations the physics background necessary for the proper operation of x-ray equipment. Interpretation is taught through use of the Learning File which offers a simulated experience in clinical radiographic interpretation.

The Use of Diagnostic Ultrasound in the Detection of Acute Rejection of Renal Allografts. Vlastimil Capek, M.D., University of Illinois, Chicago, Illinois.

For assessment of the immediate postoperative course of patients with renal transplant, a routine sequence of ultrasonic studies is very useful for early detection of acute rejection. The standardized technique allows comparative evaluation of changes in the size of the transplant, strength of interfaces and quality of calyceo-pelvic echoes.

Observed changes make diagnostic and prognostic evaluation of rejection episodes possible. Occasionally ultrasound findings are the earliest indication of this complication. Experiences with more than 70 cases were presented.

Selective Arterial Embolization for Control of Gastroduodenal Hemorrhage. Vincent P. Chuang, M.D., and Stewart R. Reuter, M.D., University of Kentucky Medical Center, Lexington, Kentucky; and Wayne County General Hospital and University of Michigan, Eloise, Michigan.

Twenty-one patients with massive upper gastrointestinal hemorrhage were treated by embolization over a two year period. The over-all success rate was 76 percent (16/21) patients. Excluding patients with gastritis, 15 patients had active arterial bleeding sites. In 14 of these, bleeding was controlled, a success rate of 93 percent. The technique and representative cases were illustrated.

Case of the Day—Adult. James L. Clements, Jr., M.D., William E. Torres, M.D., and Marjorie B. McSweeney, M.D., Emory University, School of Medicine, Atlanta, Georgia.

Each day a different case in the field of

adult radiology was presented. The answer was given the following day along with a description of the clinical condition, and a discussion of the entity. For those making the correct diagnosis, a winner's badge was awarded.

Gastric Pseudo-Ulcers: Membrana Angularis and Pyloric Torus Defects. J. L. Clements, Jr., M.D., William E. Torres, M.D., Patrick W. Peavy, M.D., and H. Stephen Weens, M.D., Emory University, School of Medicine and Grady Memorial Hospital, Atlanta, Georgia.

The membrana angularis and pyloric torus defects are two physiologic bulges which can simulate ulceration on the lesser curvature of the stomach. The features were reviewed in an attempt to facilitate recognition on the gastrointestinal examination.

Spontaneous Internal Drainage of Pancreatic Pseudocysts. J. L. Clements, Jr., M.D., James A. Johnson, III, M.D., S. Boyd Eaton, M.D., and H. Stephen Weens, M.D., Emory University, School of Medicine and Grady Memorial Hospital, Atlanta, Georgia.

Spontaneous communications of pancreatic pseudocysts with the gastrointestinal tract are rarely demonstrated. Barium contrast documentations of such communications with the esophagus, stomach, duodenum and colon were presented. Ultrasound documentations of pancreatic disease were included in some cases.

Individualized Computer Tomography of the Skull with the EMI Scanner Using the 160×160 Matrix. Steven H. Cornell, M.D., Jacob J. Musallam, M.D., Ching L. Chiu, M.D., and James H. Christie, M.D., University of Iowa Hospitals, Iowa City, Iowa.

The manufacturer's new program with 160×160 absorption coefficients being computed instead of the original 80×80 results in images of greatly improved resolution. Additionally, it is now possible to view the sections while the patient is still on the table. This enables the examiner to individualize the procedure by repeating unsatisfactory slices immediately, obtaining sections at different levels or with the head positioned differently, or injecting contrast material. Lesions initially seen poorly or not at all have been demonstrated following these modifications. This individualized approach was illustrated with numerous examples.

Glomus Jugulare Tumors: The Value of Lateral Tomography. D. Beatty Crawford, M.B., J. Powell Williams, M.D., and P. Noel Connaughton, M.B., University of Connecticut, School of Medicine and Health Center, Farmington, Connecticut; and the University of Maryland, Baltimore, Maryland.

The radiological diagnosis of glomus jugulare tumors is usually made by utilizing multidirectional tomography and vascular studies. Most emphasis has been placed on anteroposterior and modified submentovertical tomography, but in this study the exhibitors stressed the value of lateral tomography. There is a wide range of normal measurements on the anteroposterior and submentovertical views, but in this series they found this range to be much less on lateral views and felt that both quantitatively and qualitatively the lateral view is a valuable adjunct to the other radiological investigations.

This exhibit featured the normal and abnormal tomographic appearances of the jugular canal and quantitive measurements obtained from a series of lateral views.

Thorium Induced Hepatic Hemangioendothelioma: Roentgen-Angiographic Findings in Two Additional Cases with Emphasis on Recognition of Thorotrast "Carriers." Joseph L. Curry, M.D., William G. Johnson, M.D., David H. Feinberg, M.D., and John H. Updegrove, M.D., Easton Hospital, Easton, Pennsylvania.

Two additional cases of thorium related hepatic hemangioendothelioma were shown with correlated roentgen-angiographic findings and pertinent histology. The rarity of spontaneous hemangioendothelioma was emphasized. Renewed interest has been stimulated by the recently recognized rela-

tionship between this malignancy and exposure to vinyl chloride in industry.

The authors concluded that the number of human thorotrast "carriers" is unknown and represents a rapidly disappearing group of individuals. All physicians reviewing chest and abdominal roentgenograms should be aware of the characteristic appearance of thorium deposits. Angiography of the "thorotrast liver" may provide information of prognostic and therapeutic value.

Sequelae of Stomach Surgery. Anne Curtis, M.D., and Morton Burrell, M.D., Yale-New Haven Hospital, New Haven, Connecticut.

This exhibit included a number of common and rare radiographically identifiable complications following gastric surgery.

Epidural Venography in Herniated Lumbar Intervertebral Disks. Richard H. Daffner, M.D., and George F. Drasin, M.D., University of Louisville, School of Medicine, Louisville, Kentucky.

Epidural venography of the lumbar veins is a simple and relatively safe procedure for the diagnosis of herniated lumbar intervertebral disks. The accuracy equals that of myelography and exceeds it in the diagnosis of lateral herniations. It is especially useful in symptomatic patients who have a normal myelogram. A significant number of herniated disks have been found in this regard. The authors feel the study should be performed in every patient with symptoms who have a normal myelogram.

Areae Gastricae. Gerald D. Dodd, M.D., Harvey Goldstein, M.D., James Anderson, Ph.D., Lee Rogers, M.D., and Tom Harle, M.D., University of Texas System Cancer Center; M. D. Anderson Hospital and Tumor Institute; and University of Texas Medical School at Houston—Herman Hospital, Houston, Texas.

The areae gastricae (fine gastric mucosal relief) consists of tiny ovoid and/or polygonal areas measuring up to several millimeters in diameter. The pattern is visualized radiographically when barium enters the shallow grooves surrounding the areae, thereby producing a "network" appearance. The radiographic appearance of the fine relief pattern was presented and correlated with the anatomic appearance seen with gross specimens and scanning electron microscopy. Factors involved in the radiologic delineation of the pattern including patient characteristics and examination techniques were discussed. The possible clinical significance in the diagnosis of gastritis and infiltrative diseases of the stomach was presented.

The "Coat and Drain" Method of Demonstrating Ulcers, Non-malignant, and Malignant Lesions of the Gastrointestinal Tract. John Jack Douglas, M.D., St. Thomas Hospital, Akron, Ohio.

Many lesions, whether they are early or late, large or small, benign, or malignant, are readily obscured and missed by the usual techniques used for the "initial" x-ray study.

This exhibit demonstrated the desirability of obtaining routine mucosal relief delineation, and by "coating" the lesion, and obtaining "drain" films, more readily depicting the presence of ulcers, non-malignant changes (regional ileitis, enteritis with ulcerations, ulcerative colitis, superficial gastric erosive ulcerations), and malignant lesions as to the size, extent, character, and location in the gastrointestinal tract.

The emphasis was on malignant changes —particularly those involving the stomach.

Pulmonary Manifestations of Sarcoidosis. Richard D. Dunbar, M.D., John E. Hodgkin, M.D., and Lester Medford, M.D., Loma Linda University, Loma Linda, California.

Examples of the four main types of pulmonary sarcoid were shown with a description of each. Significant diagnostic data, clinical and laboratory findings of pulmonary sarcoid were described.

Aneurysmal Bone Cysts. D. Dyck, M.D., M. Hamonic, M.D., and R. I. MacPherson, M.D., University of Manitoba, Winnipeg, Manitoba.

Aneurysmal bone cysts are benign, solitary, expansile, cystic tumors found in the

vertebrae, flat and cylindrical bones.

In this exhibit, the clinical, radiologic and pathologic features of aneurysmal bone cysts were reviewed along with the differential diagnosis and treatment.

It was concluded that aneurysmal bone cysts are found mostly in the axial skeleton; some have characteristic radiologic signs that will permit a preoperative diagnosis, and radiation therapy is effective in large, difficult to excise lesions.

Reconstructed Images of Computer Generated Tomography Data. Robert A. Evans, M.D., L. Anne Hayman, M.D., Lawrence A. Larsen, M.D., and Robert S. MacIntyre, M.D., Baylor College of Medicine and the Methodist Hospital, Houston, Texas.

A new technique has been developed for the presentation and evaluation of images from computerized tomography (CT). Designed to help the radiologist assimilate the high resolution data generated by the computer, the system presents a picture containing digital and analog information simultaneously. Multi-level gray shades provide anatomic definition with each gray shade printed as an easily readable number. These optically weighted numbers provide the ability to instantly evaluate material densities over a significantly increased range. This technique definitely speeds interpretation of the data with enhancement of sensitivity and specificity.

Rheumatoid Arthritis: Usual and Unusual Manifestations. Pieretta S. Ferro, M.D., Luceil B. North, M.D., Martin D. Lidsky, M.D., F. Gyorkey, M.D., and K. W. Min, M.D., Veterans Administration Hospital, Houston, Texas.

This exhibit presented a review of the changes of rheumatoid arthritis, ranging from soft tissue swelling, cortical thinning and erosion to the most severe destructive changes in the extremities, spine and lung.

Pathology slides accompanied some of the radiographs.

Arthrographic Demonstration of Sealed Peripheral Separation of the Meniscus of the Knee. Edgar J. Filson, M.D., Raul S. Buelvas, M.D., H. Clark Deriso, M.D., Thomas L. German, M.D., Andrew T. Sheils, M.D., and Leslie L. Wilkes, M.D., Candler General Hospital, Savannah, Georgia.

The normal anatomy of the coronary ligament of the knee and the arthrographic signs of peripheral separation of the meniscus from this ligamentous attachment are demonstrated. These separations usually seal radiographically producing distinctive signs on the arthrogram but leading to continued knee disability. Their recognition is of extreme importance to both the radiologist and the orthopedic surgeon.

Radium Osteitis. John J. Fitzpatrick, M.D., and Roger D. Pinc, M.D., Cook County Hospital, Chicago, Illinois.

Roentgenograms of a 72 year old female with the world's highest known body burden of ingested radium (almost three millicuries) were presented and showed systemic changes with pathologic fracture and non-union requiring amputation.

Chest Wall Tumors in Infants and Children. E. A. Franken, Jr., M.D., and John A. Smith, M.D., Indiana University Medical Center, Indianapolis, Indiana.

Masses of the chest wall, uncommon in the pediatric age group, were classified by location, roentgen appearance, and histology:

1. Subcutaneous—benign and malignant
2. Bony lesions associated with generalized disease (*e.g.*, multiple exostosis, histiocytosis X)
3. Osseous tumors
   a. Benign
   b. Malignant—primary and metastatic
4. Extrapleural masses—benign and malignant

Their conclusions were that:

1. Metastatic tumors of the rib cage are much more common than primary neoplasms
2. Ewing's sarcoma is the most common primary osseous malignancy of the thoracic cage
3. Most chest wall masses unassociated

with generalized bone disease are malignant.

Epicardial Fat Line in Left Pleural Effusions. David Frolich, M.D., James L. Clements, M.D., and H. S. Weens, M.D., Emory University, Atlanta, Georgia.

This exhibit demonstrated the usefulness of visualization of the epicardial fat line in the diagnostic evaluation of pleural effusion on the left.

Case of the Day—Pediatric. Brit B. Gay, Jr., M.D., Turner Ball, Jr., M.D., and George O. Atkinson, M.D., Emory University School of Medicine, Atlanta, Georgia.

A different pediatric case was presented daily with pertinent clinical history and significant radiographs. The correct diagnosis was given the following day, along with a discussion of the condition. Correct diagnosticians were rewarded.

Radiologic Features of Congenital Urethral Duplication: A Report of Six Cases. Brit B. Gay, Jr., M.D., William E. Torres, M.D., Frank G. Hryshko, M.D., John R. Woodward, M.D., and Samuel S. Ambrose, M.D., Henrietta Egleston Hospital for Children, Atlanta, Georgia.

Duplication anomalies of the urethra are rare. This exhibit illustrated variation in the anomaly. A simple classification and six new cases of urethral duplication were presented.

ACR Program of Audio-Visual Materials in Continuing Education. Eugene Gedgaudas, M.D., American College of Radiology, Chicago, Illinois.

Video-tape programs on selected radiological topics were shown using a TV receiver and video-tape player.

Venous Angiography of Hemodialysis Fistulas: Experience with 72 Studies. Louis Arnold Gilula, M.D., Tom Weinberg Staple, M.D., and Charles B. Anderson, M.D., Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri.

Venous angiography provides a means of visualizing both the arterial and venous sides of a hemodialysis fistula in out-patients, without inherent risks of vascular damage accompanying an arterial puncture. This exhibit is related to an RSNA presentation in 1974 and a journal article published in *Radiology*, June, 1975. The technique was described. Several examples demonstrated the use of this procedure.

The Use of Ultrasonography in the Study of Pleuro-Pulmonary Lesions. Antonio Carmelo Gonzalez, M.D., Narumol Srisuthapan, M.D., James Luther Clements, Jr., M.D., and Ellen Laird, R.T.T., Emory University School of Medicine and Grady Memorial Hospital, Atlanta, Georgia.

This exhibit emphasized the usefulness of A and B-mode ultrasonography in the following instances:

1. Differentiation of pleural fluid accumulation from fibrotic pleural thickening, tumor implants at the pleural border, and outlining pulmonary pathology adjacent to pleural effusions that cannot be visually resolved by chest roentgenology.
2. Localization of paracardiac, extrapericardial fluid accumulations.
3. Ultrasound guidance in draining pleural fluid accumulations and placement of chest drainages.

A diagrammatic illustration explained the method of obtaining the ultrasound scan. Each of six illustrative cases showed the x-ray, ultrasound, and a legend with the case's interpretation. A final frame was used for summary and conclusion.

Complementary Role of Conventional Neuroradiology and Computerized Tomography in Pediatrics. C. F. Gonzalez, M.D., S. H. Lee, M.D., N. Thomas LaCava, M.D., M. Mitchell, M.D., M. Katz, M.D., P. N. Laffey, M.D., J. G. Teplick, M.D., and M. E. Haskin, M.D., Hahnemann Medical College and Hospital and Temple University Medical School, Philadelphia, Pennsylvania.

Correlation between conventional neuroradiology procedures in infants and children and the CT Scan was presented. The

cases were selected as having presented difficulties in interpretation that were easily overcome with the help of the CT Scan.

Angiographic Results of Microsurgical Bypass for Cerebral Ischemia. Gerald L. Grube, M.D., and Joseph R. Thompson, M.D., Loma Linda University Medical Center, Loma Linda, California.

The anastomosis of the superficial temporal artery to the middle cerebral artery is a recently developed microneurosurgical procedure for the treatment of cerebral ischemia. Sixty-six patients with transient ischemic attacks and internal carotid or middle cerebral artery occlusive lesions were studied angiographically before and (of these) 46 after STA to MCA anastomosis. This exhibit reviewed the clinical indications, angiographic evaluations, and the clinical and angiographic results in those 66 patients. Significant improvement in cerebral blood flow and good to excellent results were achieved in 85 percent, with an anastomosis patency rate of 85 percent.

Hydrostatic Reduction of Ileocecal Intussusceptions. Francis J. Y. Hahn, M.D., and Rolf L. Schapiro, M.D., University of Iowa Hospitals, Iowa City, Iowa.

This exhibit illustrated the hydrostatic reduction of ileo-cecal intussusceptions.

This included:

1. Critical review of new techniques.
2. Cautions and responsibilities of radiologists.
3. Complications and evaluation of results.
4. Clinical and pathologic correlations.
5. Contraindications and indications with case demonstrations.

Roentgen Signs of Basal Skull Fracture. Francis J. Y. Hahn, M.D., and Kenneth D. Dolan, M.D., University of Iowa Hospitals, Iowa City, Iowa.

This exhibit illustrated direct and indirect signs of basal skull fractures.

These include:

1. Direct signs
   - A. Interruption of cortical line
   - B. Distortion of sella turcica
   - C. Elevation of petrous apex
   - D. Interruption of occipital squama
2. Indirect signs
   - A. Opaque sphenoid sinus with air-fluid levels
   - B. Increased density of mastoid pneumatization
   - C. Air in subarachnoid space.

Uncommon Mediastinal Masses. Francis J. Hahn, M.D., and Rolf L. Schapiro, M.D., University of Iowa Hospitals, Iowa City, Iowa.

The radiographic manifestations of various uncommon mediastinal masses were presented. These included:

(A) Anterior and middle mediastinal masses.

1. Cavernous hemangioma
2. Angiofollicular lymphoid hamartoma (Kastleman's disease)
3. Empyema
4. Primary intrathoracic seminoma
5. A huge nodular goiter
6. Thymolipoma (2 cases)

(B) Posterior mediastinal masses.

1. Mycotic aneurysm of descending aorta
2. Esophageal varices
3. Leiomyoma of esophagus and stomach
4. Extramedullary hematopoiesis
5. Bochdalek hernia in adults.

Plain Film Diagnosis of Massive Gastrointestinal Hemorrhage. Sang Y. Han, M.D., David M. Witten, M.D., and H. S. Prim, M.D., University of Alabama School of Medicine, Birmingham, Alabama.

Four cases of massive bleeding into the stomach and one into the colon were correctly diagnosed on plain films of the abdomen. Four patients were referred for angiographic localization of the bleeding lesion. One patient had emergency upper gastrointestinal series with barium meal.

Radiological findings suggestive of bleeding in all cases were mottled, bubbly radio-

lucencies confined to the lumen of either the stomach or the colon. History revealed that none of the patients received anything by mouth for more than a week prior to the examination, excluding the possibility of retained food in the stomach and fecal material in the colon.

Recognition of these characteristic findings on plain films facilitates appropriate management of this life-threatening complication.

The Calcified Myocardial Infarct: Value of the Lateral Chest Roentgenogram. Robert E. Hanson, M.D., and Robert Lisbona, M.D., Royal Victoria Hospital, Montreal, Canada.

Reports of radiologically evident calcified myocardial infarcts have been infrequent and, with few exceptions, limited to single case reports. The authors have observed 7 cases over the past two and a half years; in 6 of these, the correct diagnosis was made by recognition of a curvilinear calcification visible only on the lateral chest roentgenogram. This feature has not been emphasized in previously reported cases; a factor which may account for its infrequent clinical recognition in the past.

The clinical material of these cases, roentgen findings and differential diagnoses were presented, as well as a brief review of the coronary occlusive pattern which results in this unusual condition.

The true incidence of calcified infarcts is undoubtedly higher than the literature indicates. With attention to the lateral projection, chest roentgenology can play a key role in increasing the clinical recognition of this entity.

Dislocations and the Radiologist. Norma F. Harris, M.D., Edward Knudson, M.D., and Robert S. McIntyre, M.D., Baylor College of Medicine and Shrine Hospital for Crippled Children, Houston, Texas.

Dislocations of interest to the practicing radiologist were selected from the many that have presented at Ben Taub Hospital and Baylor Affiliated Hospitals over the past seven years. These were selected because of their rarity, their complications and associated injuries, their subtle radiographic findings and their association with other pathology or disease states.

The Myelography Needle: A New Cannula for Aspiration. Vincent C. Hinck, M.D., Baylor College of Medicine, Houston, Texas.

Cuatico developed a cannula for aspiration of radiopaque substance through a standard spinal needle at the conclusion of positive contrast myelography. It was designed to avoid the severe nerve root pain often caused by aspiration through the spinal needle alone. Use of his cannula has certainly lowered the incidence of pain but has not eliminated it. The author suggests a reason for this and on the basis of which he designed a new cannula. It has undergone clinical trial with highly encouraging results and will soon be available commercially.

The Ligamentous Anatomy of the Ankle: A Roentgenographic Study. Jeremy J. Kaye, M.D., and Walther H. O. Bohne, M.D., Hospital for Special Surgery, New York, New York.

The major ankle ligaments were correlated with the osseous structures seen on standard radiographs by opacifying dissected ligaments with powdered tantalum. The anatomical data thus obtained were correlated with clinical ankle arthrograms.

A Roentgenographic Anatomy of the Wrist Utilizing Photographic Addition Techniques. Jeremy J. Kaye, M.D., and Walther H. O. Bohne, M.D., Hospital for Special Surgery, New York, New York.

The precise location of the various carpal bones on standard radiographs was studied in dissected specimens using photographic addition. This information facilitates interpretation of wrist roentgenograms.

Radiologic Spectrum of Amyloidosis in the Thorax. Judith Kossoff, M.D., and Steven K. Teplick, M.D., Boston University Medical Center, Boston Massachusetts.

The manifestations of amyloid disease in the thorax were discussed and the major

radiographic findings presented.

The Radiologic Spectrum of Amyloidosis in the Gastrointestinal Tract, Skeleton and Kidney. Judith Kossoff, M.D., and Stephen Teplick, M.D., Boston City Hospital, Boston, Massachusetts.

Patients with primary and secondary amyloidosis admitted to the Boston City Hospital over the past five years were presented with special emphasis placed on manifestations of the disease as related to three organ systems.

Conditions Simulating Fractures of the Atlas and Axis. Richard H. Laib, M.D., John A. Gehweiler, Jr., M.D., and William Clark, M.D., Duke University Medical Center, Durham, North Carolina.

In evaluating injuries of the upper cervical spine, consideration should be given to conditions simulating fractures. An illustrative—descriptive distillation of fractures and borderlands of the normal was presented.

Bilateral Pulmonary Hypoplasia in the Newborn. John C. Leonidas, M.D., Richard A. Fellows, M.D., and Eugene C. Beatty, M.D., Children's Mercy Hospital, Kansas City, Missouri.

Pulmonary hypoplasia in the newborn may be unilateral or bilateral and it may cause high morbidity and mortality. The various causes of pulmonary hypoplasia were presented, including the effects of maternal oligohydramnios, renal agenesis or severe dysplasia, skeletal disease and diaphragmatic hernia. Clinical material was illustrated with special emphasis on pathology and radiologic manifestations of neonatal pulmonary hypoplasia.

Breast Specimen Radiology. J. David Lewis, M.D., Katherine Shaffer, M.D., Edward Wilkinson, M.D., and John R. Milbrath, M.D., Medical College of Wisconsin, Milwaukee, Wisconsin.

Increased use of mammography has led to detection of more clinically occult breast cancers. When a cluster of calcifications discovered by mammography is the only sign of malignancy, the surgeon must be certain that these calcifications are present in the biopsy specimen. Specimen radiography is essential to assure removal and histological examination of the suspicious area seen on mammography.

Infusion Tomography of the Gallbladder in Acalculus Cholecystitis. Leon Love, M.D., Michael Shannon, M.D., Jose Manuel Cardoso, M.D., Lizandro Elizando, M.D., and Rogelio Moncada, M.D., Loyola University of Chicago, Stritch School of Medicine, Maywood, Illinois; and Hospital General Centro Medico Nacional I.M.S.S., Mexico City, Mexico.

Twelve cases of pathologically proved acalculus cholecystitis were presented. Radiographs of the patient's positive infusion tomography of the gallbladder, along with photographs of the specimens, were shown.

The Sella Turcica in Childhood Endocrine Disease. Kathleen McCarten, M.D., Lawrence R. Kuhns, M.D., Andrew K. Poznanski, M.D., and John F. Holt, M.D., C. S. Mott Children's Hospital, University of Michigan Medical Center, Ann Arbor, Michigan.

Diagnostic radiographic changes in the size and configuration of the sella turcica in endocrine conditions and tumors of childhood were shown.

Breast Screening Results After Two Years and 10,000 Patients. John R. Milbrath, M.D., Katherine A. Shaffer, M.D., J. David Lewis, M.D., and Lawrence Clowry, M.D., Medical College of Wisconsin, Milwaukee, Wisconsin.

Since July, 1972, the Medical College of Wisconsin has conducted one of the 27 Breast Cancer Screening Demonstration Projects sponsored jointly by the American Cancer Society and National Cancer Institute.

Ten thousand asymptomatic screenees over the age of 35 have been evaluated with mammography, thermography, and physical examination, in addition to patient history. All examinations were interpreted independently of the others.

The results of the first two years were presented. Results to date show one cancer is found in every 150 patients and that it is

very likely to be "early" with greater potential for cure.

Angiography in Vascular Trauma. Kamal Mojab, M.D., Leonard Lim, M.D., and Justo Rodriguez, M.D., Cook County Hospital, Chicago, Illinois.

Angiography plays an important role for accurate diagnosis of and better management in vascular trauma. Illustrative cases supported this contention.

Peritoneography: Experimental and Clinical Experience in Detecting Intra-abdominal Abscesses. Rogelio Moncada, M.D., Robert Churchill, M.D., Jack Pickleman, M.D., Jose Manuel Cardoso, M.D., Hyo H. Byun, M.D., Kenji Kimura, M.D., and Leon Love, M.D., Loyola University Medical Center, Stritch School of Medicine, Maywood, Illinois.

Experimental and clinical examples utilizing the combined technique of double contrast peritoneography and radioisotope imaging in diagnosing intra-abdominal abscess, as well as data of tissue counting of radioactivities, were displayed.

Wedged Hepatic Venography with a Balloon-Catheter. D. Novak, M.D., G. H. Bützow, M.D., and K. Becker, M.D., University Hospital Eppendorf, Hamburg, Federal Republic of Germany.

In patients with end-to-side portacaval shunt a SWAN-GANZ balloon-catheter was introduced into hepatic veins *via* the femoral or cubital vein. The blockade was achieved by insufflation of 1–2 ml of air in the balloon of the catheter. Blocked hepatic veins were opacified by injection of 10–20 ml of TELEBRIX at a flow rate of 4–5 ml/sec. Films were taken at 1 sec. intervals over a 12 sec. period. Wedged hepatic venography was performed in order to study following parameters: (a) hepatic vein filling pattern; (b) sinusoidal filling pattern; (c) filling of portal vein branches; and (d) presence of collateral veins between hepatic and splanchnic or central venous system.

Does Man Have Tumor Antibodies? Captain Charles W. Ochs, Medical Corps, US Navy, and Captain J. P. Smith, Medical Corps, US Navy, National Naval Medical Center, Bethesda, Maryland.

Radiographs showed unusual multiple small cavitating pulmonary nodules from metastatic squamous cell carcinoma of the cervix. Tumor immunology, concepts of isoimmune response, photomicrographs and gross tissue specimens were demonstrated.

Continuous Injection Supine Phlebography. Charles W. O'Dell, Jr., M.D., and Marc N. Coel, M.D., Veterans Administration Hospital and University of California, San Diego, San Diego, California.

A continuous injection supine technique of lower limb phlebography employing a long leg film changer, a Viamonte-Hobbs mechanical pressure injector, thigh cuffs, ankle tourniquet, and exercise is reliable, simply performed, painless, and has a low complication rate. It is well suited for use in patients who are acutely ill, partially or completely paralyzed, or subject to syncopal episodes which would make upright or semi-upright studies difficult. This technique gives superior filling of all the deep leg veins, the iliac veins, and the lower inferior vena cava. In a series of 100 consecutive patients it decreased the need for percutaneous femoral venographic studies to examine the iliofemoral veins and inferior vena cava from 37 to 11 percent.

Computerized Tomography of Intracranial Lesions. Enrique Palacios, M.D., Behrooz Azar-Kia, M.D., and Robert Churchill, M.D., Loyola University of Chicago and Foster G. McGaw Hospital, Maywood, Illinois.

The principle of computerized tomography was presented and illustrated with multiple cases. The method was correlated with conventional roentgenographic techniques and with pathological specimens. The use of infusion of contrast media with computerized tomography to demonstrate the lesions to better advantage was emphasized.

Neuroepithelial Cysts ("Colloid Cysts"). Are They Really of Paraphyseal Origin? Enrique Palacios, M.D., Behrooz Azar-Kia,

M.D., Albert V. Messina, M.D., and Robert Churchill, M.D., Loyola University of Chicago and Foster G. McGaw Hospital, Maywood, Illinois.

The controversial problem of the origin of the "colloid or paraphyseal cysts" was depicted. Anatomical studies have shown that the paraphysis is an extraventricular extension of the choroid plexus and derives from the neuroepithelium. Cysts arising from this structure should be referred to as neuroepithelial cysts. Unusual clinical and roentgenographic findings of these lesions which may occur in any portion of the ventricular system were presented.

The "Recto-Sigmoid Index." A Measurement for the Early Diagnosis of Hirschsprung's Disease. Rubem Pochaczevsky, M.D., and John C. Leonidas, M.D., The City Hospital Center at Elmhurst, Mt. Sinai School of Medicine, New York, New York; and the Children's Mercy Hospital and University of Missouri School of Medicine, Kansas City, Missouri.

The "Recto-Sigmoid Index" is an objective measurement which may prove useful in the early diagnosis of Hirschsprung's disease in the newborn, particularly in those cases where a colonic transition segment is not readily apparent or where its presence is subtle or questionable. On the basis of their current case material, a "Recto-Sigmoid Index" of less than one was highly suggestive of this entity. A "Recto-Sigmoid Index" of more than one or equal to one may indicate a normal colon or a condition mimicking Hirschsprung's disease such as the meconium plug syndrome.

Aortic Rupture: Angiographic and Plain Film Findings of the Left Extrapleural Apical Cap. David Robbins, M.D., Charles Putman, M.D., Joseph Simeone, M.D., Hideyo Minagi, M.D., and Jerry Fishbone, M.D., Yale University School of Medicine, New Haven, Connecticut.

By the time the clinical and usual radiographic evidence suggests an aortic rupture, the patient's chances for survival are reduced. Previously emphasized plain film findings—widening of the mediastinum, displacement of the trachea to the right, obscuration of the aortic knob, and depression of the mainstem bronchus—indicate mediastinal bleeding without necessarily alerting the clinician to the diagnosis of aortic rupture. The left extrapleural apical cap, present on plain film, may be one of the earliest, although previously unemphasized signs of traumatic aortic transsection. A probable mechanism for the appearance of the cap was demonstrated with reference to anatomical considerations and examination at autopsy.

The Dominican Republic Conjoined Twins. Henrietta Kotlus Rosenberg, M.D., Thomas J. Spackman, M.D., and Arnold Chait, M.D., The Children's Hospital of Philadelphia, Philadelphia, Pennsylvania.

The Dominican Republic Twins were of the pubopagus, tetrapus, omphalopagus type. The radiologic demonstration of their osseous structures, intra-abdominal vasculature, genitourinary and gastrointestinal tracts was presented.

The pubic bones were fused. Each baby had a septate vagina with a urethra draining into the lower end, normal uterus, fallopian tubes, and ovaries. The livers had virtually separate vasculature. Four normally positioned kidneys with each twin draining one ureter into its own bladder and the other into her sister's were present. Two separate gastrointestinal tracts from esophagus to low ileum with each ligament of Treitz in normal position were noted, and a short colon and the terminal ileum were shared.

Radionuclide Venography—An Effective, Simple, Rapid Procedure. U. Yun Ryo, M.D., Lelio G. Colombetti, Ph.D., and Steven M. Pinsky, M.D., Michael Reese Hospital and Medical Center, Chicago, Illinois.

A modified technique for radionuclide venography was developed to obtain simultaneous images of the superficial and the deep veins of the leg. Visualization of the

superficial veins has proved very useful, not only for detection of abnormalities in that group of veins, but also for a better interpretation of the venographic observations of the deep veins.

Also, a probable mechanism of positive venographic findings which indicated that delay in the clearance of radioactivity on a venogram represents a stasis due to incompetent venous flow or a turbulent flow at the area of thrombosis was studied.

Double-Blind Comparison of Meglumine Iodoxamate vs. Cholografin Meglumine. E. Nicholas Sargent, M.D., Alan H. Robbins, M.D., Miriam E. Vincent, M.D., William Boswell, M.D., Harvey I. Meyers, M.D., and Elizabeth A. Wickes, M.D., Los Angeles County/USC Medical Center, Los Angeles, California.

Meglumine iodoxamate (Cholovue) is a new intravenous cholecystocholangiographic agent which in both preclinical and early clinical trials has demonstrated a high degree of effectiveness and safety. Data from a double-blind comparison of this new agent to Meglumine iodipamide (Cholografin) were presented. Review of data from physiologic monitoring, as well as radiologic results, were shown.

Efficacy and Tolerance Study of Administration of C-Terminal Octapeptide of Cholecystokinin Following Oral Cholecystography. E. Nicholas Sargent, M.D., Andre Schulman, M.D., and Harvey I. Meyers, M.D., Los Angeles County/USC Medical Center, Los Angeles, California.

The C-terminal octapeptide fragment (OP-CCK) which has been synthesized at the Squibb Institute for Medical Research reproduces all the known biologic activities of the intact molecule of cholecystokinin. (Cholecystokinin is a 33 amino acid polypeptide.) Several potential clinical uses exist for OP-CCK. Particularly, it can be used to stimulate contraction of the gallbladder following cholecystography, and it may also be used to stimulate pancreatic secretion of pancreatic enzymes. A review of physiologic monitoring, as well as radiographic results, was demonstrated in a study of 40 patients.

Bolus Urography During Renal Failure. Harry Sassoon, M.D., Roger D. Pinc, M.D., Bashir Mandani, M.D., George Dunea, M.D., and E. R. N. Grigg, M.D., Department of Radiology, Cook County Hospital, Chicago, Illinois.

Forty patients in severe renal failure received fast injections (within five seconds) of 60 percent iothalamate at one cc per pound of body weight, proving both the feasibility and the usefulness of this procedure.

The Enlarging Head—Angiographic Evaluation of Megacephaly. Louis N. Scotti, M.D., Kenneth Maravilla, M.D., James W. Lecky, M.D., Donald R. Hardman, M.D., Khurshed Dastur, M.D., and Harold O. Barras, R. T., University of Pittsburgh, Pittsburgh, Pennsylvania.

The presence of a large head in the newborn or young child is usually a sign of severe central nervous system disease. The most common underlying denominator in these children is hydrocephalus—a clinical sign reflecting a derangement in the normal cerebrospinal fluid dynamics. Some of the many causes for hydrocephalus can be elicited through angiography. Some entities which produce an enlarged head will not have associated hydrocephalus; even in these cases, angiography can provide valuable information.

The traditional approach to the evaluation of megacephaly in young children is ventriculography. The authors presented their experience utilizing transfemoral cerebral angiography as the primary diagnostic special procedure in the evaluation of this group of patients.

Patients ranged in age from newborn to adolescence. All of the studies were performed utilizing the Seldinger technique of transfemoral catheterization, small puncture needles (20–22 gauge), and small catheter material (.038″ to .062″ outside diameter). Films were taken with alternate biplane magnification technique.

Congenital, traumatic, infectious, neoplastic, and vascular categories were presented.

Adverse Reactions to Intravascularly Administered Contrast Media: A Comprehensive Study Based on a Prospective Survey. William H. Shehadi, M.D., Richard C. Pfister, M.D., and Martin W. Donner, M.D., Westchester County Medical Center, New York; Massachusetts General Hospital, Boston, Massachusetts; and Johns Hopkins University Medical School, Baltimore, Maryland.

The material in this exhibit was drawn from 112,000 case reports obtained through a prospective survey.

This exhibit detailed the incidence of adverse reactions, types and severity, relation to procedure, age and sex distribution. The history of allergy and types of allergens, reactions to previous contrast media examinations, the role of pretesting, and the value of negative and positive results were presented.

Relative Perfusion: Prognosticator of Ischemic Ulcers. Michael E. Siegel, M.D., Buck A. Rhodes, Ph.D., C. Melville Williams, M.D., Frank Giargiana, M.D., and Henry N. Wagner, Jr., M.D., The Johns Hopkins Medical Institutions, Baltimore, Maryland.

Peripheral perfusion scans, utilizing intra-arterial injections of radioactive microspheres, were performed on 40 patients who had ischemic ulcers of the lower extremity. Point counting over the lesion and adjacent areas was utilized to quantitate the relative hyperemia of the lesion. When this relative hyperemia was at least 3.5:1, approximately 90 percent of the patients went on to heal their ulcer. In those without this degree of hyperemia, only approximately 10 percent eventually were able to heal their ulcer without surgical intervention. Assessment of relative hyperemia of the ulcer bed is a useful objective prognostic indicator of healing potential.

The Routine Air-Contrast Examination of the Esophagus. J. Skucas, M.D., and W. W. Schrank, M.D., University of Rochester School of Medicine and Dentistry, Rochester, New York.

A simple air-contrast technique for examination of the esophagus was illustrated. It involves use of an effervescent drink just prior to barium. There is no excessive dilution of barium, and the examination is performed just prior to the air-contrast views of the stomach. This technique allows demonstration of small tumors, ulcerations, or erosions in the esophagus. Distensibility of the esophagus is easily checked.

Nuclear Medicine Facility. Ernest G. Smith, Jr., M.D., and Abreu and Robeson, Inc., Crawford W. Long Memorial Hospital of Emory University, Atlanta, Georgia.

A floor plan and colored photographs of a new nuclear medicine facility were shown as examples for planning purposes.

Cancer Control Program Centers for Radiological Physics. Marilyn Stovall, B.S., University of Texas System Cancer Center and M. D. Anderson Hospital, Houston, Texas.

Activities of six Centers for Radiological Physics sponsored by the American Association of Physicists in Medicine involving quality control in medical physics related to diagnostic and therapeutic radiology were identified.

Control of Breast Cancer Through Mass Screening. Philip Strax, M.D., Guttman Institute, New York, New York.

Mortality from breast cancer in American women has not changed in 40 years, primarily because in the majority of instances the disease is not localized at the time of diagnosis. Persisting mortality reductions have been achieved in the Health Insurance Plan of Greater New York study because of earlier detection of the malignancy. The Guttman Institute demonstrates how a practical mass screening clinic using the *tandem technique* of palpation, mammography and thermography in fixed-site and outreach programs can be organized to examine large numbers of women.

Millirad Mammography for Mass Screening. Philip Strax, M.D., Guttman Institute, New York, New York.

This exhibit presented various combinations utilized in evaluating and achieving low dose mammography.

Use of Tomography in Medical Evaluation of Renal Lithiasis. Michael A. Sullivan, M.D., Robert Michael Smith, M.D., and Donald T. Erwin, M.D., Ochsner Clinic, New Orleans, Louisiana.

Tomography has been neglected in evaluating renal stones. The addition of tomography without contrast agents to the kidney, ureter or bladder allows the accurate demonstration of the number and size of stones present. Newer advances in the treatment of renal stones make it important to determine the metabolic activity of the process, and tomography is quite helpful in this. Tomograms can help determine whether the stones are changing in size or disappearing completely.

Intraorbital Calcification and Ossification. James L. Sundheim, M.D., and Marc S. Lapayowker, M.D., Temple University Health Sciences Center, Philadelphia, Pennsylvania.

The presence of intraorbital calcification can be a source of confusion to the radiologist and ophthalmologist. The exhibitors have attempted to characterize those most commonly seen and to discuss their significance.

Although calcification is the usual mode of calcium deposition in degenerate eye tissue, bone formation will occur if there is an adequate blood supply.

This exhibit showed examples of radiologically evident calcification and ossification arising from within the orbit and gave special attention to anatomic and etiologic correlation. Areas covered were cataract, phthisis bulbi, retinoblastoma, retrolental fibroplasia, foreign bodies, and primary intraorbital extraocular tumors.

"Pseudo" vs. "True" Dislocation of the Cervical Spine in Children (A Helpful Differentiating Line). Leonard E. Swischuk, M.D., University of Texas Medical Branch, Galveston, Texas.

It is often most difficult to diagnose unequivocally "pseudo dislocation" of the cervical spine in childhood. Step-off of the vertebral bodies may be so pronounced that it seems that true dislocation should be present. A helpful line was presented to aid in the solution of this problem. For the author the line has proved accurate, easy to apply and especially helpful in the emergency room setting.

"Doughnut" Sign in Brain Scanning. Y. Tarcan, M.D., W. Fajman, M.D., D. Berg, D.O., and Joseph A. Marc, M.D., Emory University Affiliated Hospitals, Atlanta, Georgia.

The presence of a ring-like lesion ("doughnut" sign) on the radioisotope brain scan and/or cerebral angiogram was first described as indicative for brain abscess. However, this was later shown to be erroneous. Cavitating tumors, infarctions, subdural hematomas, parasitic cysts, and even bone (calvarial) lesions can produce this sign.

The presence of the "doughnut" sign on one scan study does not insure its presence in another. Examples of this sign in different disease entities were illustrated.

The Gastrointestinal Manifestations of Hepatorenal Polycystic Disease: A Radiologic Study. Sidney Ulreich, M.D., Morton Burrell, M.D., and Robert M. Lowman, M.D., Yale University School of Medicine, New Haven, Connecticut.

It is not uncommon for genitourinary abnormalities to mimic gastrointestinal disease. The anatomic proximity of the kidneys to the intraperitoneal organs, together with reflex responses, are the basis for the gastrointestinal symptoms. Progressing deterioration of renal function may also produce gastrointestinal symptomatology. The pressure effects, from renal cystic masses of polycystic disease, on contiguous abdominal structures are readily demonstrated by gastrointestinal contrast studies.

This exhibit emphasized the significance

of the displacement phenomena produced by hepatorenal cystic disease.

Arteriographic Features of Renal Parenchymal Disease. Severiano Valenzuela, M.D., and Peggy Fritzsche, M.D., Loma Linda University, School of Medicine, Loma Linda, California.

Extra and intrarenal arteriographic patterns are diagnostically helpful in differentiation of renal parenchymal disease.

A detailed arteriographic analysis of chronic pyelonephritis, chronic glomerulonephritis, and arteriolar nephrosclerosis was made and compared to a normal magnified model. A summary of the illustrated discriminatory radiographic hallmarks was featured.

The Johns Hopkins Radiology Computer Reporting System. Paul S. Wheeler, M.D., and Donald W. Simborg, M.D., The Johns Hopkins Medical Institutions, Baltimore, Maryland.

Radiologists at Johns Hopkins Hospital generate their own reports directly by computer. Reporting speed and flexibility approach that of dictation following an initial learning period of two weeks. Support services include report delivery, storage and retrieval, statistics, and billing. A commercial stand-alone version of the system is being developed.

Practical Application of Physics to Design of General Purpose Angiographic Room. Alan Furness White, M.D., Georgetown University Hospital, Washington, D.C.

Angiography requires more expertise in the application of the principles of physics than any other field in diagnostic radiology. The author demonstrated room design, equipment selection, equipment modification of special purposes such as abdominal compression, simplified tube rating charts and radiation protection, all in terms of basic physics. There is no room which combines all the optimum qualities, but certain compromises are much more deleterious than others in terms of patient care, leading to poor quality radiographs and longer procedures.

Osteosarcoma: Value of Angiography in the Differential Diagnosis. Issa Yaghmai, M.D., Medical College of Virginia, Richmond, Virginia.

Angiographic features of 62 cases of proven osteosarcoma were analyzed to determine the usefulness of this procedure in differentiation of osteosarcoma from other bone lesions. The following findings were noted:

1. Osteosarcomas are the most vascular bone malignancy, regardless of the appearance of plain radiographs.
2. Hypervascularity may be mixed with areas of hypovascularity, the latter representing foci of necrosis, cartilage formation, or localized arterial occlusion.
3. The blood supply of the tumor is predominantly derived from its peripheral soft tissue vessels.
4. In addition to typical neovascularity, there is often evidence for rapid shunting of blood and early venous filling.
5. Angiography can clearly demonstrate the extension of the tumor into the soft tissues, invasion of the adjacent veins, and collateral circulation subsequent to venous obstruction due to either direct involvement or by tumor emboli.

These findings provide useful diagnostic criteria for differentiating osteosarcomas from other benign or malignant bone lesions which may simulate the pathologic appearance of the tumor.

Cerebellopontine Angle Masses: Radiologic-Pathological Correlation. Wen C. Yang, M.D., Smiljian Puljic, M.D., Norman E. Leeds, M.D., and Mannie M. Schechter, M.D., Albert Einstein College of Medicine, Bronx, New York.

Review of 200 cerebellopontine angle masses, accumulated during the years of their neuroradiological practice, showed the following entities: (1) neurinoma; (2) meningioma; (3) cholesteatoma; (4) arach-

noid cyst; (5) chordoma; (6) chondroma; (7) glioma; (8) hemangioblastoma; (9) chemodectoma; (10) aneurysm; (11) redundant vertebro-basilar artery; (12) arteriovenous malformation; (13) hematoma; and (14) solitary metastasis. The purpose of this exhibit was to bring to attention the wide gamut of mass lesions in the cerebellopontine angle by illustrating representative roentgenographic features with pathological correlation. Films of different radiologic modalities including cerebral angiography, pneumoencephalography, positive contrast cisternography, and computer assisted tomography were selected to best demonstrate each entity.

Byron G. Brogdon, M.D.
Chairman
Scientific Exhibits Committee

TECHNICAL EXHIBITS

The Technical Exhibits at the Seventy-sixth Annual Meeting of the American Roentgen Ray Society, were held at the Marriott Motor Hotel, Atlanta, Georgia, September 30–October 3, 1975.

The Exhibits were well prepared, well explained, and well attended. The Hotel arrangements permitted easy access by visitors who passed through the Technical Exhibits on the way to and from the Scientific Exhibits.

The Society wishes to express sincere thanks to the following firms which participated in this year's Technical Exhibits:

AGA Corporation, Secaucus, New Jersey; American Massage, Inc., Lighthouse Point, Florida; Ames Color-File Corporation, Somerville, Massachusetts; Barnes-Hind Pharmaceuticals, Sunnyvale, California; Bar-Ray Products, Inc., Brooklyn, New York; Charles Bessler, Company, Florham Park, New Jersey; Burkhart Roentgen, New York, New York; CGR Medical Corporation, Baltimore, Maryland; Churchill-Livingston, New York, New York; Cook Inc., Bloomington, Indiana; Cooper Scientific Corporation, Medi Tech Division, Watertown, Massachusetts; Data Service Agency, Maryland Heights, Missouri; Du Pont, Wilmington, Delaware; Eagle Enterprises, Woodbridge, New Jersey; Eastman Kodak, Rochester, New York; Educational Reviews Inc., Leeds, Alabama; Eimac, Division of Varian, Salt Lake City, Utah; Electro-Catheter Corporation, Rahway, New Jersey; Elema-Schonander Inc., Elk Grove, Illinois; Encyclopaedia Britannica, Inc., Chicago, Illinois; Eureka (Litton), Chicago, Illinois; E-Z-Em, Westbury, New York; C. B. Fleet Company, Inc., Lynchburg, Virginia; GAF Corporation, New York, New York; General Electric Company, Milwaukee, Wisconsin; Grumman Health Systems, Woodbury, New York; Grune & Stratton, Inc., New York, New York; Hewlett-Packard, McMinnville, Oregon; Walter H. Hopkins, Atlanta, Georgia; Humanetics, Inc., Carrollton, Texas; Kilgore International Inc., Coldwater, Michigan; Liebel-Flarsheim, Cincinnati, Ohio; Litton Medical Systems, Atlanta, Georgia; Logetronics, Inc., Springfield, Virginia; Low X-Ray, Valhalla, New York; Machlett Laboratories, Inc., Stamford, Connecticut; Mallinckrodt, Inc., Hazelwood, Missouri; Medical Data Association, New York, New York; Medrad Inc., Pittsburgh, Pennsylvania; Medx Incorporated, Palantine, Illinois; C. V. Mosby Company, St. Louis, Missouri; Neutron Products, Inc., Dickerson, Maryland; Ohio Nuclear, Solon, Ohio; Pako Corporation, Minneapolis, Minnesota; Pearl of the Week, Lexington, Kentucky; Pfizer, Inc., New York, New York; Philips Medical Systems, Inc., Shelton, Connecticut; Picker Corporation, Cleveland, Ohio; W. B. Saunders Company, Philadelphia, Pennsylvania; Frank Scholz X-Ray Corporation, Needham Heights, Massachusetts; Siemens Corporation, Iselin, New Jersey; Spectrum X-Ray Corporation, Westville, New York; Squibb Hospital Division, Princeton, New Jersey; S & S X-Ray Products, Inc., Brooklyn, New York; Charles C Thomas, Publisher, Springfield, Illinois; Unirad Corporation, Denver, Colorado; Universal Medical Instrument Corporation, Ballston Spa, New York; Williams & Wilkins Company, Baltimore, Maryland; Winthrop Laboratories, New York, New York; Xerox Corporation, Xeroradiography, Pasadena, California; Yearbook Medical Publishers, Chicago, Illinois.

NEWS ITEMS

GENERAL DIAGNOSTIC RADIOLOGY

There will be a seminar in Diagnostic Radiology entitled "General Diagnostic Radiology Updated," on March 5 and 6, 1976 a the Bowman Gray School of Medicine of Wake Forest University in Winston-Salem, North Carolina.

The Guest Faculty will include Drs. John Caffey, Robert Cooley, Elias Theros, and Mr. A. F. Naylor, Radiology Consultant.

For further information, please contact: I. Meschan, M.D., Department of Radiology, Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, North Carolina 27103.

SYMPOSIUM ON CURRENT CONCEPTS IN GASTROINTESTINAL AND BILIARY RADIOLOGY

The Department of Diagnostic Radiology of the University of Kentucky Medical Center in Lexington will present a Symposium on "Current Concepts in Gastrointestinal and Biliary Radiology," April 28–30, 1976.

The program is designed for the practicing radiologist, gastroenterologist and gastrointestinal surgeon. The format consists of brief lectures, panel discussions and case presentations. Ultrasound and computerized tomography techniques are covered in the program. Prior to the meeting the registrants will receive by mail sets of x-rays of the cases to be discussed. The Guest Faculty consists of Drs. Robert Berk, Joseph Bookstein, George Leopold, Roscoe Miller, Thomas Meany, Jerome Wiot, and Bernard Wolf.

The fee for the Symposium is $300.00.

For further information, please contact: Frank Lemon, M.D., Office of Continuing Education, College of Medicine, University of Kentucky, Lexington, Kentucky 40506.

RADIOLOGY OF THE GENITOURINARY TRACT

The Department of Radiology of Duke University Medical Center will present a course on "Radiology of the Genitourinary Tract," March 22–26, 1976, in Durham, North Carolina.

The course is designed for radiologists but is open to other physicians whether in training or practice. The emphasis will be on personalized, tutorial type teaching of basic subjects on the genitourinary tract by recognized authorities using their original roentgenograms with ample opportunity for discussion. The subject matter will cover all facets of genitourinary tract disease including comprehensive coverage of diagnostic techniques. A detailed abstract book with references will be provided.

The registration fee is $300.00 and enrollment will be limited to 100 registrants.

For further information, please write to: Robert McLelland, M.D., Department of Radiology, Box 3808, Duke University Medical Center, Durham, North Carolina 27710.

CONGRESS ON NUCLEAR MEDICINE

The First Congress on Nuclear Medicine, under the patronage of the European Nuclear Medicine Society and sponsored by the Swiss Society of Radiology and Nuclear Medicine, will be held May 12–15, 1976, in Lausanne, Switzerland.

Among the subjects to be considered are the diagnostic applications of the cyclotron, radioimmunoassays, whole body scans, kidney studies, kinetic models, thrombosis, and pulmonary dynamic studies. Simultaneous translation into English, French, and German will be available.

For further information, please write to: Dr. B. Delaloye, Division Autonome de Médecine Nucléaire, CHUV, 1011 Lausanne, Switzerland.

NEW TECHNIQUES IN DIAGNOSTIC IMAGING II

The Department of Radiology, University of Pittsburgh, will hold its second international course on "New Techniques in Diagnostic Imaging," April 8–10, 1976, at a ski resort, 70 minutes from Pittsburgh in the Laurel Mountains.

The course is designed to highlight the collaborative effort between radiologists and physicists—engineers in the imaging area. Subjects include electronic radiological imaging, computer signal processing, television, CT scanning, image processing, and new developments in ultrasound. Special attention will be given to applications in GI fluoroscopy, neuroradiology and neurosurgery, angiography, cardiology, interventional techniques, and the design of new CT scanners with evaluation of those in current use.

For further information, please write to: E. Ralph Heinz, M.D., Professor and Chairman, Department of Radiology, University of Pittsburgh, Pittsburgh, Pennsylvania 15261.

NEW YORK ROENTGEN SOCIETY SPRING CONFERENCE

The New York Roentgen Society will present its Spring Conference, April 22–24, 1976, in New York City.

Subjects and refresher courses will include the gastrointestinal tract, the skeleton, neuroradiology, the genitourinary tract, chest, pediatric radiology, radiation therapy and ultrasound. The course is approved for A.M.A. Category 1 Credits.

For further information, please write to: Albert A. Dunn, M.D., Department of Radiology, Roosevelt Hospital, 428 West 59th Street, New York, New York 10019.

PEDIATRIC GENITOURINARY RADIOLOGY

The Institute for Pediatric Radiology will offer a program for radiologists, pediatricians, urologists, obstetricians, and gynecologists. The meeting will be in New Orleans, Louisiana, on Saturday and Sunday, March 6 and 7, 1976.

The objectives include updating techniques, knowledge, and skills in pediatric genitourinary radiology; practical instruction; and clinical applications. All presentations will be made by Charles E. Shopfner, M.D.

For further information, please write to: Wanda Patton, Executive Secretary, Institute for Pediatric Radiology, 4148 North Cleveland Avenue, Kansas City, Missouri 64117.

INTEGRATED CANCER MANAGEMENT

The Good Samaritan Hospital, Division of Oncology, and the Arizona Division of the American Cancer Society will cosponsor a program covering integrated management of cancer, with exchange of ideas between radiation oncologists, medical oncologists and surgeons. The meeting will be held in Phoenix, Arizona, February 18–21, 1976.

Registration will be limited to 350, and the fee is $175.00.

For the detailed program and further information, please write to: Robert H. Thoeny, M.D., Program Chairman, Division of Oncology, Good Samaritan Hospital, 1033 East McDowell Road, Phoenix, Arizona 85006.

BOOK REVIEW

TRAITE DE RADIODIAGNOSTIC. Tome XVII: Temporal, Fosses Nasales, Cavités Accessoires. (Treatise on Diagnostic Radiology—the Temporal Bone, Nasal Fossae, and Paranasal Sinuses.) By J. Vignaud, G. L. Dulac, J. Francois, Y. Guerrier, P. Rabischong, Ph. Tennenbaum, and G. Valvassori. Cloth. Pp. 454, with many figures. Price, 540 F. Masson & Cie, 120 Boulevard Saint-Germain, Paris, France, 1974.

This multiauthored book is number XVII, Volume 1, in a series of 20 books on diagnostic radiology.

There are twenty-two chapters in two parts. Part 1, The Temporal Bone, includes chapters I–XVI. Part 2, The Nasal Fossae and Paranasal Sinuses, includes chapters XVII–XXII.

In the introduction, the authors review some of the historic background in the development and evolution of various phases and techniques of otology, culminating in microscopic cophosurgery (surgery of deafness), and the parallel development of "radio-otology." The surgeon, with special instrumentation, performs microsurgery and the radiologist uses magnification and multidirectional tomography to produce cuts of one millimeter thickness.

In the several chapters of Part 1, the subjects discussed include the anatomy of the temporal bone, of the nerve elements and blood supply of the bony structure and organs of hearing, functional studies of the ear, anatomical variations and various pathological conditions, trauma to the temporal bone, tumors of the temporal and the acoustic apparatus.

The conclusion at the end of Chapter III, on trauma to the temporal bone, places in true perspective the comparative value as well as the limitations of the clinical and radiologic methods of examination.

Part 2, chapters XVII–XXII, discusses the normal radiological anatomy, anatomical variation and malformation, fractures, cerebrospinal fluid rhinorrhea, foreign bodies, as well as the various pathological conditions including sinusitis and allergy.

This is a hard cover book, handsomely bound, with good quality paper, good printing, and excellent workmanship. The book is profusely illustrated. The illustrations are of unusually fine quality. These consist of photographs of anatomical specimens, line drawings, companion radiographs and micro-radiographs. Sections of anatomical specimens are made in multiple planes, some at 1/10 millimeter levels, bringing out exquisitely delicate and fine detail not hitherto obtained.

Legends with detailed descriptions and accurate labeling accompany each illustration.

The text flows freely and smoothly throughout, with none of the gaps or redundant overlaps of a multiauthored product.

The usefulness of this book would have been greatly enhanced if the text, or the legends were multilingual, or at least bilingual. English subtitles, often used in some European publications, would have insured wider circulation and greater utilization.

The unconventional location of the table of contents at the end of the book, following the index, is a bit distracting. Until it is found, the readers' search seems fruitless and frustrating.

This reviewer, whose teaching career started in the department of anatomy, before going into radiology, particularly appreciates the scholarly excellence and meticulous detail of this book. This work is an outstanding contribution. It may be too detailed and highly specialized for the general radiologist.

It is recommended as a reference book in the library of the general radiologist, but it is a *must* in the library of the anatomist, otologist, and the radiologist specializing in this area.

WILLIAM H. SHEHADI, M.D.

BOOKS RECEIVED

INDEX FOR ROENTGEN DIAGNOSES. Third edition. Prepared under the auspices of The American College of Radiology, Commission on Diagnostic Radiology; Division on Computer Applications in Diagnostic Radiology; Committee on Diagnostic Coding Index and Thesaurus. Co-chairmen, E. Robert Heitzman, M.D., and Harold G. Jacobson, M.D. Paper. Pp. 157. The American College of Radiology, 20 North Wacker Drive, Chicago, Ill. 60606, 1975.

HOSPITAL STATISTICS, 1975 EDITION. 1974 Data from the American Hospital Association Annual Sur-

vey. Paper. Pp. 205. American Hospital Association, 840 North Lake Shore Drive, Chicago, Ill. 60611, 1975.

Mammography, Thermography and Ultrasonography in Breast Disease. By Kenneth T. Evans, M.B., Ch.B., D.M.R.D., F.R.C.P., F.F.R., Professor of Diagnostic Radiology, Welsh National School of Medicine; Director, Department of Diagnostic Radiology, University Hospital of Wales; and I. Huw Gravelle, B.Sc., M.B., Ch.B., D.M.R.D., F.R.C.P., F.F.R., Consultant Radiologist, University Hospital of Wales; Clinical Teacher in Diagnostic Radiology, Welsh National School of Medicine. Paper. Pp. 148, with some figures. Butterworths, 2265 Midland Avenue, Scarborough, Ont., Canada M1P 4S1, 1975.

Fonction de Transfert de Contraste d'un Appareillage de Radiologie: Etude Expérimentale. By Docteur Pierre Girod. Paper. Pp. 61, with some figures. Association Corporative des Etudiants en Médecine de Lyon, 8, avenue Rockefeller, 69373 Lyon Cedex 2, France, 1975.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. James Franklin Martin, 300 S. Hawthorne Rd., Winston-Salem, N. C. 27103. Annual Meeting: Washington Hilton, Washington, D. C., Sept. 20–24, 1976.

AMERICAN RADIUM SOCIETY
Secretary, Richard H. Jesse, Dept. of Surgery, M. D. Anderson Hosp. and Tumor Institute, Houston, Tex. 77025. Annual Meeting: Bayshore Inn, Vancouver, B. C., Canada, May 9–14, 1976.

RADIOLOGICAL SOCIETYOF NORTH AMERICA
Secretary, Dr. Theodore A. Tristan, Harrisburg, Pennsylvania. Annual Meeting: McCormick Place on the Lake, Chicago, Ill., Nov. 30–Dec. 5, 1975.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.
Oral examinations will be held: Statler Hilton, Dallas, Texas, Dec. 8–13, 1975; Marriott Motor Hotel, Chicago, Ill., June 14–19, 1976; Sheraton Biltmore, Atlanta, Ga., Dec. 6–11, 1976.
Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.
Deadline for filing applications for any examination in 1976 is September 30, 1975.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.
Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Dr. Robert G. Waggener, Dept. Radiol., Univ. Tex. Health Sciences Center, San Antonio, Tex. 78284 Annual Meeting:

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wis., 54601. Annual Meeting:

AMERICAN NUCLEAR SOCIETY
Program Chairman, Werner K. Kern, IRD, Space Nuclear Systems, F 309, U. S. Atomic Energy Commission, Washington, D.C. 20545.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

FOURTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Rio de Janeiro, Brazil, Oct. 23–29, 1977.
President Elect: Prof. Dr. Nicola C. Caminha; *General Secretary:* Dr. Durval Couto. Av. Churchill, 97-50 and-LC-39 20,000 Rio de Janeiro, Brazil.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

TWELFTH INTER-AMERICAN CONGRESS OF RADIOLOGY
President, Dr. Publio Vargas Pazzos, Casilla 5227, Guayaquil, Ecuador. Meeting in Quito, Ecuador, 1979.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Manuel Viamonte, Jr., M.D., Mt. Sinai Medical Center, 4300 Alton Rd., Miami Beach, Fla. 33140.
Secretary, Gaston Morillo, M.D., Jackson Memorial Hosp., 1700 N.W. 10th Ave., Miami, Fla. 33152.
Counselor for the United States, J. A. del Regato, M.D., Univ. of So. Fla. College of Medicine, 13000 No. 30th St., Tampa, Fla. 33612.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Secretary-General, P.O. Box 14, 79 Lauriston Place, Edinburgh BHI IDB, Scotland.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Secretary, Dr. Buenaventura U. V. Angtuaco, Congress Secretariat Headquarters, 2161 Taft Ave., Manila, Philippines. Meeting: Manila, Philippines, Nov. 10–14, 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lawrence E. Fetterman, 1720 Springhill Ave., Suite 201, Mobile, Ala. 36604. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY, CHAPTER ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

AMERICAN OSTEOPATHIC COLLEGE OF RADIOLOGY
Annual Scientific Meeting: Americana Hotel, Bar Harbor, Fla. Program Administrator: Lawrence J. Jordan, D.O., Suite 21, Ponce American Bldg., 4601 Ponce de Leon Blvd., Coral Gables, Fla. 33146.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Irwin M. Freundlich, 1501 N. Campbell Ave., Dept. Radiol., Tucson, 85724. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. Richard Seibold, Jr., Wadley Hosp., Texarkana, Ark.—Tex. 75501. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Arch W. Templeton, Univ. of Kansas Med. Center, 39th and Rainbow Blvd., Kansas City, Kansas 66103. Annual Meeting.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. James H. Larose, South Fulton Hosp., 1170 Cleveland Ave., East Point, Ga. 30344. Meets 4 times during the academic year at an Atlanta Restaurant.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Bavarian-American Radiologic Society
Secretary, LTC. Marco J. DiBiase, M.D., Radiology Service, Heidelberg, 130 Station Hosp., APO N. Y. 09102. Meets quarterly.

Blockley Radiological Society
Secretary-Treasurer, Dr William H. Green, 616 East 24th St., Chester, Pa. 19013.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Ralph Brancaccio, 7901 4th Ave., Brooklyn, N. Y. 11209. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. George J. Alker, Jr., E. J. Meyer Mem. Hosp., Buffalo, N. Y. 14215. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. Duane J. Gillum, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Alfred Brettner, Auburn Mem. Hosp., Auburn, N. Y. 13021. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Daniel E. Lewis, 309 East State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, March, and May at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer Dr. E. Jerome Schoolar, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harold J. Lasky, 55 E. Washington St., Suite 1735, Chicago, Ill. 60602. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Charles M. Greenwald, 7007 Powers Blvd., X-Ray Dept., Parma, Ohio 44129. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. John Pettigrew, Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Robert R. Burns, 1400 Stemmons Ave., Dallas, Tex. 75208. Meets the 3rd Monday of every month at 6:30 P.M., at the Airport Hotel, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Ekkehard S. Schubert, Wilmington Med. Center, P. O. Box 1951, Wilmington, Del. 19899.

East Bay Radiological Society
Secretary-Treasurer, Dr. Richard H. Culhane, 4 Vista del Mar, Orinda, Calif. 74563. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary. Dr. Paul J. Popovich, 1350 S. Hickory St., Melbourne, Fla. 32901. Meets twice annually, in the spring with the Annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Herbert Johnson, Davis Blvd., Tampa, Fla. 33606. Meets on 4th Thursday of the months January, March, May, October, and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Jay I. Rozen, Suite 216, 6400 Prospect, Kansas City, Mo. 64132. Meets 5 times a year.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary, Dr. David C. Hillman, P. O. Box 610544, North Miami, Fla. 33161. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Michael J. McCabe, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:00 P.M.

Health Physics Society
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Robert D. Dooley, Hinsdale Med. Center, Hinsdale, Ill. 60521. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John A. Knote, Home Hosp., Lafayette, Ind. 47902.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Dale L. Roberson, 1948 First Ave., NE, Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific session is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph In-

firmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Harry L. Stein, North Shore Univ. Hosp., 300 Community Dr., Manhasset, N. Y. 11030.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Arthur F. Schanche, 333 North Prairie Ave., Inglewood, Calif. 90301. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference. Century Plaza Hotel, Los Angeles, Calif.

LOUISIANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

LOUISIANA-TEXAS GULF COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. J. Gardiner Bourque, 3155 Stagg Dr., Suite 230, Doctors Bldg., Beaumont, Texas 77701.

MAINE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Peter E. Giustra, Dept. Radiology, Penobscot Bay Medical Center, Rockland, Maine 04841. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

MASSACHUSETTS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Joseph T. Ferrucci, Jr., Massachusetts Gen. Hosp., Boston, Mass. 02114.

MEMPHIS ROENTGEN SOCIETY
Secretary-Treasurer, Dr. R. Randolph Ramey, Baptist Memorial Hosp., 899 Madison Ave., Memphis, Tenn. 38146. Meets first Monday Oct. through June at the University Club.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

MICHIGAN RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Francis P. Shea, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

MICHIGAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary-Treasurer, Dr. William I. Knapp, St. Mary's Hosp., 830 S. Jefferson, Saginaw, Mich. 48601. Meets bimonthly during the academic year.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Marvin E. Goldberg, Box 292, Mayo Memorial Health Sciences Center, Minneapolis, Minn. 55455.

MISSISSIPPI STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

MISSOURI RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

MONTANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

NEBRASKA CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert Bodmer, Suite 622 Doctors Bdg., Omaha, Neb. 68131. Meets the third Wednesday of the month, Oct. through March, at the Clarkson Hosp., Omaha, Neb. and the fourth Friday of April at the Lincoln Country Club, Lincoln, Neb.

NEVADA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

N.E. SOCIETY FOR RADIATION ONCOLOGY
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Melvin E. Clouse, 185 Pilgrim Rd., Boston, Mass. 02115. Meets the third Friday of Oct. and Nov., and third Thurs. of each month. Jan. through May, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass. at 3:00 P.M. Annual Meeting: May 20, 1976.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

NEW MEXICO SOCIETY OF RADIOLOGISTS, CHAPTER OF ACR
Secretary, Dr. W. M. Jordan, 1100 Central Ave. S.E., Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

NEW ORLEANS RADIOLOGICAL SOCIETY, INC.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference.

NEW YORK ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Thomas C. Beneventano, 111 East 210th St., Bronx, N. Y. 10467. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., April 22–24, 1976. A.M.A. Cat. I credit. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

NEW YORK STATE CHAPTER OF ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 6 Secor Dr., Port Wash., N. Y. 11050.

NORTH AMERICAN SOCIETY OF CARDIAC RADIOLOGY
Secretary-Treasurer, Dr. Erik Carlson, Univ. Calif., San Francisco, Calif. 94143.

NORTH CAROLINA CHAPTER OF ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

NORTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Marshall Landa, P. O. Box 6001, Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

NORTHEASTERN OKLAHOMA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Donald R. Morton, Dept. of Radiology, St. Clare's Hosp., Schenectady, N. Y. 12304. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. John D. Earle, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. Patrick J. Grinsell, 1207 Fairchild Ct., Woodland, Calif. 95695. Meets on the fourth Monday, Sept. through May at the Mansion Inn, Sacramento, Calif.

NORTHWESTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

NORTHWESTERN OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Richard W. Siders, 421 Michigan, Toledo, Ohio 43624. Meets 3rd Wednesday from September to May.

OHIO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. James Farmer, Cleveland, Ohio.

OKLAHOMA STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

ORANGE COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. William E. Danko, 2601 E. Chapman, Orange, Calif. 92667. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

OREGON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ray F. Friedman, 3324 S. W. 44th St., Portland, Ore. 97221. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Kenneth D. Moores, 1118 9th Ave., Seattle, Wash. 98101. Annual Meeting: Benson Hotel, Portland, Ore., May 7–9, 1976.

PENNSYLVANIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Joseph A. Marasco, Jr., St. Francis General Hosp., Pittsburgh, Pa. 19141.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. Marvin E. Haskin, 230 N. Broad St., Philadelphia, Pa. 19102. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. Klaus M. Bron, Presbyterian-Univ. Hosp., Pittsburgh 15213. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

RADIATION RESEARCH SOCIETY
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting.

RADIOLOGICAL SOCIETY OF CONNECTICUT, INC., CHAPTER OF ACR
Secretary, Dr. Gerald L. Baker, 85 Jefferson St., Hartford, Conn. 06106. Meets in Oct., Jan., March, and June.

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

RADIOLOGICAL SOCIETY OF NEW JERSEY, CHAPTER OF ACR
Secretary, Dr. Fred M. Palace, 11 Dale Dr., Morristown, N.J. 07960. Monthly meetings in East Orange and Trenton, N.J. Semi-Annual Meeting in Nov.

RADIOLOGICAL SOCIETY OF RHODE ISLAND, CHAPTER OF ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA
Secretary-Treasurer, Dr. A. Franklin Turner, LAC-USC Medical Center, Room 3418, 1200 North State St., Los Angeles, Calif. 90033. Meets three times a year, usually October, February and May.

RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

REDWOOD EMPIRE RADIOLOGICAL SOCIETY
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

RICHMOND COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Frederick S. Vines, 11830 Rothbury Dr., Richmond, Va. 23235. Meets third Thursday of each month at the Aberdeen Barn.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, Rochester General Hosp., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual Meeting: Brown Palace Hotel, Denver, Colo., Aug. 19–21, 1976.

SAN ANTONIO-CIVILIAN MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

SAN DIEGO RADIOLOGICAL SOCIETY
President, Dr. Donald J. Fleischli, 7849 Fay Ave., La Jolla, Calif. 92037. Meets the first Wednesday of each month Sept. through June at the University Club.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Robert L. Koch, 3838 California St., Suite 106, San Francisco, Calif. 94118.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION OF RADIOLOGY, NATIONAL MEDICAL ASSOCIATION
Secretary, Dr. Ruby Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broadway, Baltimore, Md. 21205. Annual meeting: Marriott Hotel, Atlanta, Ga., Sept. 28–29, 1975.

Society of Gastro-intestinal Radiologists
Secretary-Treasurer, Dr. John R. Amberg, University Hospital, 225 W. Dickinson St., San Diego, Calif. 92103.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual Meeting.

South Bay Radiological Society
Secretary, Dr. Robert Mindalzun, Valley Radiologists, Inc., 100 O'Connor Dr., San Jose, Calif. 95128. Meets the 2nd Monday of each month at the Santa Clara County Medical Society Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society Chapter of ACR
Secretary-Treasurer, Dr. Richard T. Love, Santa Barbara Med. Clin., 215 Pesetas Lane, Santa Barbara, Calif. 93110. Meets fourth Tuesday of alternate months, Sept., Nov., Jan., March and May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Association
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. J. W. Maxwell, P.O. Box 2144 Mobile Ala. 36601. Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 30–Feb. 1, 1976.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Jerry W. Grise, Methodist Hosp., 1265 Union Ave., Memphis, Tenn. 38104. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bdg., San Antonio, Tex. 78205.

The Fleischner Society
Secretary, Dr. E. Robert Heitzman, State University of New York Upstate Medical Center, Syracuse, N. Y. 13210. Annual Meeting:

Tri-State Radiological Society
Secretary, Dr. Thomas Harmon, St. Mary's Hosp., Evansville, Ind. 47750. Meets bimonthly on the third Wednesday at area Hospitals.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Patrick D. Lester, Dept. Radiol., University of Utah Medical Center, Salt Lake City, Utah 84112. Meets quarterly at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 3500 Kensington Ave., Suite 2-A, Richmond, Va. 23221.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Donald J. Hesch, 3216 N.E. 45th Place, Seattle, Wash. 98105.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Leonard Cutler, 16 Guion Place, New Rochelle, N. Y. 10802. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. June Unger, Wood VA Hosp., Wood, Wis. 53193. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wis. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Rafael Umaña-Umaña, Apartado 8-5340, San José, Costa Rica.

Asociacón de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9², Calle A 0–05, Zona 1, Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. César Rosa, Box 9387, Santurce, Puerto Rico 00908. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St. Catherine St. West, Montreal 107, Que., Canada. Annual Meeting:

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, P. D. Thomson, 28 Portland Pl., London, W1N 4DE.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wotzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. José Marcos Pires de Oliveira, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

Sociedad Argentina de Radiología
Secretary-General, Dr. Alba de la Torre, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

Sociedad Colombiana de Radiología
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Luis Blum, Casilla 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiología
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. J. Corbaton, Clinica Infantil "La Paz," Av. Generalissimo, 117 Madrid 34, Spain. Annual Meeting:
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 730, Paris Cadet 15, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger.
Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting:

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK. 8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Norsk Forening for Medicinsk Radiologi
General Secretary, Gunnar Stake, Rikshospitalet, Oslo, Norway.

Scandinavian Radiological Society
Secretary-General, Professor C. F. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägn 5 C., Finland. Annual Meeting.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary-General, Dr. Pilar Gallar Barberá, Villanueva, 11, Madrid 1, España. Meets every other Saturday evening of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa i-Portugal.

Svensk Förening för Medicinsk Radiologi
Secretary, Dr. Hanz Ringertz, Röntgendiagnostika Avdelningen, Karolinska Sjukhuset 104 01 Stockholm 60, Sweden.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Bengal Radiological Association
Honorary Secretary, Dr. B. Chatterji, 262 Rash Behari Ave., Calcutta 700019, India. Meets 1st Fri. and 3rd Wed. of every month.

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Majid Rooholamini, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
Secretary, Dr. M. A. Lerner, Hesharon Hosp., Petah-Tigva, Israel.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, C. A. Beetlestone, M.B., B.S., D.M.R.D., Dept. Radiol., University College Hosp., Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. A. Visser, P. O. Box 8850, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

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ROENTGEN DIAGNOSIS

Neck and Chest

George, Ronald B., Herbert, Stephen J., Shames, Jay M., Ellithorpe, Dean B., Weill, Hans, and Ziskind, Morton M. Pneumothorax complicating pulmonary emphysema. *J.A.M.A.*, Oct., 1975, *234*, 389–393. (Address: Ronald B. George, M.D., Pulmonary Diseases Section, LSU School of Medicine, P.O. Box 3932, Shreveport, La. 71130.)

The importance of chest roentgenography in pneumothorax was emphasized in this paper which describes two groups of patients who developed this entity. In the younger patient who develops spontaneous pneumothorax without the complication of chronic obstructive lung disease, the usual cause is rupture of pleural blebs formed as the result of defects in the walls of subpleural alveoli. In the older patients, however, spontaneous pneumothorax is most often due to chronic bronchitis and emphysema and results from a rupture of intrapulmonary bullae through the intact visceral pleura. Treatment in the younger group, who recur more frequently, is more often associated with thoracotomy than in the older group, but it is usually much more recognizable and much better tolerated than in the older group with pre-existing chronic lung disease and other pre-existing serious conditions.

The difficulty arises because of the frequent lack of chest pain and increased breathlessness and anxiety out of proportion to the amount of pneumothorax present. Emphysema patients are frequently found to have breathlessness, anxiety, decreased breath sounds and hyperresonance, and a chest roentgenogram, therefore, becomes the only method to detect the presence of pneumothorax. It should be noted that the older age group is not as likely to develop air trapping or tracheal shift, because of the loss of elasticity and compliance of the chest and lungs in a degree of associated pneumothorax visible on the roentgenogram may be surprisingly small in relation to the symptom. There is more likely to be widening of the interspaces and diaphragmatic depression on the involved side than there is shift of the mediastinal structures.

Several cases and excellent roentgenograms are presented and the authors conclude that pneumothorax is frequently a benign complication if it is recognized and treated promptly.—*Robert P. Andrews, M.D.*

Ottinger, Leslie W. Ruptured arteriosclerotic aneurysms of the abdominal aorta: reducing mortality. *J.A.M.A.*, July, 1975, *233*, 147–150. (Address: Department of Surgery, Massachusetts General Hospital, Warren Building, 275 Charles Street, Boston, Mass. 02114.)

Despite improved techniques in management, over-all mortality from ruptured arteriosclerotic aneurysms of the aorta remains unacceptably high, and this study was carried out to identify preoperative factors which contribute to the poor results. One hundred patients were reviewed. Mortality was highest in the group who had a preoperative episode of systolic hypotension less than 80 mm of mercury, preoperative cardiac arrest, free intra-abdominal rupture, or preoperative anuria. Dr. Ottinger makes the important points that 85 percent of the aneurysms involved in this study were not diagnosed preoperatively, but that none were smaller than 7 cm in diameter, and that routine physical examination over the age of 40 should carefully search for abdominal aneurysms.

Since mortality doubles with the above described developments, and risk is usually the same if elective resection is done as soon as symptoms occur, early diagnosis is highly desirable, as well as early surgical intervention. The most important point made is that pain in the presence of an aneurysm or pulsatile abdominal mass must be considered diagnostic of a rupture. If resection is rapidly carried out with satisfactory volume replacement, avoidance of measures that acutely increase blood pressure (such as passage of a nasogastric tube which results in straining) and when possible use of an antigravity suit, especially if the patient has to be transferred from one hospital to another, mortality will be reduced. Since mortality under the conditions described is in excess of 50 percent and the mortality for elective resection is 10 percent and is near that level if surgical attack is carried out shortly after rupture, then early diagnosis remains the best preventive measure.—*Robert P. Andrews, M.D.*

Talia, Bartolo, and Palmieri, Beniamino. L'ecografia tiroidea nella diagnosi differenziale tra neoplasie benigne e maligne. (Thyroid echography in the differential diagnosis between benign and malignant neoplasms.) *Radiol. med.*, March, 1975, *61*, 246–258. (Address: Dr. Bartolo Talia, Istituto di Radiologia e Terapia Fisica, dell'Università di Modena, Modena, Italy.)

The indications for echotomography of the thyroid can be summarized as follows: (1) evaluation of size, shape and position of the gland; (2) differentiation between a solid or a cystic lymph node; (3) differential diagnosis between a benign or a malignant lesion.

Fifty patients with different types of thyroid disease were examined with the usual sonographic technique at low sensitivity and satisfactory results were obtained in the demonstration of a thyroid hyperplasia and of benign lesions. However, the

sonograms of patients with malignant thyroid tumors were not considered to provide a definite differential diagnosis.

Because one of the most important histopathologic characteristics of the malignant lesion is its tendency to infiltrate the adjacent parenchyma, the authors injected with 2 cc of sterile water the areas of the thyroid which appeared in the preliminary sonograms to be involved by an infiltrative process. No untoward reactions were observed in the 25 patients subjected to this procedure. In 10 cases the sonographic conclusions were of "probable benign lesion"; in 10 of "questionable malignant process." A third group of 5 patients affected by thyroid hypertrophy were considered as control.

The results obtained in the second group showed that the sterile solution moving in a centrifugal fashion between the infiltrated segments of parenchyma and between the necrotic or hemorrhagic elements, resulted in the production of stronger echoes which permitted a clear demonstration of the irregular, almost ragged, contours of the lesions. In benign lesions the sterile solution remained in the injected area giving rise to localized and well-defined echoes. In the third group the sonograms before and after injection of the sterile water showed no significant difference.

The authors feel that their procedure should clarify those sonograms of the thyroid in which a questionable malignant lesion is suspected.—*A. F. Govoni, M.D.*

Balius-Juli, R. Embolismo graso post-traumatico. (Post-traumatic fatty embolism.) *Anales de Medicina*, March, 1975, *61*, 147.

Following trauma (fractures) two types of fatty embolism can occur: (a) Pulmonary embolism; and (b) generalized embolism.

The author found, during autopsy studies, that 75 percent of fatal accident cases showed evidence of fatty pulmonary embolism. In 20 percent of these cases embolism was massive. It is noted that significant trauma to soft organs can also lead to embolism. A case of pulmonary emboli due to liver fragments in a case of ruptured liver is mentioned.

In cases of significant pulmonary embolism, hypoxia would be the most important consequence.

In cases of generalized fatty embolism, symptoms appear 36–72 hours after the traumatic episode and include: cerebral symptoms, hyperthermia, alteration of consciousness (coma or agitation), central tachycardia, and mild variable non-focal neurological signs; respiratory symptoms, related to hypoxia, with or without roentgen evidence of infarction or pulmonary edema; eyeground signs consisting of whitish spots around the retinal vessels; skin petechia, particularly in the upper chest, neck and axillae. Various tests used in diagnosis (fat cells in the urine, sputum etc.) are non-reliable, and diagnosis should be based on clinical evaluation.

Treatment should be mainly prophylactic: careful handling of fractures, prompt immobilization of fractures, avoiding continuous-traction treatments.—*Julian B. Alvarez, M.D.*

Serstock, Doris S., and Zinneman, Horace H. Pulmonary and articular sporotrichosis: report of two cases. *J.A.M.A.*, Sept., 1975, *233*, 1291–1293. (Address: Horace H. Zinneman, M.D., Veterans Administration Hospital, 54th Street and 48th Avenue S., Minneapolis, Minn. 55417.)

The authors report two cases of sporotrichosis which are rare; one, a case of primary sporotrichosis of the lung was treated successfully with Amphotericin B and penicillin. The second case was delineated in the knee of an elderly patient and improved with Amphotericin B systemically and in the joint space. The roentgen findings shown and in the first case demonstrate a right upper lobe infiltrate, and in the second case show moderate nonspecific degenerative changes in the knee joint.

In their conclusion, the authors discuss various modes of treatment including the methods used in these patients as well as high temperature baths and diathermy, which are occasionally effective.—*Robert P. Andrews, M.D.*

Abdomen

Pafaianni, F., Refi, C., Galeffi, J., Grani, M., and Dal Pozzo, M. La colecistite enfisematosa (descrizione di due casi). (Emphysematous cholecystisis: report of 2 cases.) *Ann. radiol. diag.*, 1975, *45*, 429–435. (From: Spedali Riuniti di S. Maria Sopra i Ponti Ospedale Generale Provinciale, Arezzo, Italy.)

Emphysematous cholecystisis is a rather rare inflammatory process involving the gallbladder and characterized by the intraluminal presence of gas in the viscus.

Of the several organisms which have been isolated and are thought to be responsible for this phenomenon, the most common are *E. coli*, *Clostridium welchii* and *Clostridium oedematiens*. However, other important causes are thought to be the reflux of gastric and pancreatic juices into the biliary system and anoxemia of the walls of the gallbladder.

The roentgen findings are well known and vary with the stage of the pathologic process. Usually a standard roentgenogram of the abdomen demonstrates either a homogeneous or a mottled gas shadow in the right upper quadrant, corresponding to the usual appearance of the gallbladder. In the ercet position a fluid level is noted.

From an analysis of the literature it appears that the age-group most frequently affected is between 40 and 50 years.

Until the present, about 60 cases have been reported and the authors added 2 more and observed two males aged 58 and 71, respectively.

(See also the paper on the same subject by L. Blum and A. Stagg in the April 1963 issue of the Journal.)—*A. F. Govoni, M.D.*

Mackenzie, M., Fordyce, J., and Young, D. G. Subphrenic abscess in children. *Brit. J. Surg.*, April, 1975, *62*, 305–308. (From: Royal Hospital for Sick Children, Yorkhill, Glasgow, Scotland.)

Subphrenic abscess remains a serious complication of intra-abdominal sepsis or surgery. Nineteen consecutive childhood cases of subphrenic abscess which were treated are analyzed. This complication of intra-abdominal suppuration following surgery still carries a grave prognosis. In this series four of the patients died (21 percent), however, two of the deaths were related to subphrenic abscess while the remaining two were not directly related.

Among the clinical signs and symptoms of presentation were pyrexia, leucocytosis, abdominal pain, abdominal swelling and tenderness and anemia. Roentgenographic evidences were elevation of the hemidiaphragm, pleural effusion and gas under the diaphragm. Fluoroscopy showed limitation of movement of the hemidiaphragm on the side of the abscess in every patient in which it was performed. However, complete immobility of the diaphragm is probably a late manifestation of the condition since movement was retained with both subhepatic abscesses and better than with suprahepatic abscesses, although better in the former than in the latter. The primary conditions preceding the development of subphrenic abscess were acute appendicitis in over half of the cases, primary peritonitis (21 percent) and post-surgical (10 percent).

Treatment consisted of antibiotic therapy and surgical intervention. The earlier the diagnosis the better the prognosis.—*Lionel W. Young, M.D.*

Skeletal System

Kozlowski, K., Robertson, F., and Middleton, R. Radiographic findings in Larsen's syndrome. *Australasian Radiol.*, Sept., 1974, *18*, 336–344. (From: The Departments of Radiology and Orthopaedics of the Royal Alexandra Hospital for Children, Sydney, Australia.)

Larsen's syndrome is characterized by multiple, usually bilateral joint dislocations, positional deformities of the hands and feet, finger abnormalities, scoliosis and peculiar facies. The authors report roentgenographic findings in four cases from Australia. They indicate that these cases were found among 14 cases filed as congenital dislocation of the knees. This is in accord with Silverman's analysis of the literature which uncovered 46 cases of Larsen's syndrome, although only 13 had been reported as such. Among annotations other than Larsen's syndrome have been arthrogryposis, multiple congenital anomalies, multiple joint dislocations or knee and hip dislocations. The abnormalities may show regression with age and under treatment, the diagnosis may be more difficult later in childhood and in adults.

The authors tabulate the roentgenographic data and also offer a roentgenographic differential diagnosis. In infancy such entities as arthrogryposis syndrome, chromosomal abnormalities, multiple congenital joint dislocations, multiple congenital unclassified anomalies and diastrophic dwarfism are considered. In childhood the differential diagnosis includes diastrophic dwarfism, spondyloepiphyseal dysplasia, multiple congenital unclassified anomalies and Perthes disease.—*Lionel W. Young, M.D.*

D'Ettorre, and Borghi, A. Possibilità limiti della diagnosi radiologica degli osteoblastomi benigni. (Possibilities and limitations in the radiographic diagnosis of benign osteoblastoma.) *Ann. radiol. diag.*, 1975, *45*, 319–328. (From: Istituto Ortopedico Rizzoli, Servizio di Radiologia, Bologna, Italy.)

The roentgenologic findings of benign osteoblastoma are not specific because the polymorphism of the lesion is related to its stage of evolution and its site of origin.

The clinical, pathological, and roentgenographic findings observed in 45 patients with proved osteoblastomas were reviewed in order to evaluate the role of roentgenology in the correct diagnosis of these lesions.

The lesions were subdivided into five groups in accordance with the different roentgenographic patterns: (1) area of osteolysis without perifocal sclerotic reaction, 5 cases; (2) area of osteolysis with perifocal process of sclerosis, 16 cases; (3) "ballooning" of the involved segment of bone with central calcification, 10 cases; (4) pseudocystic appearance, 8 cases; and (5) osteosclerotic and hyperostocic, 4 cases.

The patterns of Groups I, III, and IV are considered to represent the various early stages in the evolution of the tumor, while the bony changes of Groups II and V would instead represent the further progress of the lesion.

The only relative constant and characteristic roentgen pattern of osteoblastoma is noted in its localization in the vertebral apophysis. When the lesion arises from the epiphysis and metaphysis of a long bone the differential diagnosis should consider such lesions as benign chondroblastoma, aneurysmatic cyst, etc. In its location in the diaphyses of long bones and because of the presence of osteolytic

and ostoblastic changes, the differential diagnosis is made more difficult and one should consider among others such pathologic processes as Brodie's abscess, nonostenogenic fibroma and osteoid osteomas.—*A. F. Govoni, M.D.*

Blanquet-Benedicto, A., Orduno-Serra, M., and Ley-Gracia, A. Lipocalcinosis tumoral. (Tumoral lipocalcinosis.) *Anales de Medicina*, May, 1975, *61*, 334.

This entity is also known as lipo-granulomatosis. It is a benign process of unknown origin, which usually starts in adolescence.

It is manifested by the presence of a densely calcified nodular mass adjacent to large joints, such as hips shoulders, or elbows. Histologically it is made up of osteoblasts, osteoclasts, fibrous tissue, giant cells, and xanthomatous cells. Clinically it presents as a painless, hard, para-articular mass, and any symptoms are due to compression of nerves, etc. Treatment is surgical.

This article is a case report of a 51 year old male. He had two episodes of pain, within the last three years, in the left gluteal area, radiating to the thigh and leg. A large mass, which the patient stated he had since his youth, was palpable in the left trochanteric area: it had never produced local symptoms. Roentgen studies of the lumbosacral spine, including myelography were negative. Roentgenograms of the left hip showed a large cauliflower-like calcified mass adjacent to the greater trochanter. At surgery it was noted that this mass was stretching the sciatic nerve, and this explained the symptoms. The mass was completely excised and the symptoms disappeared.—*Julian B. Alvarez, M.D.*

RADIOISOTOPES

Siegel, Michael E., Walker, William J., Jr., and Campbell, John L., II. Accumulation of ^{99m}Tc-diphosphonate in malignant pleural effusions: detection and verification. *J. Nuclear Med.*, Oct., 1975, *16*, 883–885. (Address: Michael E. Siegel, Division of Nuclear Medicine, Malcolm Grow USAF Medical Center, Andrews Air Force Base, Washington, D.C. 20331.)

^{99m}Tc-diphosphonate and closely related compounds have been reported to accumulate in extraosseous tissues such as astrocytomas, lymphomas, neurofibromas and breast carcinomas. Two patients with malignant pleural effusions had bone scans and an increased activity was noted on the side having the pleural effusion. There was a small posterior pleural effusion in the patient with a bronchogenic carcinoma, and a moderate pleural effusion in the postmastectomy patient.

Qualitative and quantitative studies were done on the pleural effusion in the breast case in addition to pre and post-thoracentesis scanning. The ^{99m}Tc-diphosphonate was in the noncellular fluid component of the pleural effusion.

The above study suggests a specificity of the ^{99m}Tc-diphosphonate for malignant pleural effusions.—*Charles W. Cooley, M.D.*

MISCELLANEOUS

Charkes, N. David, Malmud, Leon S., Caswell, Taylor, Goldman, Leonard, Hall, John, Lauby, Vincent, Lightfoot, William, Maier, Willis, and Rosemond, George. Preoperative bone scans: use in women with early breast cancer. *J.A.M.A.*, Aug., 1975, *233*, 516–518. (Address: N. David Charkes, M.D., Section of Nuclear Medicine, Temple University Hospital, 3401 N. Broad Street, Philadelphia, Penn. 19140.)

A group of women suspected of having breast cancer had preoperative Sr-87m bone scans. No patient had signs or symptoms suggestive of bone metastases or other distant spread. Thirty-five of the patients were subsequently found to have breast cancer. Thirteen of the patients had abnormal scans and in ten the increased uptake could be explained by nonmalignant causes such as arthritis, kyphoscoliosis, disk disease, etc. Three of the 13 patients (9 percent) had bone metastases, as shown by bone scanning but not roentgenographically.

Twenty-two of the 35 patients had axillary lymph node metastases. Bone metastases were suspected by scans in 14 percent (3 of 22) of patients with regional lymph node metastases and none in the 13 with no regional lymph node metastases.

New bone metastases developed in 8 patients during the follow-up period of 20 months, all in the group with axillary lymph node involvement.

The bone scan is not specific for bone metastases. Bone scanning can detect bone metastases earlier than can be shown roentgenographically.

Preoperative bone scanning is of value in detecting unsuspected metastases and if metastases are present radical surgery would be contraindicated.—*Charles W. Cooley, M.D.*

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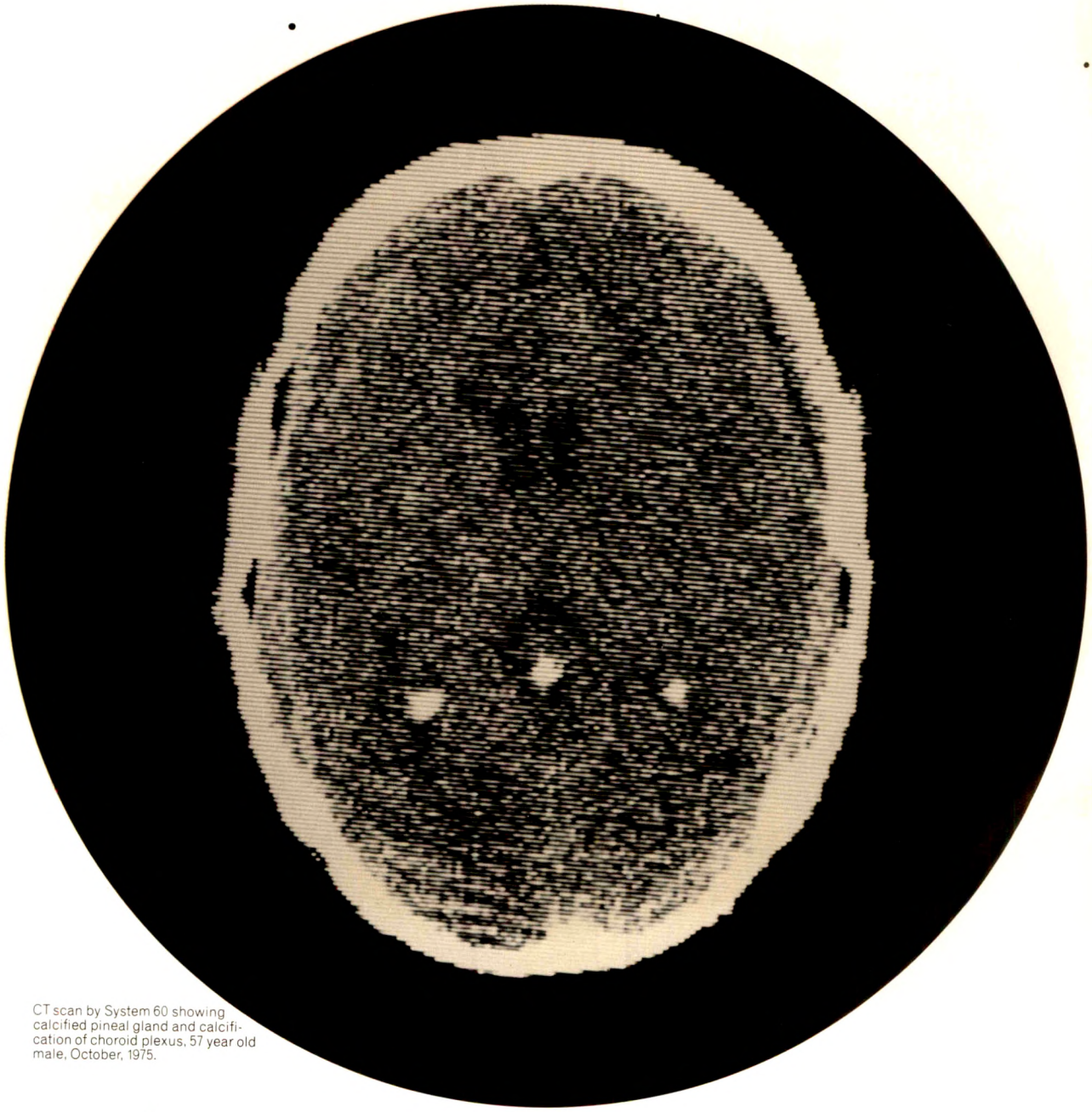

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Du Pont announces Cronex® Quanta II blue-emitting intensifying screens that enable radiologists to increase the relative speed of imaging systems while still achieving excellent diagnostic clarity. These new screens are based on barium fluorochloride phosphor technology developed by Du Pont research.

Maximum Convenience for Imaging Requirements

Quanta II screens are fully compatible with the Du Pont family of quality films as well as all other conventional films. There is no need to purchase special films to realize increased speed.

Because of this compatibility, you can now have a wide selection of system speeds and be assured of maximum convenience and flexibility in meeting your imaging requirements. Here are just a few of the film/screen combinations now available:

Imaging Requirement	Film/Screen	Results
High Speed	Cronex® 4/Quanta II	Twice the speed of Cronex® 4/Hi-Plus. Excellent diagnostic clarity.
Improved Diagnostic Clarity at Conventional Speed	Cronex® 7/Quanta II	Low Haze. Low noise. Clarity like Cronex® 2DC but with 90-second processing. Speed equal to Cronex® 4/Hi-Plus.
High Speed with Excellent Visualization of Bone Detail and Soft Tissue	Cronex® 6 Plus/Quanta II	Superb low-density contrast. Extended recording range. Twice the speed of Cronex® 4/Hi-Plus.

can increase system speeds diagnostic clarity.

Excellent Diagnostic Clarity

Quanta II screens provide diagnostic clarity similar to Cronex® Hi-Plus. The increased speed means there is less chance of blurred images caused by patient motion. In addition, since smaller focal spots can be used, Quanta II is ideal for specialized procedures such as magnification angiography.

Lower Operating Costs

Quanta II screens are reasonably priced and can help reduce operating costs in Radiology Departments. Their increased speed means less tube load so that X-ray equipment will last longer. You can even consider lower-powered, less costly generators. And, because Quanta II screens are blue-emitting, no special safelights are required.

To learn more about these new high-speed, blue-emitting screens, ask your Du Pont Technical Representative for a free demonstration of Quanta II—today's screen for tomorrow's needs.

Or, write Du Pont Company, Room 24784, Wilmington, Delaware 19898.

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MEDICAL X-RAY

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UTY REPLACEABLE BEARINGS IN TRANSPORT SYSTEM **40.** WATE
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2. VARIABLE SPEED ADAPTABILITY **43.** STAINLESS STEEL RACKS
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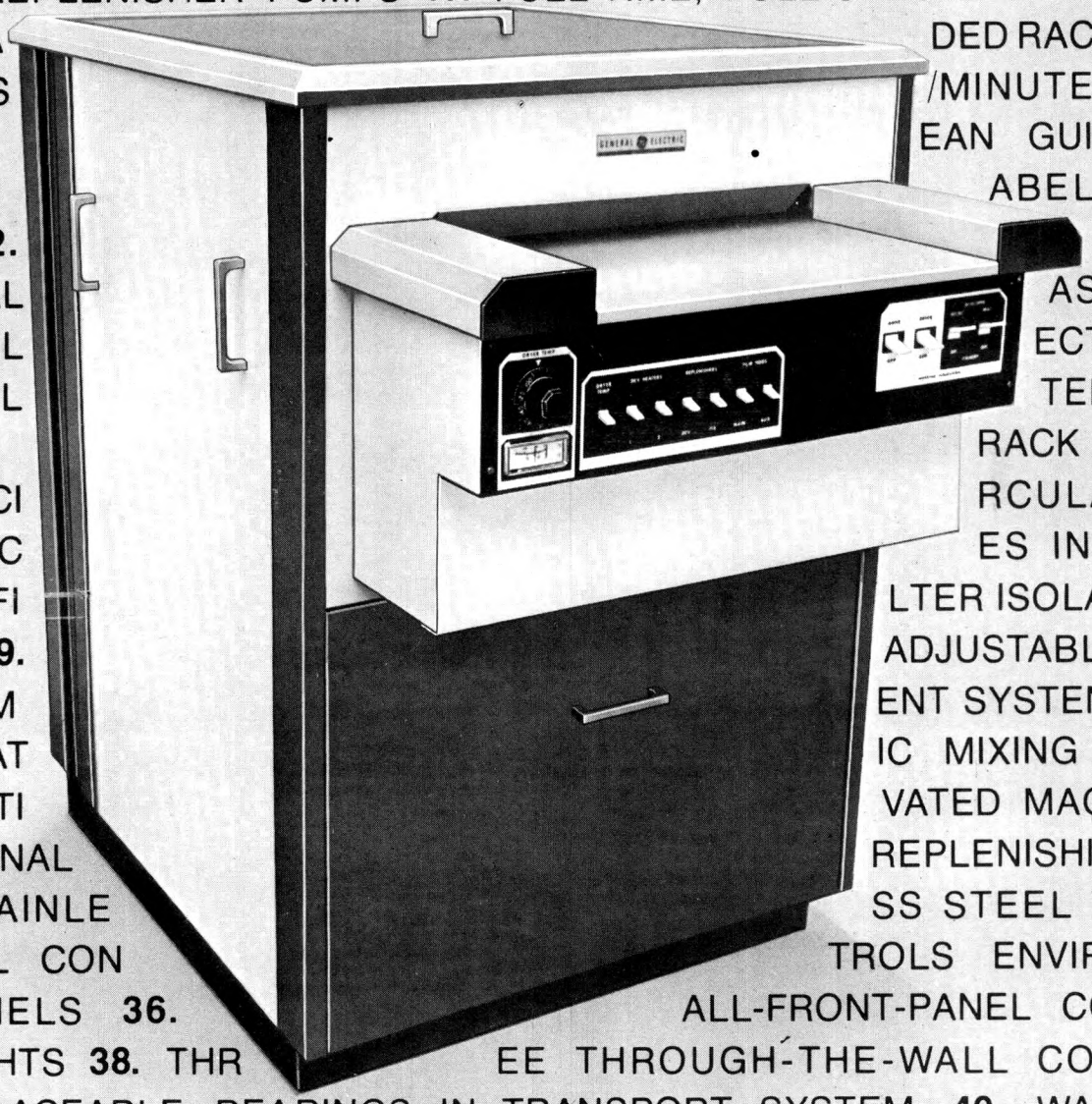
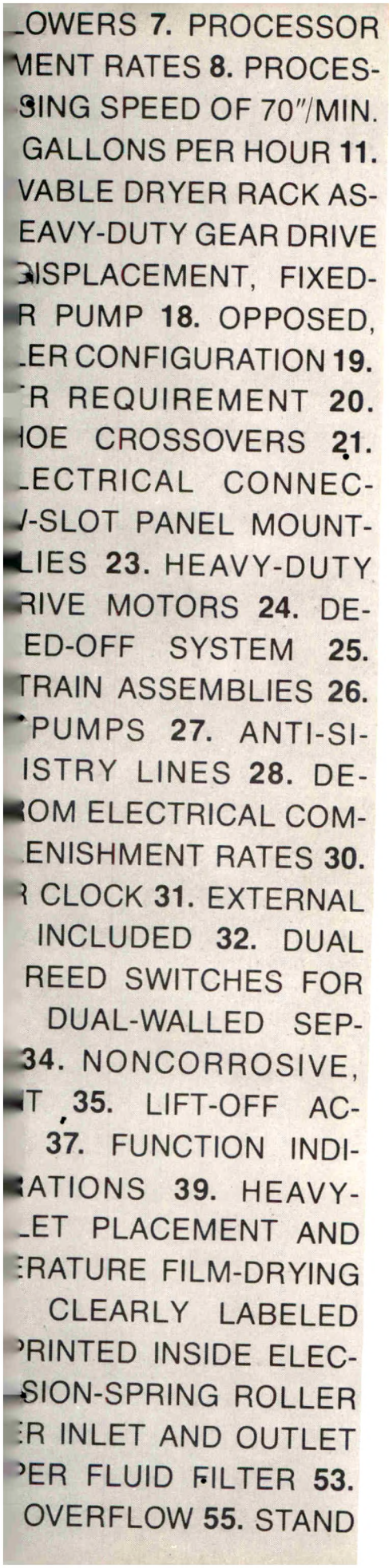
TRONICS COMPARTMENT **47.** VACUUM BREAKERS ON MIXING VALVE **48.**
COMPRESSORS **49.** CALIBRATED DRYER TEMPERATURE CONTROL **50.** P
PLACEMENT **51.** FILM-FEED AUDIBLE ALERT **52.** CARTRIDGE-TYPE DE
AUTOMATIC FILM-FEED TIME-DELAY DEVICE **54.** SEPARATE SILVER RECO
DIRECTS WITH BUILT-IN OVER LOW **56.** PARTS AND LABOR WARRANTY





In a world of look alikes, the GE 400 film processor stands alone.

Only the full size GE 400 fully automatic film processor is a second-effort performer. Dual systems (2 developer heaters, 2 thermostats, 2 dryer heaters and fans) offer back-up performance which maintains film processing capability and minimizes downtime due to component failures.

Proven performance factor: 99.14%

A GE 400 processor, on duty 24 hours a day for a full year in the emergency room of a large metropolitan hospital, logged over 8,760 operational hours with only 14 hours total downtime. That's 99.14% around-the-clock performance.

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The GE processor senses both film length and width, to help ensure consistent film quality, and a savings in chemistry. Two other significant economies: water savings as much as 75 gallons per hour in the stand-by mode; a price tag of as much as $3000 under some competitors!

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General Electric Medical System Milwaukee, Toronto, Madrid

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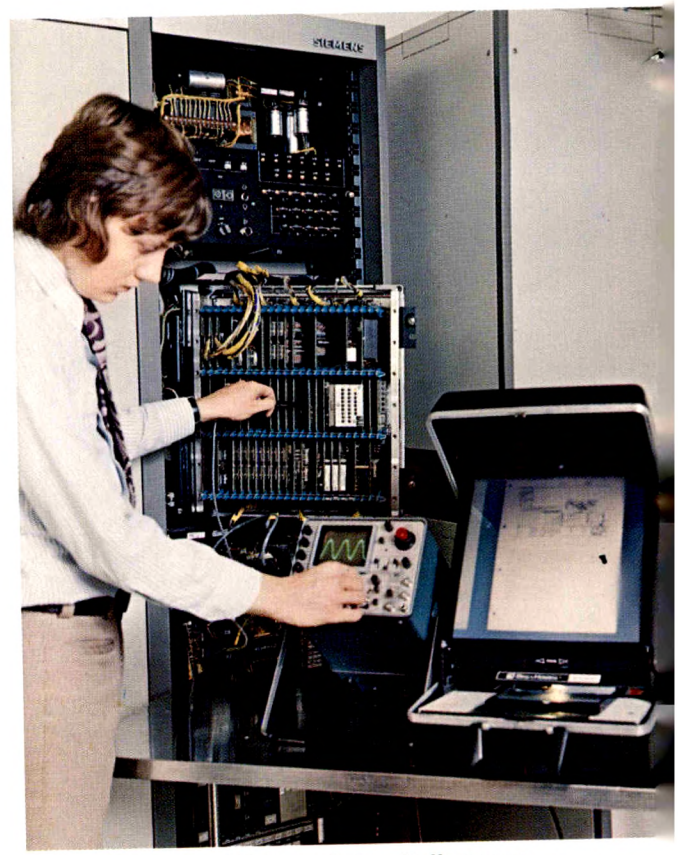
In addition, Siemens service technicians carry complete Microfilm Case Histories of every X-ray system they service. These case histories include circuit diagrams, installation, modification and service records. And they are portable, eliminating the need to check office files for vital information.

Optimatic Generators with designed-in serviceability. Three new service innovations from Siemens. Write for detailed literature.



A Microfilm Case History of circuit diagrams for a Siemens X-ray unit.

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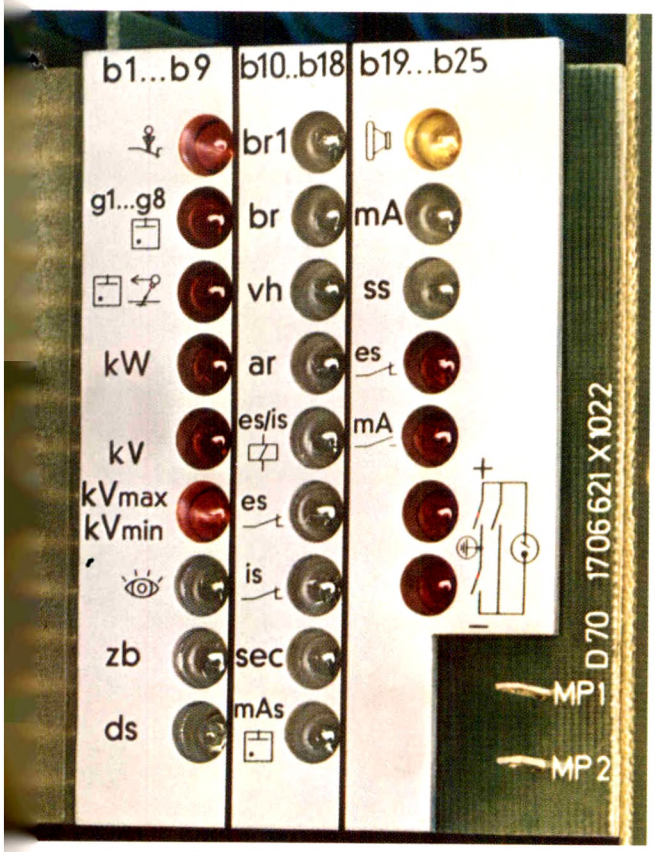
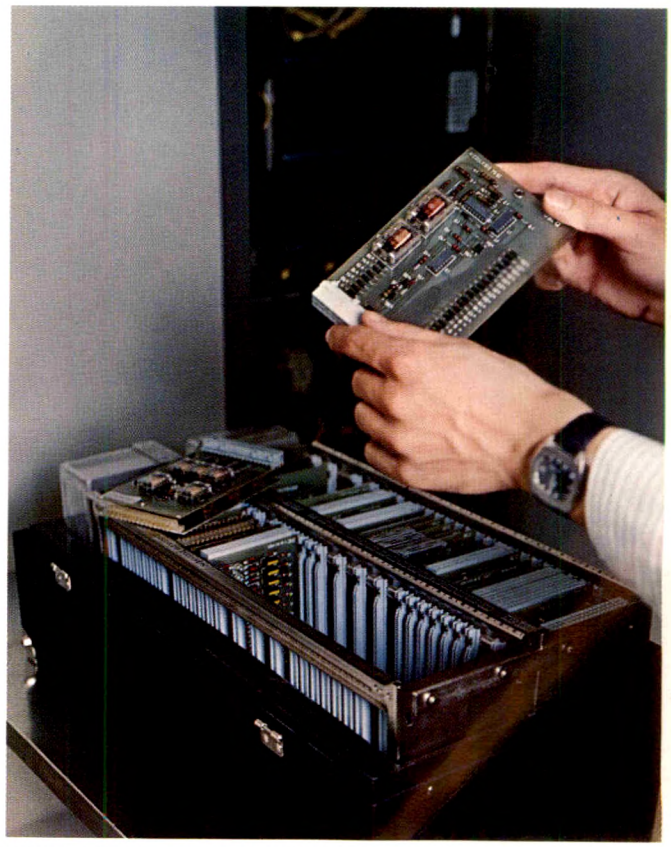
Medical Systems Division
186 Wood Avenue South, Iselin, New Jersey 08830 (201) 494-1000.
In Canada: Siemens Canada Limited, P.O. Box 7300, Pointe Claire 730-P.Q.



omatic Fault Finder built into the
ral generator control cabinet.



A typical replacement circuit board in the Siemens Service Support Kit.

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These are convincing reasons to "buy Hunt" in a time when department heads and hospital administrators are concerned with reducing the rising cost of film processing while maintaining quality results.

You get more from Hunt than a "money saving package." Hunt chemical systems have a "packed formula" that will help you achieve consistent, optimum results with major domestic and imported 90-second films.

For more details contact a Hunt representative at one of the many Hunt branches and sales offices throughout North America. He has the facts and the experience to offer technical assistance as your department may require.

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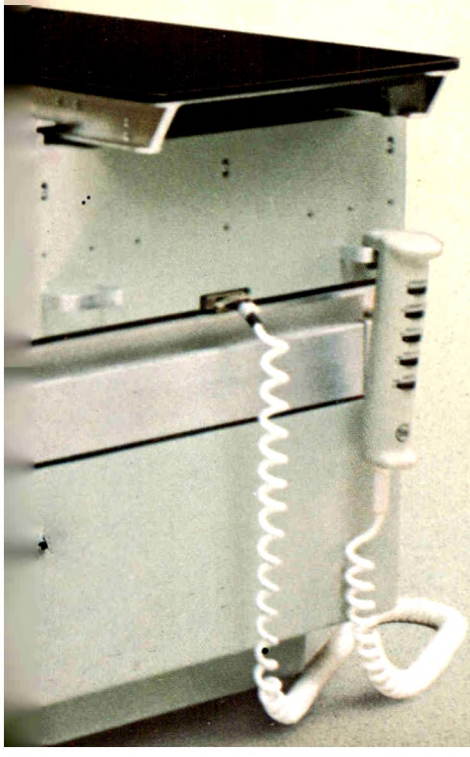

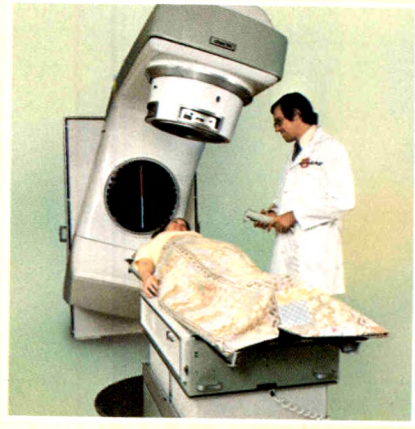

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ARTHROGRAPHY OF THE SHOULDER: The Diagnosis and Management of the Lesions Visualized *by* **Julius S. Neviaser,** *George Washington Univ. Medical School, Washington, D.C.* Writing to encourage the use of arthrography, the author passes on lessons learned in performing over 3,000 arthrographies and the knowledge of the anatomy of the shoulder gained at postmortem examinations and in the operating room. Topics covered include anatomy, techniques of arthrography, adhesive capsulitis, ruptures of the rotator cuff, lesions of the biceps tendon, dislocations of the shoulder, fractures of the surgical neck of the humerus, and interesting cases. A bibliography is also featured. *'75, 288 pp., 265 il., $22.50*

LYMPHOPROLIFERATIVE DISEASES *compiled and edited by* **David W. Molander,** *Memorial Hospital for Cancer and Allied Diseases, New York. (23 Contributors)* A compilation of the known facets of lymphosarcoma and related diseases is presented in this volume along with pathologic anatomy, immune mechanisms, clinical diagnosis and treatment employing irradiation and chemotherapy. The use of isotopes in managing this disease as well as in the diagnosis of occult foci of the disease is also covered. The new terminology, non-Hodgkin's malignant lymphoma, is used and new pathologic designations are included. *'75, 592 pp. (6 3/4 x 9 3/4), 256 il. (9 in color), 45 tables, $39.50*

THE BIOLOGICAL AND CLINICAL BASIS OF RADIOSENSITIVITY *edited by* **Milton Friedman,** *Instituto Regina Elena, Rome, Italy. (35 Contributors)* To reduce the gap between laboratory and clinical schools, authorities from the fields of radiobiology, radiation pathology, preclinical radiobiology and experimental clinical radiotherapy are asked to relate their investigations to the common denominators of radiosensitivity and chemosensitivity. Topics discussed include changes produced by irradiation at the molecular level, recovery from irradiation, factors such as DNA strand breaks which can influence radiosensitivity, and clinical concepts of radiosensitivity such as tumor lethal dose. *'74, 592 pp. (7 x 10), 373 il., 67 tables, $49.50*

CORONARY ANGIOGRAPHY (2nd Ptg.) *by* **Harold A. Baltaxe,** *Cornell Medical College, New York;* **Kurt Amplatz,** *Univ. of Minnesota Medical School, Minneapolis; and* **David C. Levin,** *Cornell Medical College, New York.* Gross and radiographic anatomy and the diverse pathologic processes of the coronary artery tree are described in this book. A section describing equipment and different techniques of catheterization is included. Also included are chapters on complications, their order of frequency and postoperative complications due to new surgical procedures. *'75, 256 pp., 173 il., $22.50*

HEALTH PROTECTION OF RADIATION WORKERS *by* **W. Daggett Norwood,** *Hanford Environmental Health Foundation, Richland, Washington.* The present knowledge regarding the hazards to the safety and well-being of persons working with radiation is summarized in this book. Discussed are established standards for health, safety and environmental protection along with their implementation. Even though it is addressed primarily to physicians and nurses, teachers, health physicists, occupational hygienists, safety specialists, biologists and others who are concerned with protecting the health and safety of radiation workers, this book will also contribute to the education of the public regarding the hazards of radiation. *'75, 468 pp., 16 il., 16 tables, $27.50*

THE FUNDAMENTALS OF RADIOLOGICAL SCIENCE *by* **John Hale,** *Hospital of the Univ. of Pennsylvania, Philadelphia.* Training programs in radiology have recently been restructured in that the subject matter is now being divided into the two areas of basic radiological science and clinical applications. This text is devoted to the first area of basic radiology and the fundamentals of mathematics and physics common to all specialities. Chapters on such subjects as radiation-producing machines or interaction of radiation with matter are offered. With the rapidly evolving technology in clinical application, students should have an understanding of the basic principles of radiology found in this text. *'74, 356 pp., 172 il., 3 tables, $14.75*

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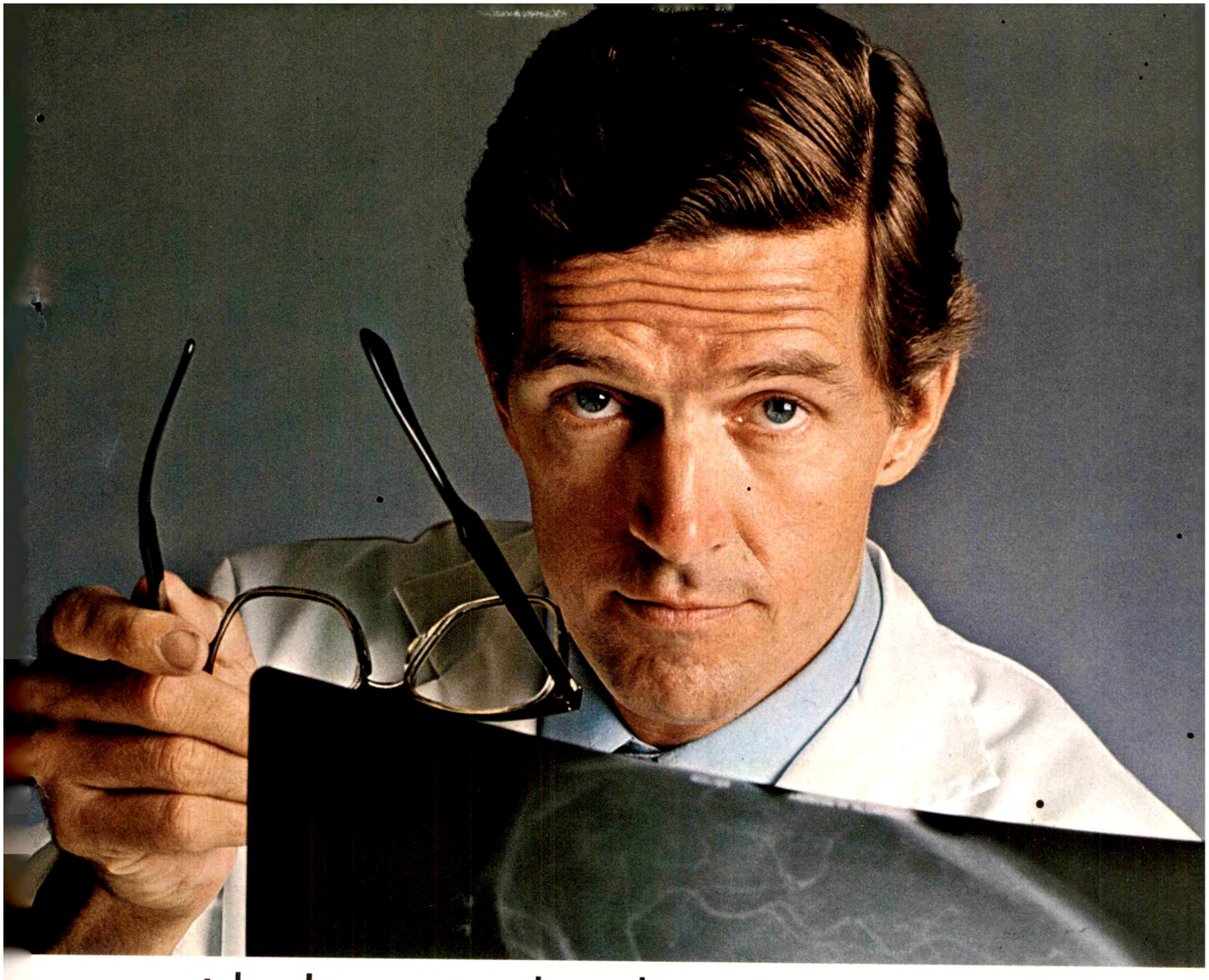

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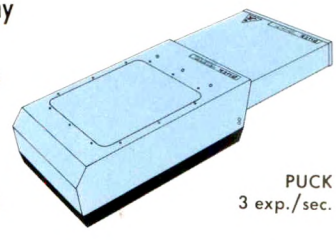

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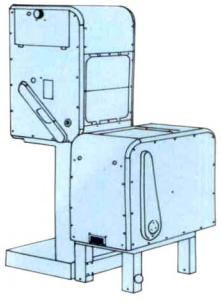

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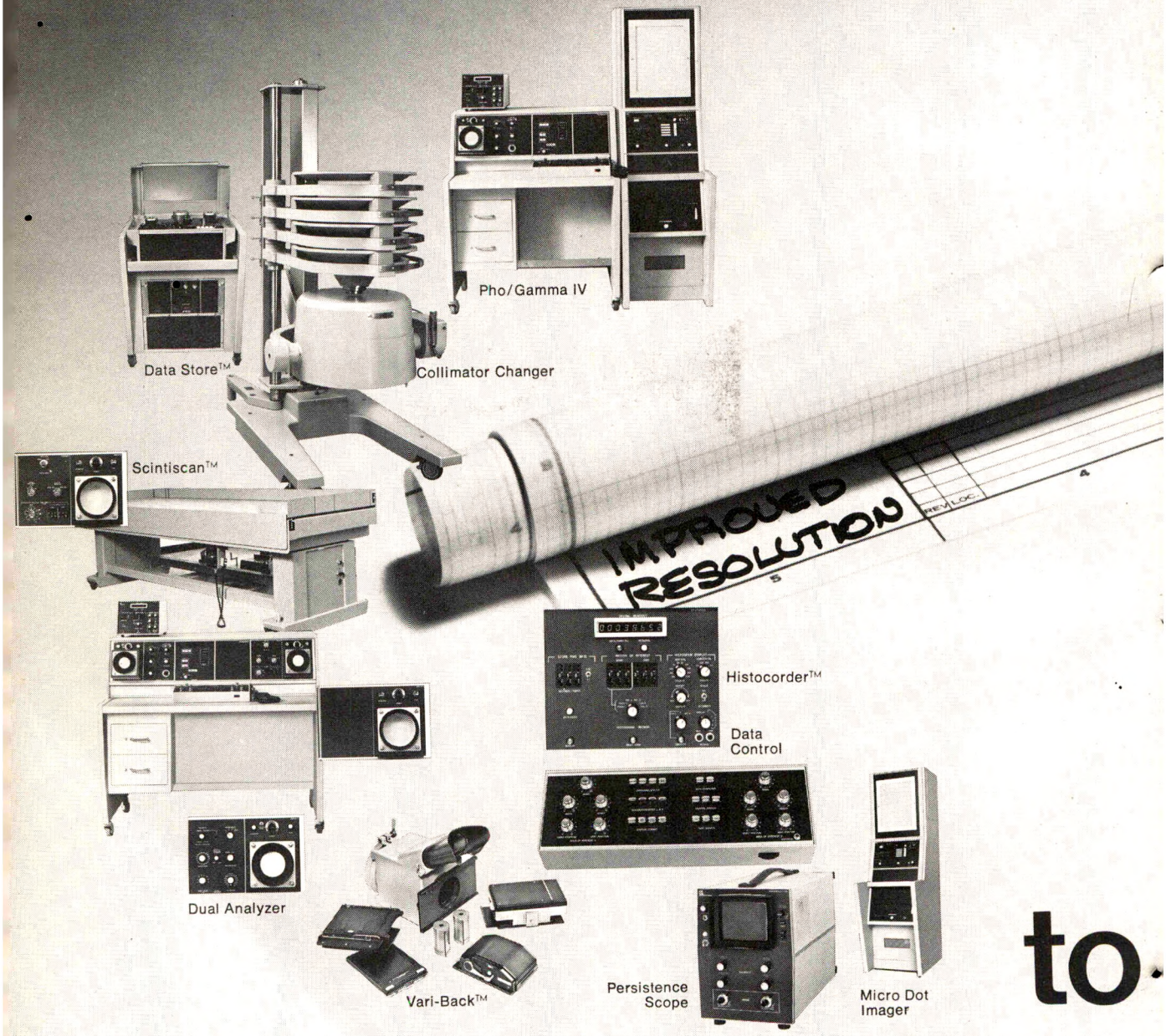

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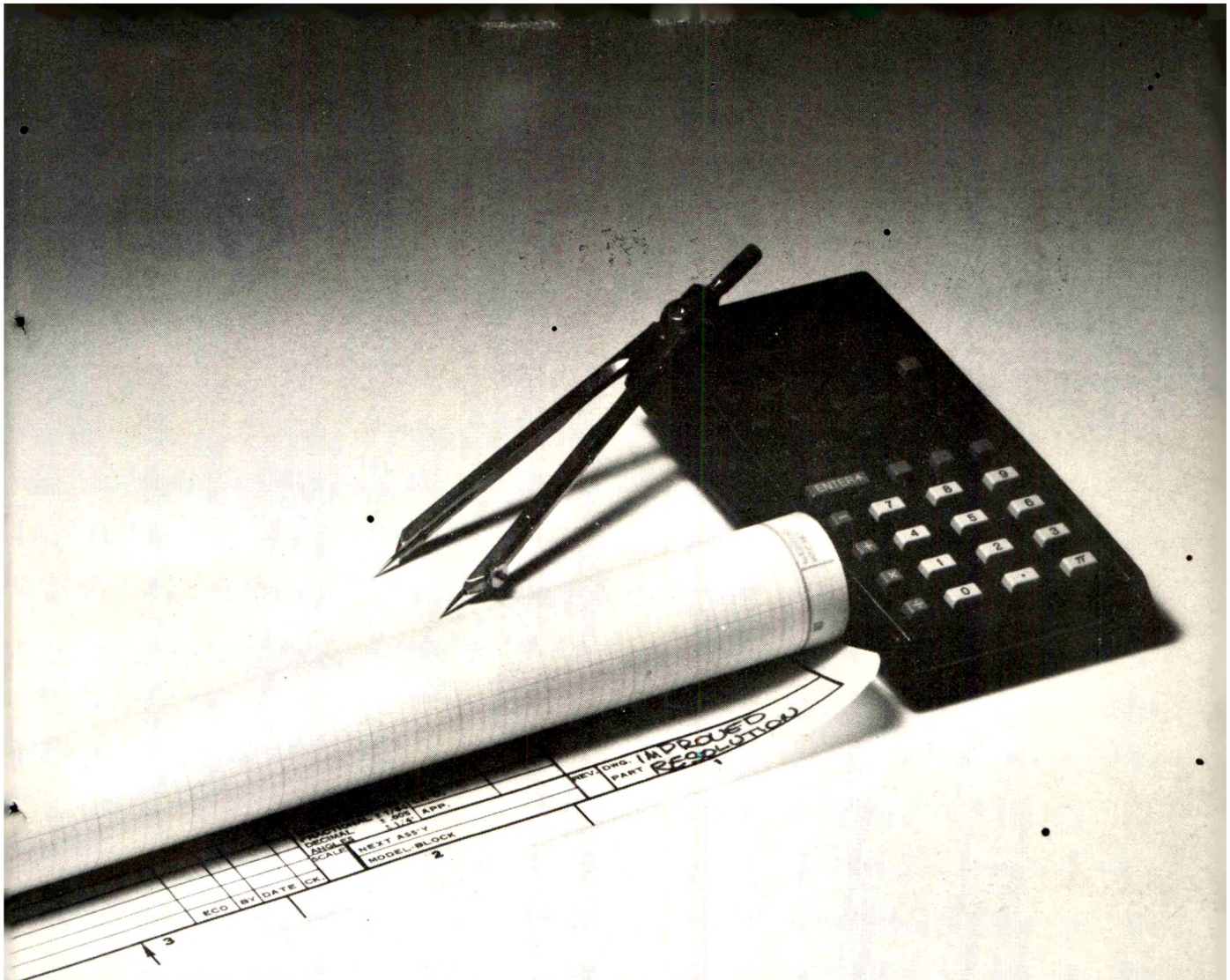

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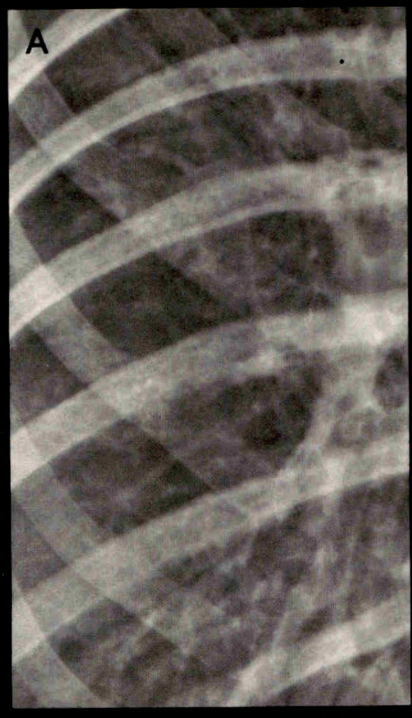
The Lungs in Hodgkin's Disease

As a rule, when the lungs are involved in Hodgkin's disease, so are the mediastinal lymph nodes. Of the following examples, all but Case I followed the rule.



CASE I: When first examined, this twenty-year-old female had fever and bilateral cervical adenopathy. At this time, the roentgen study of her chest revealed no abnormalities. Three months later, however, both lungs revealed changes without mediastinal or hilar adenopathy.

FIG. A:
A segment of the right hemithorax taken from this patient's original chest examination reveals no abnormalities.

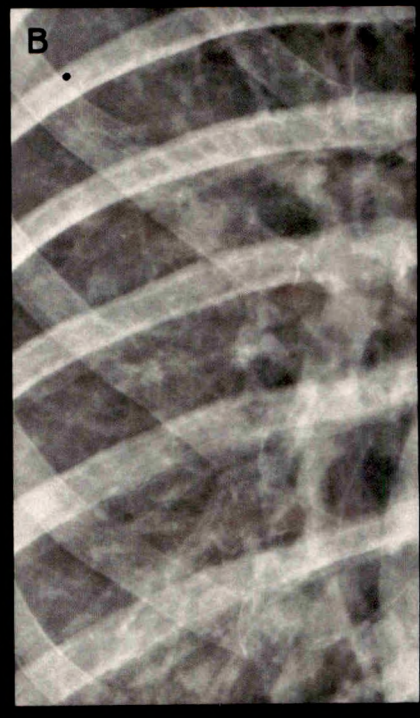
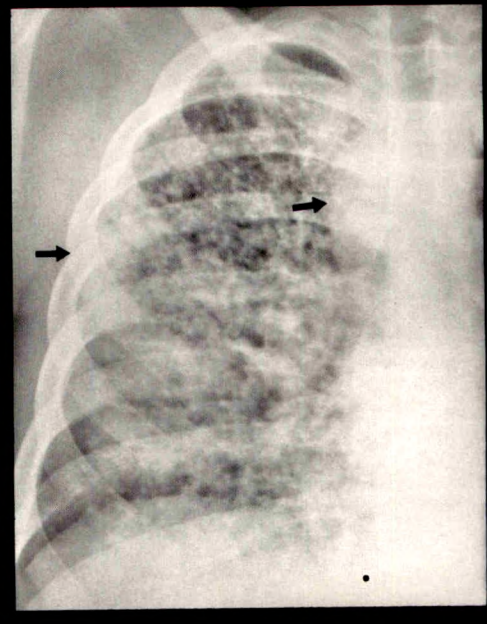


FIG. B:
The same segment of the right hemithorax (as demonstrated in Fig. A) three months later. Interstitial and vascular changes are dramatic.

CASE II: A nineteen-year-old male with chills, fever, inguinal adenopathy and weight loss.

It is not difficult to imagine that the infiltrates seen in Case I might have eventually developed a similar degree of parenchymal, mediastinal and pleural involvement (arrows).



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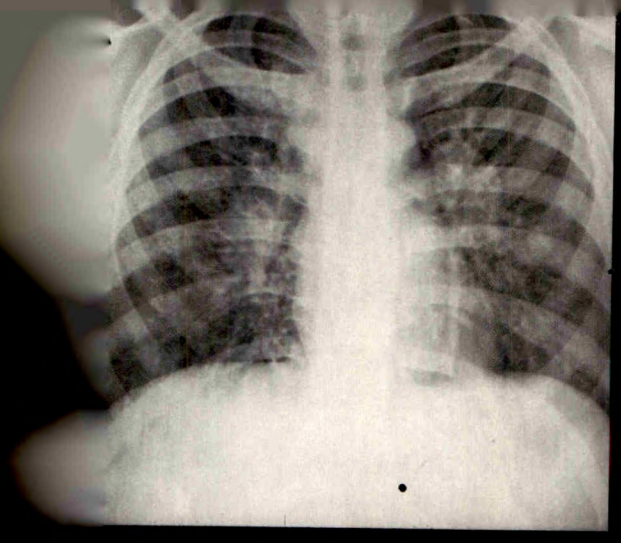
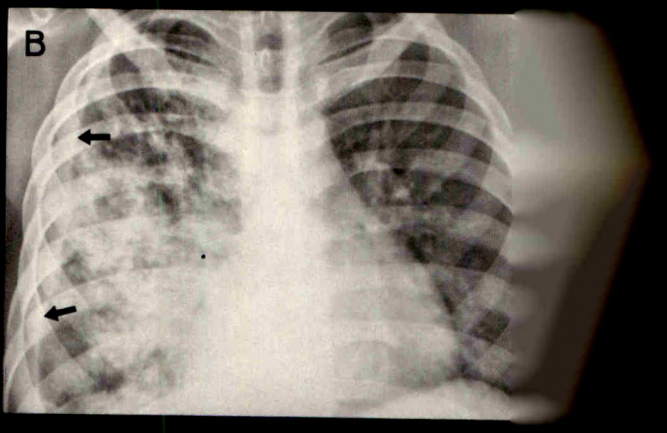
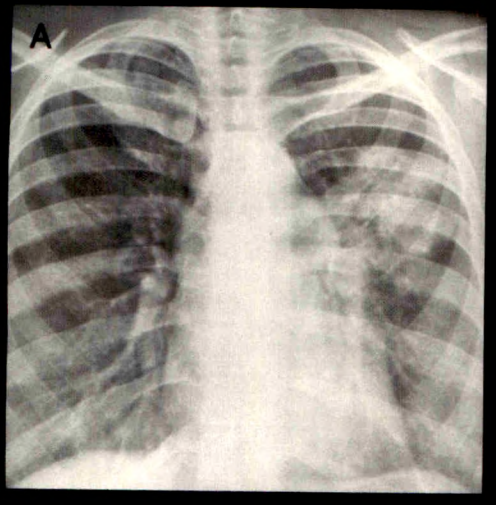
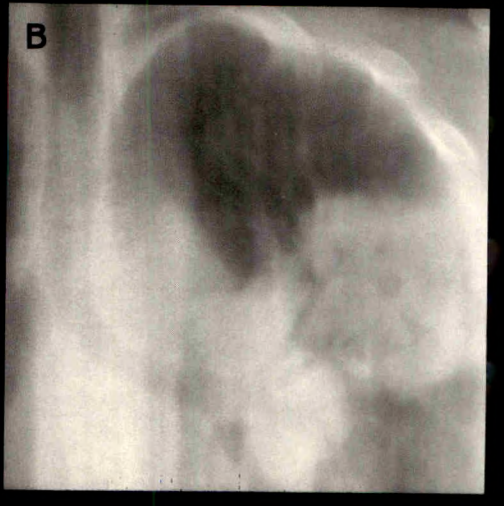


CASE III: A thirteen-year-old boy with fever and wet
ness. The original chest radiograph, Fig. A, revea
mediastinal adenopathy, hilar adenopathy, plus ear
bilateral parenchymal invasion.

Six months later, Fig. B, following radiation therap
and chemotherapy, the right hemithorax reveals p
gressive disease with pleural involvement (arrow
The left hemithorax seems somewhat improved.







SE IV: A twenty-four-year-old female who suddenly
eloped chills, fever, and rusty sputum. Whereas usu-
a lung mass associated with hilar and mediastinal
nopathy of the type seen in Fig. A suggest broncho-
ic carcinoma, this patient's age and history favored
ore acute process. The shaggy and irregular superior
gin of the mass, the bronchial distortion and small
ties demonstrated by body section radiography Fig. B,
noteworthy. This proved to be Hodgkin's disease.

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CONTRAINDICATIONS: At present, there are no known contraindications to the use of ^{99m}Tc-stannous pyrophosphate complex.

WARNINGS: The contents of the Phosphotec (Technetium 99m-Stannous Pyrophosphate Kit) vial are intended only for use in the preparation of ^{99m}Tc-stannous pyrophosphate complex and **are NOT to be directly injected into a patient prior to labeling.**

Phosphotec (Technetium 99m-Stannous Pyrophosphate Kit) is not radioactive. However, after ^{99m}Tc-sodium pertechnetate is added, adequate shielding of the resulting preparation must be maintained.

Radiopharmaceuticals should be used only by physicians who are qualified by specific training in the safe use and safe handling of radionuclides, produced by nuclear reactor of cyclotron, and whose experience and training have been approved by the appropriate federal or state agency authorized to license the use of radionuclides.

This radiopharmaceutical should not be administered to patients who are pregnant or during lactation unless the information to be gained outweighs the possible potential risks from the radiation exposure involved.

Ideally, examinations using radiopharmaceuticals, especially those elective in nature, of a woman of childbearing capability, should be performed during the first few (approximately 10) days following the onset of menses.

PRECAUTIONS: It is essential that the user follow the directions carefully and adhere to strict aseptic procedures during preparation of the product.

As in the use of any other radioactive material, care should be taken to insure minimum radiation exposure to the patient consistent with proper patient management, and to insure minimum radiation exposure to occupational workers.

To minimize visualization of the bladder, the patient should be encouraged to void immediately prior to the examination; prior hydration of the patient may be useful.

Use the preparation within 12 hours after labeling with ^{99m}Tc.

ADVERSE REACTIONS: At present, adverse reactions have not been reported following the administration of ^{99m}Tc-stannous pyrophosphate complex.

HOW SUPPLIED: Phosphotec (Technetium 99m-Stannous Pyrophosphate Kit) is supplied in a kit containing five vials.

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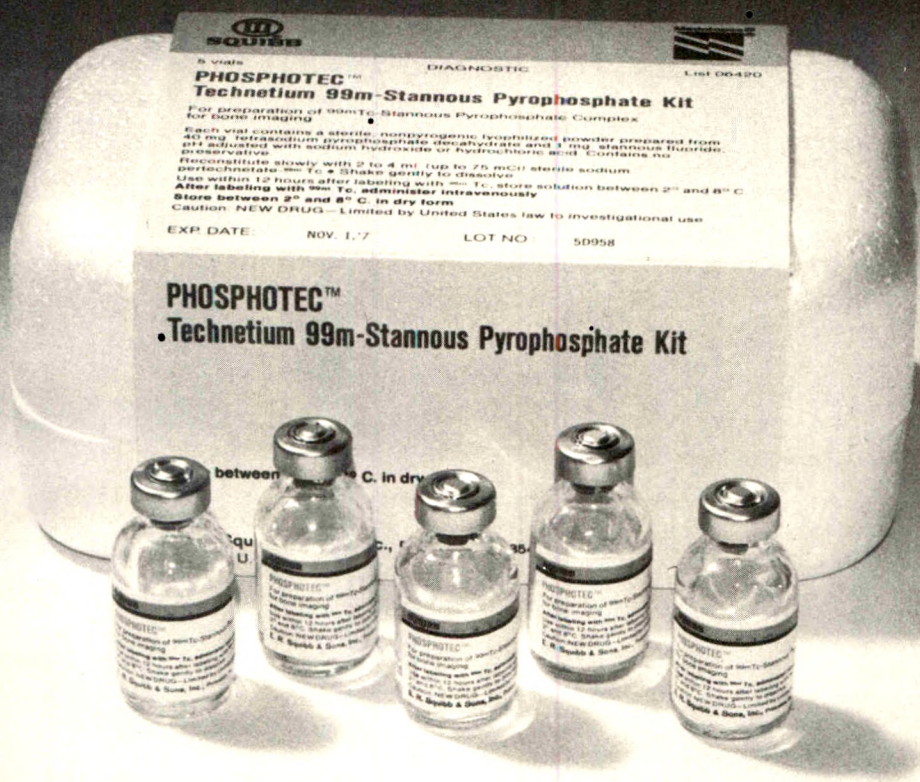

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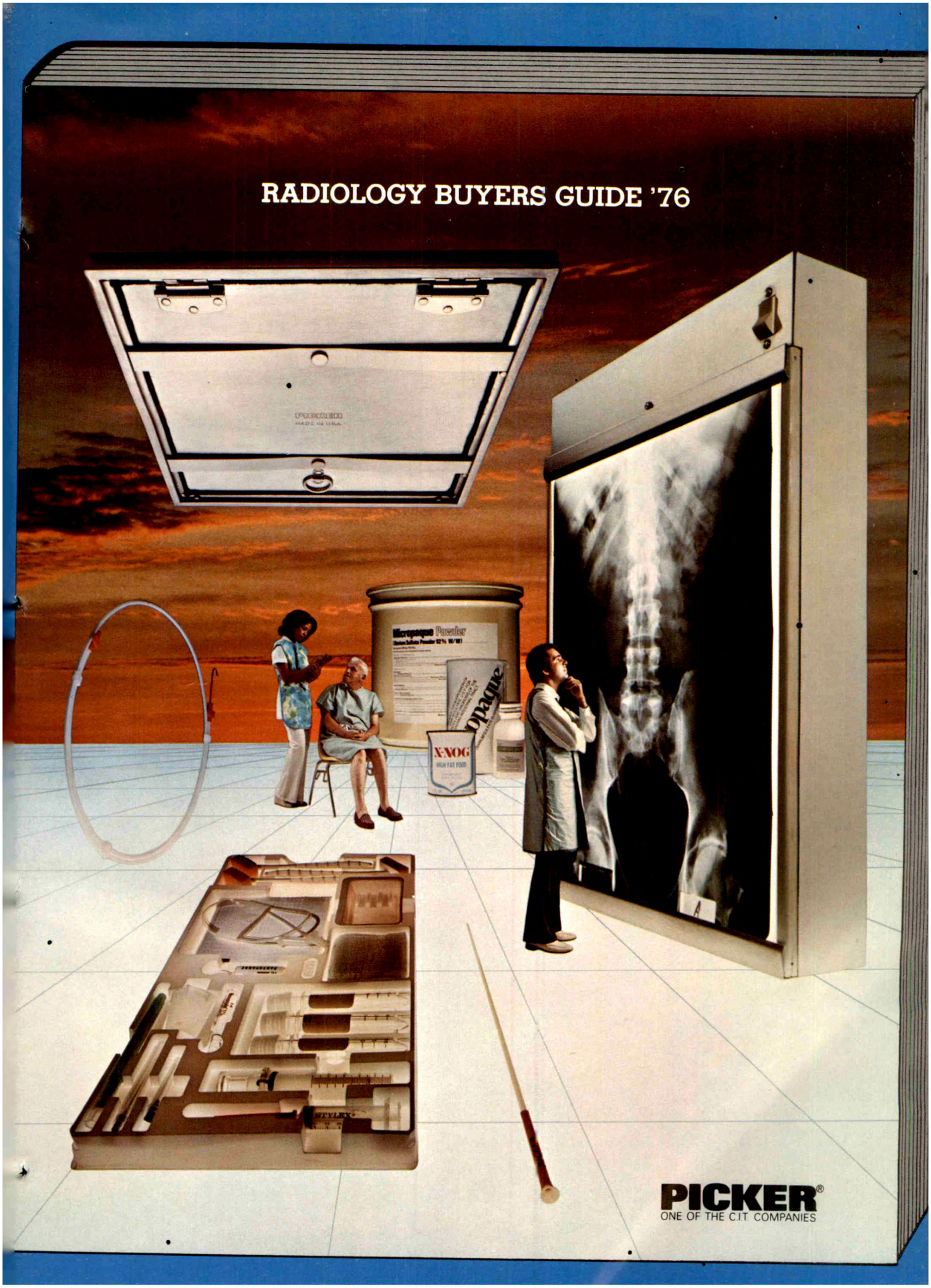

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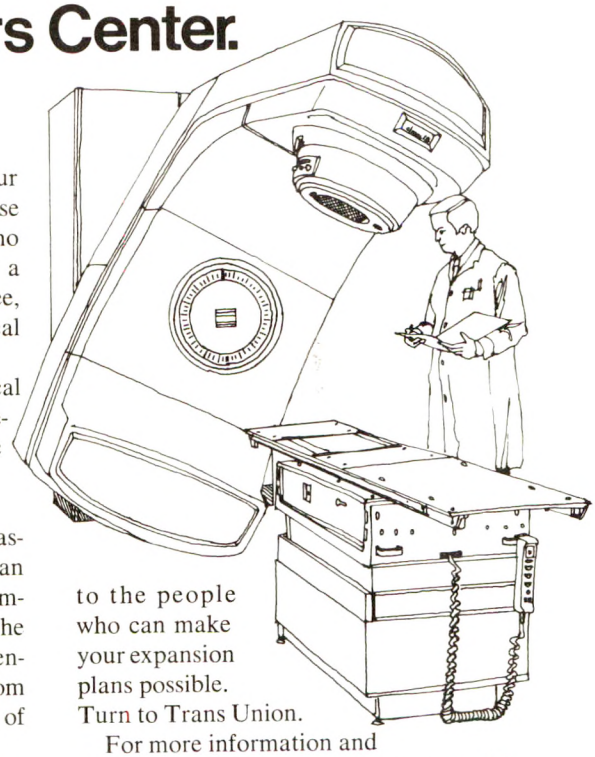

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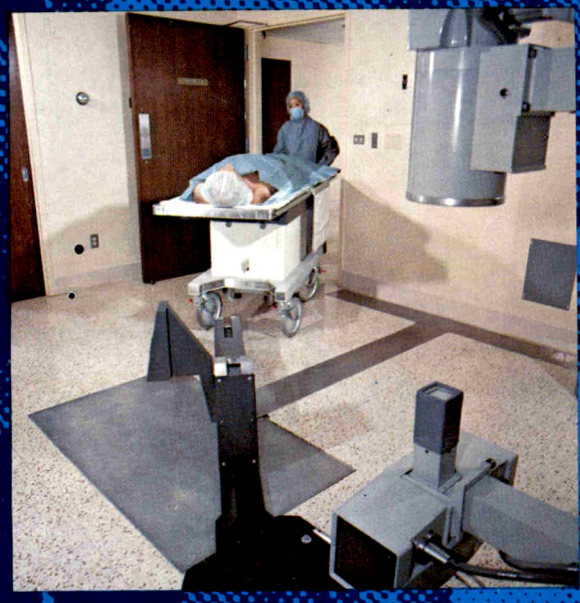

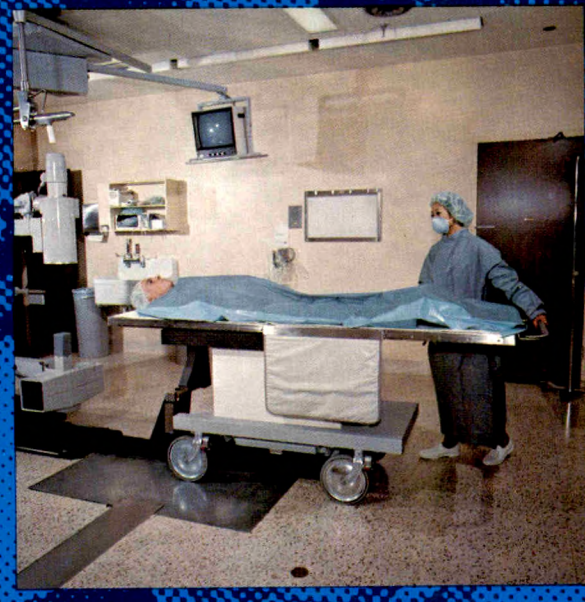

In certain cases, the cranio-caudal (or half-axial) projection improves visualization of cardiac anatomy.

For example: the cranio-caudal view of the left main coronary artery *clearly* separates the proximal divisional branches. In other areas, too, you're better able to define the degree of occlusion... or obtain data to support or substantiate evidence given by other projections.

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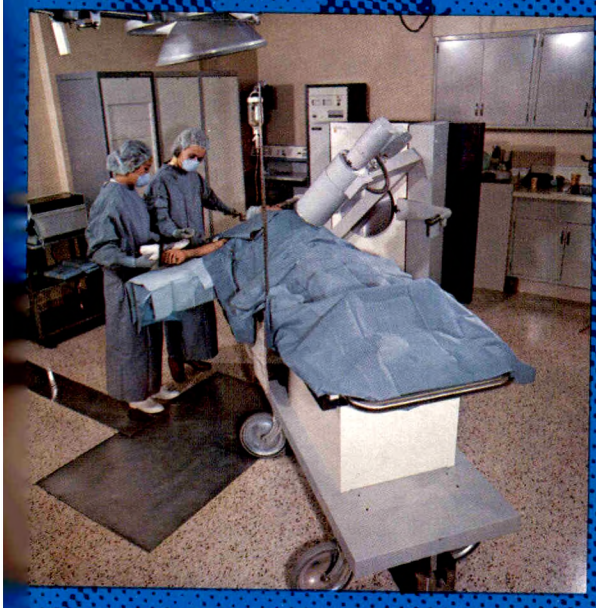

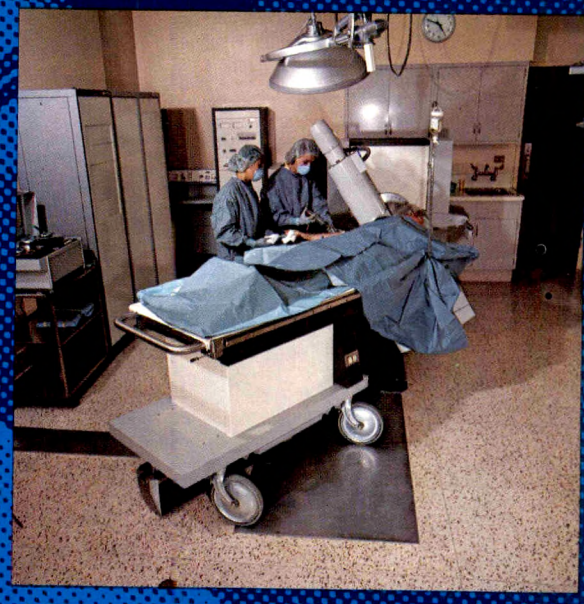

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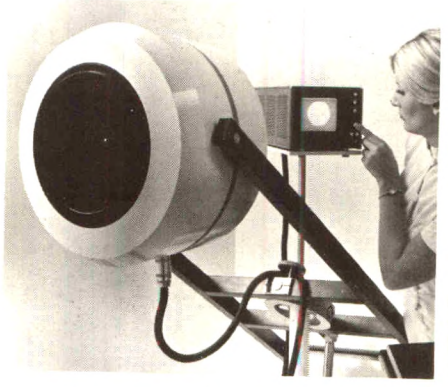

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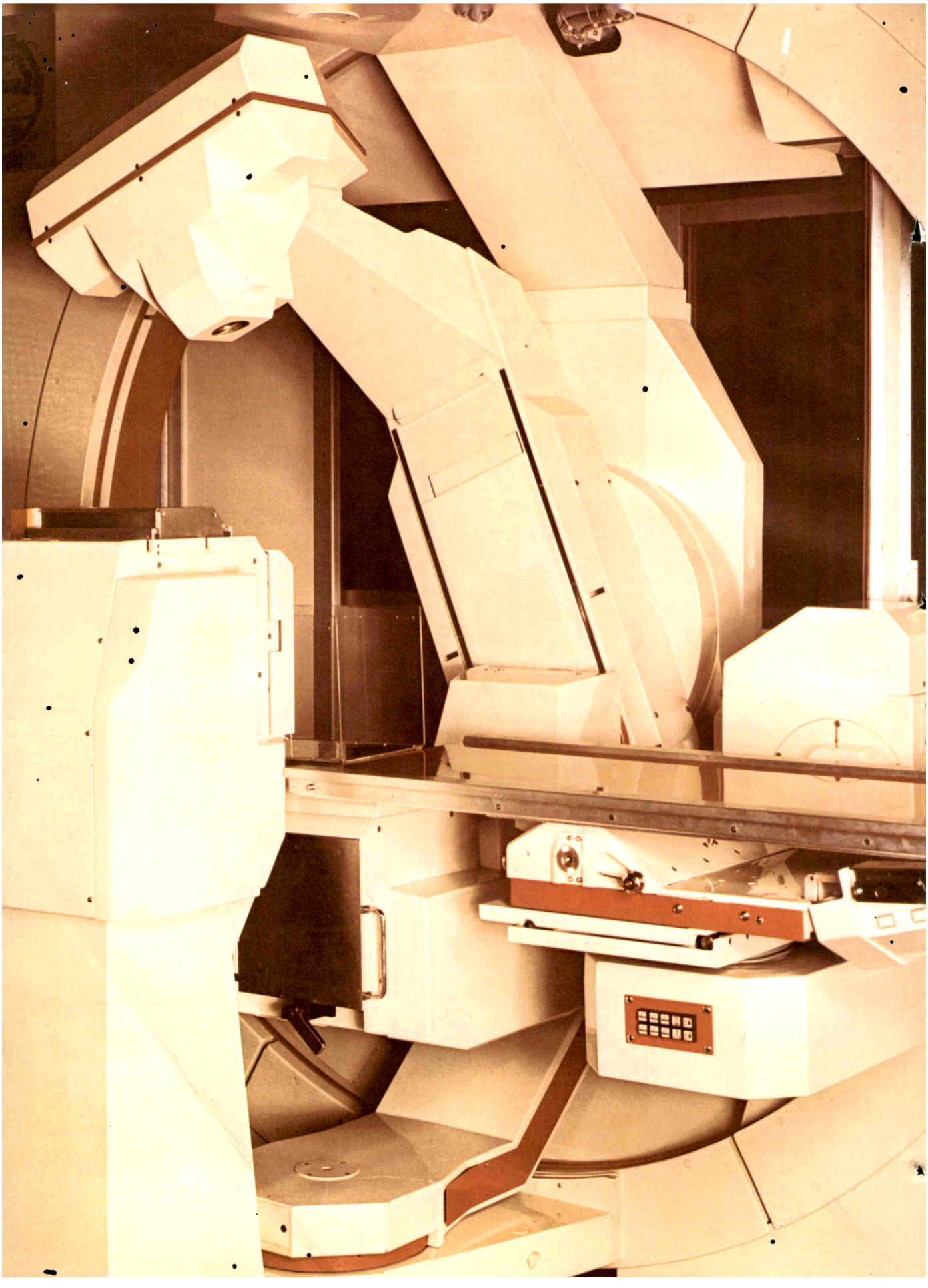

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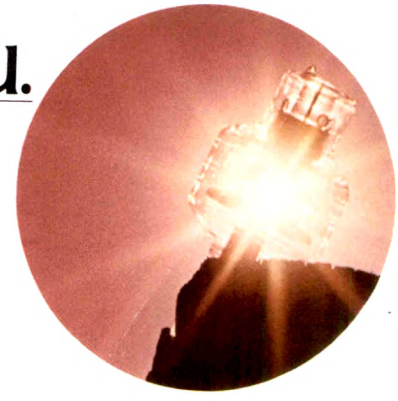

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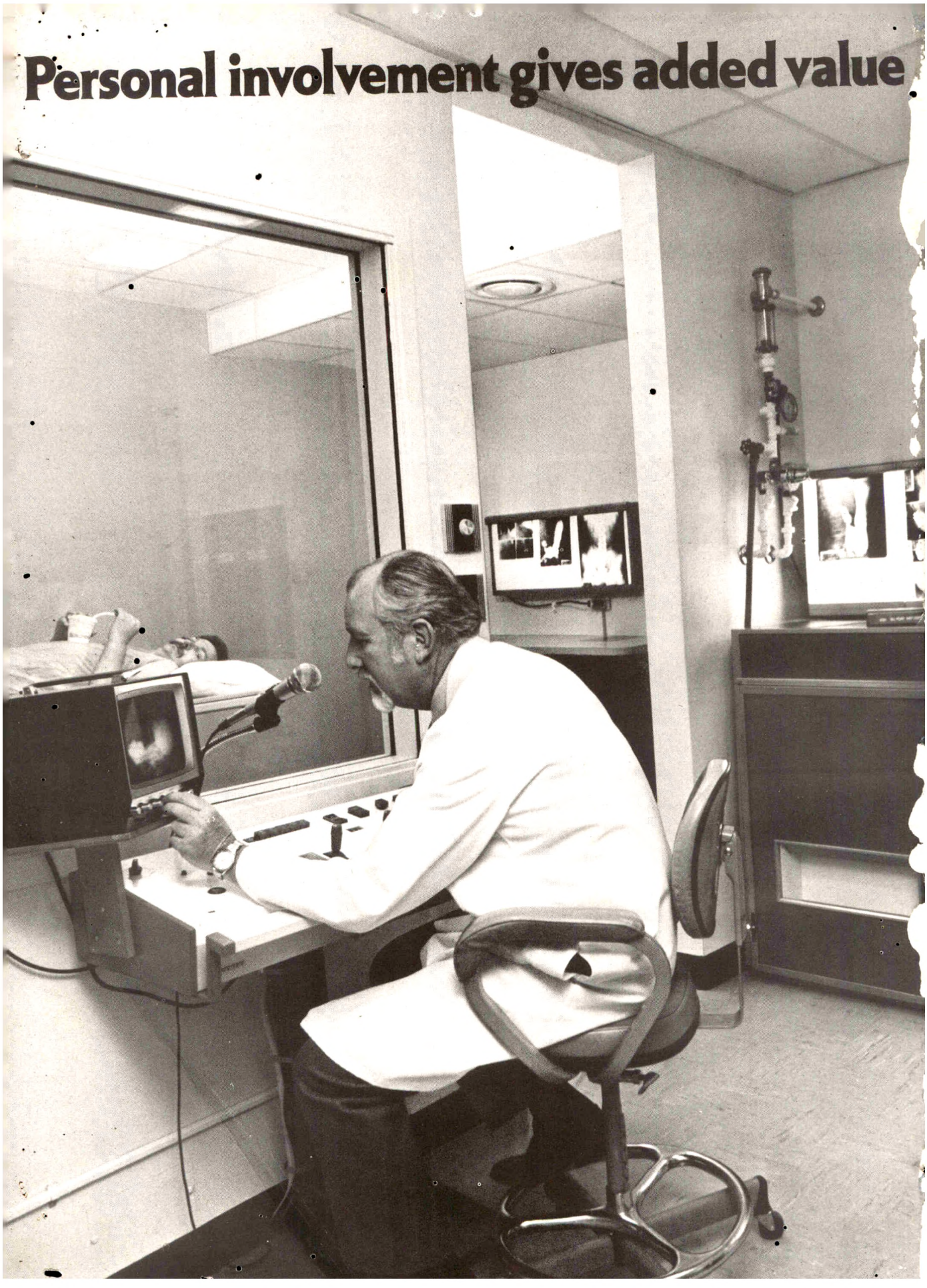

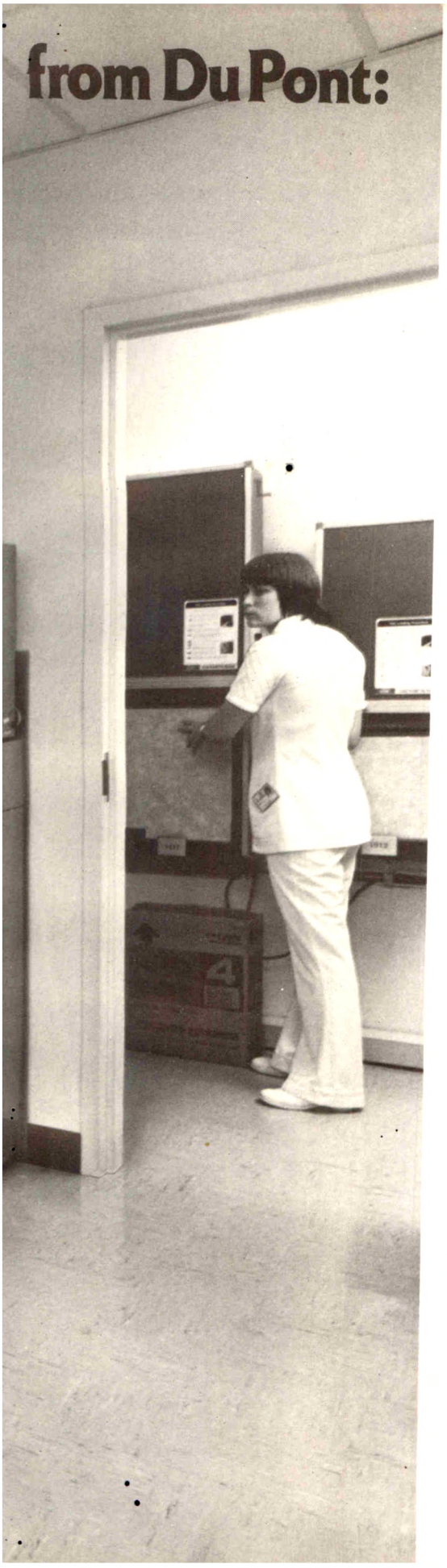

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Gordon Ritchie (right), Du Pont Technical Representative, pictured with the Administrative Director of Radiology at Methodist Hospital.

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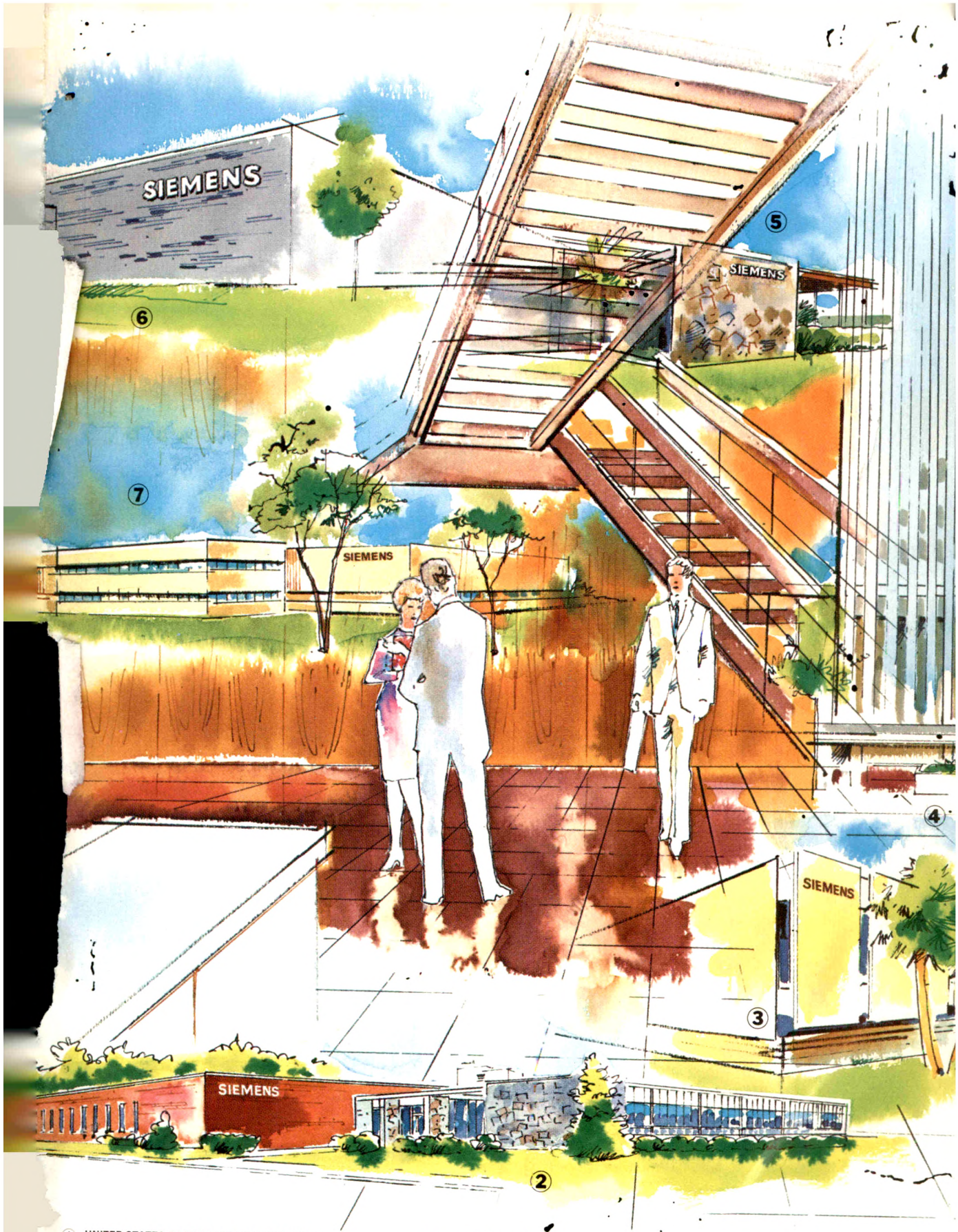

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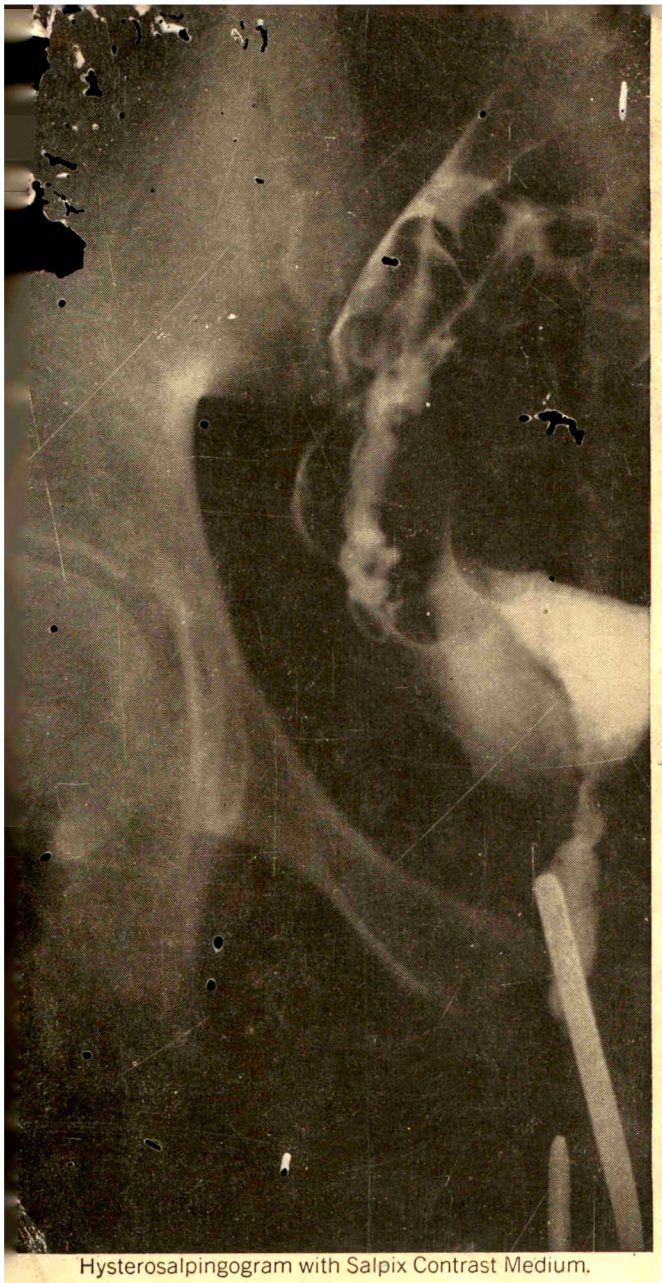

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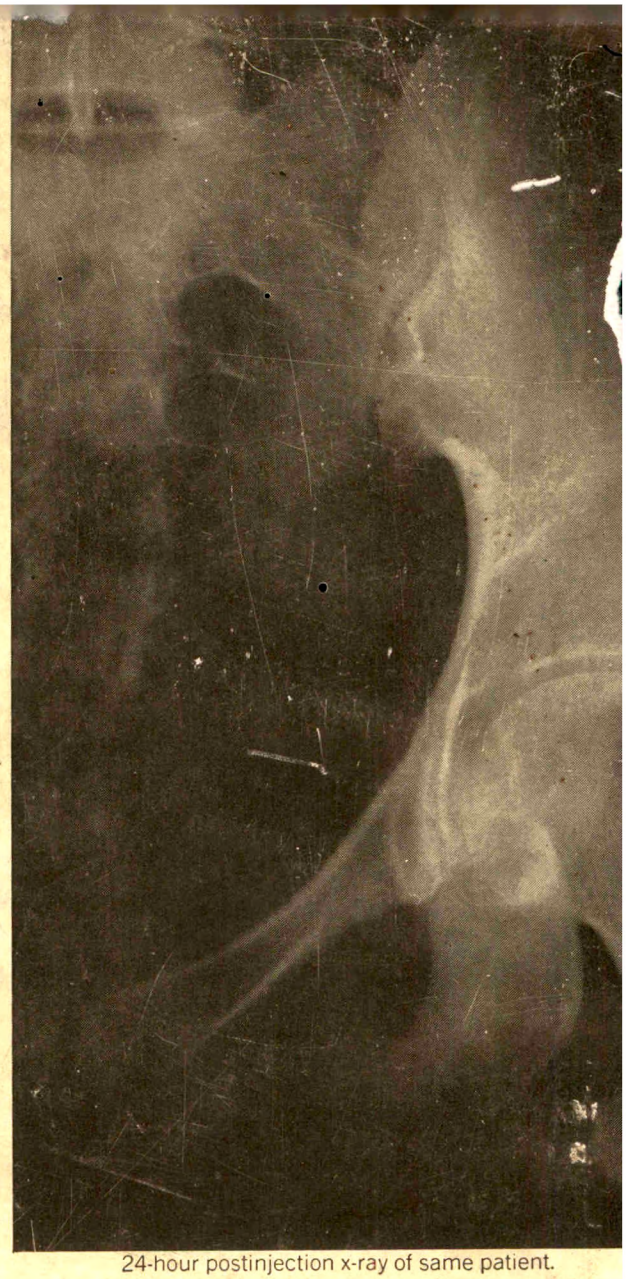

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